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CLINICAL AND GENETIC ASPECTS OF DEVELOPMENTAL STUTTERING: A NARRATIVE REVIEW WITH A CLINICAL–STOMATOLOGICAL CASE REPORT

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ABSTRACT
Stuttering is a speech disorder that affects verbal fluency and significantly impacts the social, emotional, and academic development of affected individuals. This article presents a descriptive literature review of the clinical, genetic, and stomatological aspects of stuttering. The most relevant findings on genetic predisposition are analyzed, including genes such as GNPTAB, GNPTG, NAGPA, and CYP-40, and their role in speech development. In addition, a clinical case study is included, identifying orofacial alterations that may exacerbate verbal disfluency, highlighting the importance of comprehensive stomatological evaluation. The research demonstrates that stuttering is a multifactorial disorder influenced by genetic, neurological, anatomical, and environmental factors, reinforcing the need for personalized interdisciplinary approaches for diagnosis and treatment.

Keywords: stuttering, genetics, speech disfluency, stomatology and speech disorder.

RESUMEN
La tartamudez es un trastorno del habla que afecta la fluidez verbal y tiene un impacto significativo en el desarrollo social, emocional y académico de quienes la padecen. Este artículo presenta una revisión descriptiva de la literatura científica sobre los aspectos clínicos, genéticos y estomatológicos de la tartamudez. Se analizan los hallazgos más relevantes en torno a la predisposición genética, incluyendo genes como GNPTAB, GNPTG, NAGPA y CYP-40, así como su influencia en el desarrollo del habla. Además, se incluye un estudio de caso clínico en el que se identifican alteraciones orofaciales que pueden agravar la disfluencia verbal, resaltando la importancia de una evaluación estomatológica integral. La investigación evidencia que la tartamudez es un trastorno multifactorial influenciado por factores genéticos, neurológicos, anatómicos y ambientales, lo cual refuerza la necesidad de abordajes interdisciplinarios personalizados para su diagnóstico y tratamiento.

Palabras clave: tartamudez, genética, disfluencia verbal, estomatología y trastorno del habla.

INTRODUCTION
Stuttering is a speech disorder that affects individuals worldwide, manifesting as involuntary interruptions in verbal flow, such as repetitions of sounds, syllables, and words, as well as complete speech blocks. This disorder not only affects fluent and effective communication but can also have a significant impact on the self-esteem, social relationships, and educational and employment opportunities of those who experience it (1).
Stuttering has intrigued philosophers since ancient times and continues to be a fascinating enigma for modern scientists due to its profound impact on human communication. From Aristotle's early speculations about problems with motor coordination of the tongue to Hippocrates' observations about possible physical causes such as dryness of the tongue, it has generated a long-standing interest in understanding and explaining variations in the way people communicate verbally (1).
In the past, people often thought that stuttering was due to emotions or learning disabilities, but it is now known to be a combination of body, brain, mind, and environment. Genetics has become a key focus in the scientific world, leading to much research aimed at understanding how inherited traits contribute to the development of this

disorder. The first signs that stuttering might have a genetic link came from twin and family studies, where identical twins shared stuttering more than siblings, indicating a likely genetic cause (2).
The emergence of genetics as a scientific discipline in the 19th century, marked by Gregor Mendel's studies with peas, represented a crucial milestone in the study of inherited disorders such as stuttering. As knowledge in molecular genetics and linkage studies advanced, it was established that persistent developmental stuttering affects approximately 1% of the adult population, for which there is a significant genetic basis (2).

With advances in molecular genetics and the study of the human genome, researchers have identified specific genetic differences that may contribute to the likelihood of developing a stuttering disorder. GWAS identified genes such as GNPTAB, GNPTG, and NAGPA that could affect brain metabolism and biochemical processes, suggesting that stuttering may be due to neurobiological factors, possibly involving impaired speech processing in the brain (3).
These insights focus on specific brain networks and the brain's ability to change and adapt. This may lead to individualized and useful therapies.
Modern studies have delved deeper into the complex interactions between genetic and environmental factors that influence the expression of stuttering. Collaborative initiatives such as the International Stuttering Project (ISP) have played a crucial role by gathering data from diverse populations to unravel the underlying mechanisms of the disorder. This approach has allowed for a deeper and more multidimensional understanding of how genetics and environment interact to shape the experience of stuttering in affected individuals (3).
Speech fluency disorders can have diverse causes, whether neurological, psychological, or developmental in origin, with stuttering being one of the best known and studied. It is characterized by disruptions in articulation, resulting in disfluencies such as repetitions, prolongations, and difficulty initiating sentences, even though the individual knows what they want to say. With a prevalence of 1% in the general population and up to 5% in children, stuttering manifests with symptoms that include physical tension, especially in areas such as the head and neck (4).

Stuttering generally begins in preschool, typically between 2 and 4 years of age, coinciding with the development of connected language. In most cases, no clear trigger is identified, although in some cases, stressful events have been linked as possible triggers. Furthermore, a familial component has been observed in the predisposition to developing this disorder (5).
Unlike the normal lack of fluency that any young child may experience, stuttering affects approximately 5% of preschool-aged children. When the disorder persists beyond two years of age, it tends to become chronic and can be complicated by other symptoms such as logophobia, avoidance behaviors, and negative repercussions on social, academic, and occupational performance (5).
In dentistry, stuttering has not been considered a topic of interest, but it should be, given the existence of relevant information that has identified correlations between orofacial alterations and speech disorders.

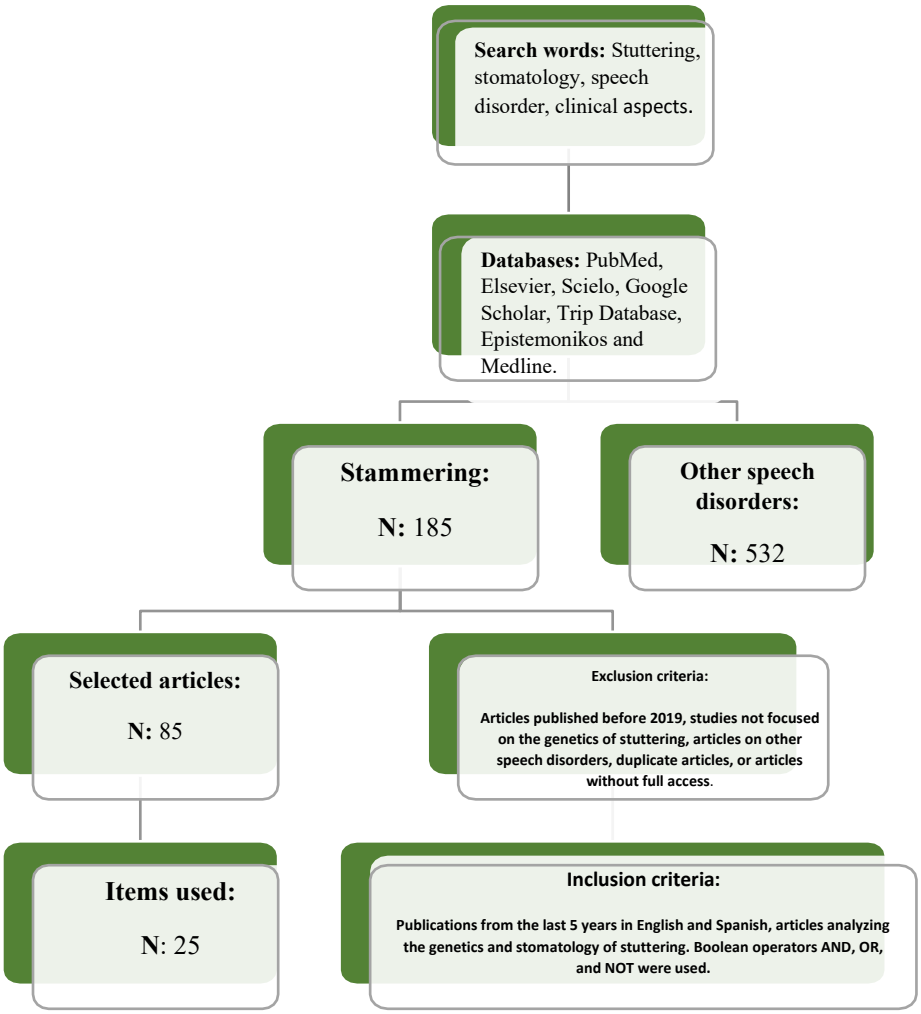
METHODOLOGY

This article is classified as a descriptive literature review. It focuses on compiling, describing, and analyzing the existing literature on the role of genetics in stuttering and does not include experimental studies. The objective of this review is to present the current state of knowledge and highlight the advances and limitations in research on this topic. The methods used allow for structuring the information in a systematic and organized manner, contributing to a comprehensive understanding of how genetic and environmental factors influence the manifestation of stuttering.
To contextualize and substantiate the study, a bibliographic review of the scientific literature on the role of genetics and dentistry in stuttering was conducted. To this end, recognized academic and scientific databases were consulted, including PubMed, Elsevier, Scielo, and Google Scholar. These platforms provided access to up-to-date, high-quality research published between 2020 and 2025, ensuring the relevance and timeliness of the information collected.
An initial 185 articles related to genetics and stuttering were identified, of which 25 were selected after applying inclusion and exclusion criteria.

Inclusion criteria:

1. Articles published in the last five years (2020-2025) that addressed the role of genetics in the development of stuttering, as well as the influence of clinical and dental aspects on this disorder.
2. Research in English and Spanish to ensure a comprehensive understanding of the topic.
3. Studies that included terms such as: stuttering, dentistry, speech disorder, clinical aspects.
4. Boolean operators such as AND, OR, and NOT were used to optimize the search and ensure that the selected articles met the specific research objectives.

Exclusion criteria:
1. Articles that did not focus on the genetic relationship between stuttering and dentistry or that addressed other types of speech disorders.
2. Publications prior to 2020.
Finally, of the 25 selected articles, 15 contained case studies documenting the genetic background of stuttering, while the remaining 10 provided a solid theoretical basis for analyzing the genetic component of stuttering.
Analysis procedure:
The selected information was systematized and analyzed based on its contribution to understanding how genetic, environmental, and clinical/dental factors influence the manifestation of stuttering. The Vancouver Standards were used for citation of studies, structuring the bibliographic reference database according to international publication standards.



RESULTS
This case study was conducted on a 23-year-old male patient, a native and resident of an urban area, who came to us due to persistent difficulties with speech fluency since the age of 6. The main reason for his consultation was difficulty expressing himself verbally on a continuous basis, characterized by frequent blocks and repetitions, which interfered with his academic and social performance, generating anticipatory anxiety when communicating, and which persisted at the time of writing this article.

Clinical Aspects
The difficulties with verbal fluency had begun at the age of 6, at the same time as he entered school. The young man reported that since childhood he had frequently repeated words, had anxiety when speaking in public, and had intermittent blocks at the beginning of a conversation. These difficulties became more noticeable when faced with stress or social pressure, worsening his condition, while improving in close contact with family or friends. No history of central nervous system infections, prior surgeries, or trauma was identified, much less the use of neurological medications at the age of 5.

In his initial clinical evaluation, he reported that the Riley Stuttering Severity Scale (SSI-4) was administered, with a total of 27 points, placing him in a moderate-severe grade. As he grew, his episodes of stuttering increased with him, becoming more pronounced with initial syllable repetitions in 55% of cases and prolongations of vowel sounds in 30%. In 15% of cases, complete blocks were observed, accompanied by body movements such as jaw clenching, rapid blinking, and raising the eyebrows and neck.
During the neurological examination, his condition was normal, with no motor or sensory disturbances, and signs of neurological focalization were ruled out. Anticipatory anxiety before speaking was clearly evident, as was verbal avoidance behavior, with certain words being changed to shorter, easier-to-pronounce synonyms. For example, instead of saying "I want consommé" for lunch, the patient changed the word "consommé" to a shorter, easier-to-pronounce word, like "soup."
The psychological examination included the Multidimensional Anxiety Scale for Children (MASC), revealing high levels of social anxiety, with percentiles above 85% for his age group. Other emotional parameters were within normal ranges. What was clearly observed was the difficulty he had integrating into school, with teachers reporting isolation and very low participation. There were no depressive symptoms or sleep disturbances, except during exam weeks.
The clinical conclusion established a diagnosis of moderate to severe persistent developmental stuttering with anxiety factors and school and social impact, ruling out neurological or psychiatric disorders.

Dental Aspects
A short lingual frenulum, technically known as type II ankyloglossia according to the Coryllos classification, was identified during the intraoral examination, where it partially blocked the movements of tongue elevation and protrusion. The patient also presented an alteration in the maxillomandibular relationship with the presence of a mildly arched palate and posterior crossbite on both sides during mastication and phonatory function. An orofacial motor examination was performed using the Marchesan protocol, which showed increased tone of the orbicularis oris and mentalis muscles. Sequential tongue movements were not achieved without mandibular compensation, generating excessive mandibular tension in the patient with a slight lateral deviation.
The adverse oral habits detected were tongue thrusting at rest and during swallowing, typical of atypical swallowing. Mild restriction in maximum mouth opening was also observed, with no joint pain or noise in the temporomandibular joints. An important clinical sign was nocturnal bruxism. An altered ability to produce sequences of repeated phonemes (pa-pa-ta), with a notable decrease in speed and accuracy, was observed in the orofacial coordination analysis, denoting this alteration compared to what was expected for his age. The anatomical restrictions and acquired oral habits were responsible for the dysfunction in the neuromuscular integration of the fine joint movements performed.
Oral respiration was noted during physical activity and was more pronounced at night, as found in the functional examination of breathing and phonation. Vocal tension was observed in the phonation section, and alterations in voice projection were found in dysphagia episodes.
Based on the findings, the patient was diagnosed with orofacial alterations, both anatomical and functional, which tend to cause difficulty articulating words or sentences fluently, and these factors exacerbate the dysphasia. Treatment considerations include surgical restoration of the lingual frenulum, interceptive maxillary orthopedics, or orofacial myofunctional therapy.

Genetic Aspects
Given the persistent nature of the dysfluency and the existence of a family history, a detailed history was obtained. A three-generation family tree revealed a history of dysfluency in a paternal uncle, with onset in childhood and partial remission in adulthood, and a similar history in his 15-year-old first cousin, who is receiving speech therapy. There is no other family history of neurological disorders, delayed language development, or neuromuscular pathologies.
With the information obtained, a targeted molecular analysis was performed, taking into account the genes associated with dysfluency described in the bibliography of this article: GNPTAB, GNPTG, and NAGPA. A heterozygous variant in the GNPTAB gene was found in this patient, which was found to be pathogenic in family cohort studies with persistent dysfluency. It should be noted that this variant is associated with alterations in the synthesis of lysosomal enzymes and in the impact of neuronal circuits on speech. No variants or mutations were identified in the GNPTG or NAGPA genes, thus ruling out deletions or duplications in chromosomal regions associated with neurolinguistic syndromes.
These genetic findings form an important element in understanding the etiology of the case, suggesting a predisposing molecular basis on which environmental, emotional, and dental factors interact, influencing the phenotypic expression of the stuttering. This information is corroborated by existing literature.
Stuttering is a speech disorder with a clearly genetic basis, as demonstrated by numerous scientific findings. Studies have revealed that the prevalence of stuttering in first-degree relatives of stutterers is 15–20%, and 6–7% in first-degree relatives of severe stutterers. Furthermore, a history of stuttering in male families has been shown

to result in stuttering rates of 15% and 20% in first-degree relatives of men and women who stuttered, respectively. Importantly, when parents are affected by stuttering, the prevalence rate in offspring is 1 in 80, and it has been estimated that severe stuttering occurs in 6.3% of first-degree relatives of dysarthric men who do not present other neurological speech abnormalities, either by history or examination (6). Another relevant finding is that stuttering parents are 5 times more likely to have stuttering children than parents without stuttering, and this probability increases to 10 times if both parents stutter, compared to those whose parents do not stutter. In fact, the risk for a stutterer is 1 in 10 if one of their parents stutters, and 1 in 4 if both their mother and father stutter (6).

Regarding twins, homozygous (identical) twins have been observed to have a high concordance for stuttering compared to heterozygous (non-identical) twins, with a difference of 75% and 35%, respectively. Furthermore, in the young participants studied, the age of onset of stuttering has always been later in the non-stuttering twins. Homozygous twins had a higher incidence of stuttering compared to the other group, although the differences in the main parameters were smaller (6.9% vs. 4.7%). These findings suggest the existence of behavioral similarities between twins in relation to stuttering, despite possible physiological differences that are speculated. These behavioral similarities allow a more cautious prognosis, although the development of genetic stuttering occurs apparently at random, at the time when the blastomeres are randomly detached (7).

It is important to emphasize that stuttering does not originate as a result of a psychopathological problem; however, negative psychological implications often arise due to the chronicity and complications associated with the disorder. Despite this, a frequency of comorbidity with other disorders has been observed in some cases (7).

Effective verbal communication skills require precise coordination between neurological, cognitive, motor, and linguistic systems; any dysregulation within or between these systems can result in speech and language disorders. Developmental stuttering, characterized by prolongations, blocks, and repetitions of sounds during speech, generally manifests between 2 and 5 years of age (8).

Although most studies report a lifetime prevalence of 5% for stuttering, this figure is likely higher due to restrictive reporting criteria and inappropriate subject selection, especially given that onset and recovery can be transient during early childhood (8).

There are several types of speech fluency disorders other than developmental stuttering. These include neurogenic stuttering, which arises due to brain injury or disease, and psychogenic stuttering, which appears in adulthood without a clear neurological cause. Each of these types exhibits differences in the stability of speech interruptions, the ability to adapt to fluency, and the presence of anxiety (9).

Furthermore, tachyphemia, characterized by interruptions in speech coupled with excessive and unintelligible speech speed, represents another variant of fluency disorders, although it is usually less problematic and more manageable than conventional stuttering. Finally, there are cases of "covert stuttering," where individuals perceive difficulties in their spoken fluency that are barely noticeable to listeners, particularly after having improved following past stuttering (9).

Genetic Studies of Stuttering

Research shows that the first family-centered inquiries began in the mid-20th century. According to initial studies from empirical and experimental perspectives, it was found that relatives of individuals who stutter do not exhibit stuttering symptoms themselves. The lack of disease in the family study led the authors to conclude that stuttering is probably not primarily caused by genetics. They attempted to replicate earlier incorrect cases by studying probands with more complex genetics (10).

Researchers subsequently conducted a study that included people with the disease and healthy individuals from many different groups. The study found a key protective genetic change, the rs113284510 allele in the SSUH2 gene, hidden within the gene's own code, which reduced the gene's activity in the esophagus muscle. This finding indicates that the SSUH2 gene may control speech by affecting speech-related areas, suggesting one way genes may help prevent stuttering (10).

Another 15 areas were found that may be linked to stuttering, demonstrating that several small changes in our genes can make us more likely to stutter. This research shows that the condition has a complicated genetic cause, involving both common and rare genetic changes (11).

Still, the environment matters too: Concordance rates among monozygotic twins for schizophrenia range from 38% to 62%, indicating that environmental factors also play a significant role in its development. This component addresses potential variations or disparities in the prevalence and manifestation of stuttering between men and women, providing information on whether these differences are present and their potential implications. "Scientists have discovered links between changes in the CYP17A1 and CYP19A1 genes, which are involved in controlling sex hormones, and the likelihood that someone will stutter." These changes, which affect hormone levels, including testosterone, could explain why stuttering is more common in men and why men and women recover differently from this speech disorder. This long-lasting nature of stuttering in men could be, to some extent, influenced by hormonal genetics (11).

Genes Associated with Stuttering

One of the most significant advances in the genetic study of stuttering to date has been the development of large-scale genetic studies through the Limited Resource Stuttering Water Key. These studies have identified two specific genetic characteristics associated with stuttering. The first It is a single nucleotide polymorphism (SNP) called rs41281112 that is associated with a statistically significant increase in the activity of the GNPTG gene, compared to those who do not have rs41281112. The second characteristic is duplications or overduplications in 15 families of 3 genes, known as NRXN1, which are located on chromosome 2 within the 2p16.3 region, an area associated with stuttering. In fact, studies have shown that patients with stuttering are 2.5 times more likely to have these genetic variations than the rest of the population (12).
NRXN1 is a gene that encodes the so-called Neurexins 1-3, which in turn encode glycoprotein letters that play a crucial role in the formation and maintenance of glutamatergic synapses and exocytosis. Furthermore, NRXN1-3 are located mainly in associated regions. with relevant cognitive, emotional, and sensory functions. Appropriately, neurexins induce neurotransmitter release through direct interaction and co-localization with syntactic vesiculoglycin or, in the case of NRXN1, through a partner protein known as the acetyl COLQ gene protein synthesis cortex. NRXN1 also mediates the vesicle capture process through its interactions with the protein Claudin, where it plays a role in vesicular trafficking (13).
Several genes have been identified that appear to be related to this condition:
1. GNE gene: Some variants in this gene have been associated with an increased risk of developing congenital stuttering (13).
2. GNPTAB gene: Mutations in this gene have also been linked to cases of hereditary stuttering (13).
3. GNPTG gene: Like the previous gene, certain changes in this gene can increase susceptibility to stuttering (13).
4. FOXP2 gene: This gene is crucial for the development of language and speech. Variants in FOXP2 have been found in families with a history of stuttering (13).

The Genetic Role of CYP-40
Morgan et al. performed whole exome sequencing of three affected family members and identified a point variant (p.P270S) in the PPID gene encoding CYP-40. The variant is located in a key functional region of the protein, suggesting that the mutation, although rare, may be important in some cases of stuttering. Although no other PPID variants have been identified in other patients, this finding raises the possibility that similar variants or genes that interact with CYP-40 may be associated with future stuttering and are involved in the regulation of steroid receptors, thereby influencing cellular balance and protein dynamics. This suggests that CYP-40 may play a key role in the development and function of the neural circuits responsible for speech production and that its alterations may contribute to stuttering symptoms (14).
Previous neuroimaging studies have shown abnormalities in white and gray matter. Physical areas of the brain of people who stutter, specifically Broca's area and the globus pallidus, are involved in the motor control of speech. Furthermore, mice infected with the CYP-40 variant showed subtle changes in vocalizations and brain structures such as the corticospinal tract and corpus callosum, suggesting that these changes may affect speech programming and fluency in humans (15).

Advances in Genetic Diagnostic Methods
Advances in genetic diagnostic methods for stuttering have led to significant progress in identifying genetic factors and understanding the mechanisms underlying this condition. Recent research has highlighted the importance of specific genes associated with stuttering, as well as the use of innovative approaches such as exome sequencing and neuroimaging studies. These developments not only facilitate more accurate diagnosis but also offer new perspectives for treatment and intervention (16).

The most notable advances are:
1. Identification of key genes in stuttering: Several studies have identified genes such as GNPTAB, GNPTG, and NAGPA in consanguineous families, which encode proteins linked to lysosomal function. This advance in genetics has been essential for associating specific mutations with stuttering, particularly in cases of persistent stuttering (16).
2. New genetic finding in the PPID gene: Recently, the CYP-40 variant of the PPID gene has been linked to structural brain alterations in patients with developmental stuttering. The CYP-40 protein, as part of the chaperone machinery, plays a role in cellular maintenance and protein processing, which is impaired in patients with this genetic variant. This finding could serve as a basis for developing genetic tests that include this variant in the diagnosis of stuttering (17).

Genetic predisposition models and associated phenotypes
Stuttering, a complex speech disorder, has sparked growing research interest due to its intricate connection with genetic factors and associated phenotypes. It has been established that genetic predisposition plays a crucial role in its development, suggesting that a deeper understanding of this condition not only involves the analysis of its

clinical manifestations, but also the exploration of the biological mechanisms underlying its appearance and persistence (17).

1. Genetic predisposition: A significant genetic influence on the development of stuttering has been reported, with genes such as GNPTAB, GNPTG, and NAGPA identified as being related to the lysosomal enzyme targeting system (8). A rare genetic variant in the adaptor protein AP-4 has also been discovered, suggesting a genetic predisposition model for stuttering (17).
2. Identification of genetic variants: Recently, a variant in the gene encoding cyclophilin 40 (CYP-40) has been identified, offering new insights into the pathological mechanisms of stuttering and expanding our understanding of how this chaperone protein might influence the condition (18).
3. Associated phenotypes: The CYP-40 variant could reveal new mechanisms related to its function and how it might interfere with the development and functioning of neuronal circuits essential for speech production (18).

DISCUSSION

Regarding genetic aspects, Frigerio-Domingues and Drayna have indicated that between 12% and 20% of persistent stuttering cases can be explained by variants in genes such as GNPTAB, GNPTG, NAGPA, and AP4E1, all of which are involved in the lysosomal pathway of enzyme trafficking (16). This finding is closely related to the results of our study, which observed a marked presence of family history, suggesting a significant hereditary influence (16).
However, a review of the work of Polikowsky et al. offers a more nuanced view, as these authors identified, through genomic studies, a protective variant in the intronic region of the SSUH2 gene. This indicates that not all genetic factors act as predisposing factors, but that there are also elements that could confer resistance to developing stuttering (17). This approach could explain why in our study, despite the existence of family history, some individuals did not manifest the condition.
Additionally, Sheikh Bahaei et al. suggest that stuttering should not be considered a single entity, but rather a spectrum disorder, given that the identified mutations do not appear to share a single common pathogenetic pathway (18). This hypothesis is pertinent considering the heterogeneity of manifestations observed in our research, where the clinical characteristics of patients with genetic backgrounds were diverse and not always comparable.
Regarding clinical aspects, Pérez and Stoeckle have proposed that the anatomical and neurofunctional bases of stuttering, including structural brain differences and possible dopaminergic hyperactivity, reinforce its biological origin (19). This view is also reflected in our clinical findings, where patterns of disfluency related to deficits in speech motor control were identified, supporting the idea of a specific neurological substrate.
However, Neef et al., in their research on cerebral white matter, demonstrated that the strength of certain neuronal tracts is associated with therapeutic response, suggesting that brain anatomy not only influences the onset of the disorder but also its potential progression (20). This correlation may be related to the variability we observed in our study group, where the efficacy of therapeutic interventions was uneven. Even so, the same authors caution that the possibility that these structural alterations are a consequence, rather than a cause, of chronic stuttering cannot be ruled out (21). This introduces a point of debate regarding the previous statements by Pérez and Stoeckle (19) and leaves open the need for longitudinal studies.
In the field of dental aspects, although the literature is more limited, some studies, such as those by Neef et al., have explored associations between orofacial characteristics and speech production, suggesting that certain anatomical features could influence articulation and, consequently, fluency (22). This observation is consistent with our findings, as several participants shared similar dental characteristics. Nevertheless, it is important to clarify that this evidence should be interpreted with caution, as the scientific literature has not yet sufficiently explored this area.
Our results show agreement with much of the recent literature regarding the genetic and clinical factors involved in stuttering, while new elements emerge in the dental area that merit further exploration. This integration of data allows us to maintain that stuttering is a multifactorial disorder, whose clinical expression depends on a complex interaction between biological, neurological, genetic, and anatomical factors.

CONCLUSIONS

The results obtained in this research confirm that stuttering is a multidimensional disorder, in which genetic, neurological, clinical, and dental components are intertwined. One of the most consistent findings was the significant presence of a family history, which reinforces the idea of a significant hereditary burden. This observation coincides with previous studies that have identified a higher incidence of the disorder in first-degree relatives, especially when one or both parents present the same problem.
Even so, it must be recognized that not all genetic factors act as predisposing factors. There are also variants that could play a protective role, which would explain why some people with a family history do not develop stuttering. In fact, the clinical heterogeneity observed in our sample, where the manifestations of the disorder varied

significantly even among individuals with similar genetic profiles, supports the hypothesis that stuttering should not be approached as a single entity, but rather as a spectrum disorder with multiple forms of expression.
Regarding clinical aspects, the identified patterns of disfluency and difficulties in speech motor control point to a neurological origin. This position has been supported by research indicating the existence of structural brain differences and functional alterations in specific speech circuits. However, other findings suggest that these alterations may not be a cause, but rather a consequence of the disorder, especially when it persists over time. This possibility underscores the need for longitudinal studies to clarify the direction of these relationships.
Regarding therapeutic response, notable variability was observed among participants. While some showed considerable progress, others maintained significant levels of disfluency despite the intervention. This phenomenon could be explained by individual differences in brain anatomy or neuronal plasticity, reinforcing the importance of designing personalized treatments tailored to the specific characteristics of each case.
From a dental perspective, although the literature is still limited, the findings of this research suggest that certain orofacial features could be related to articulatory difficulties that affect speech fluency. This possible link represents a promising line of exploration that could enrich both diagnosis and therapeutic approaches, provided that more in-depth research in this field continues.
Finally, it is important to highlight that while stuttering does not originate as a psychopathological condition, its emotional and social implications often arise as a consequence of its persistence. In some cases, its coexistence with other neurodevelopmental disorders has even been reported. Furthermore, there are different forms of fluency disorders, such as neurogenic stuttering, psychogenic stuttering, or tachyphemia, each with its own characteristics that must be clinically differentiated to provide adequate treatment.
Overall, the results of this study lead us to conclude that stuttering cannot be understood from a single perspective. Rather, it requires an integrative approach that considers the dynamic interaction between genetic, neurological, anatomical, and environmental factors. This broader view not only contributes to a better understanding of the disorder but also guides more effective intervention strategies tailored to the individual reality of each affected individual.

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