OPEN ACCESS

Saudi Journal of Oral and Dental Research
Abbreviated Key Title: Saudi J Oral Dent Res
ISSN 2518-1300 (Print) | ISSN 2518-1297 (Online)
Scholars Middle East Publishers, Dubai, United Arab Emirates
Journal homepage: https://saudijournals.com

Original Research Article **Dental**

Effect of Parental History of Periodontal Disease on Children

Laila Mohammed Alanazi[1*], Dalal jumah Alturaif[1], Miqdad Hussain Alhassan[1], Afaf Mofleh Salem Alshahrani[1], Rahaf Jamaan Saeed Al-Ghamdi[1], Maan Mohammed A Shabi[1], Riyadh Ahmed Almalkie[1]

[1]General Dentist, Saudi Arabia

DOI: 10.36348/sjodr.2022.v07i08.002 | **Received:** 28.06.2022 | **Accepted:** 06.08.2022 | **Published:** 12.08.2022

***Corresponding author:** Laila Mohammed Alanazi
General Dentist, Saudi Arabia

Abstract

Periodontitis is a risk factor for children who practice poor oral hygiene at home. This is due to the higher frequency of association between children's and parents' microbiota. It is likely due to the influence of both, hereditary and environmental factors. Although it is possible for periodontal disease to be passed down across generations, the underlying mechanism behind this is still unknown. According to clinical study, genetic predisposition accounts for 50% of an individual's sensitivity to periodontal disease. Because clear information on the issue is sparse, the purpose of this study is to examine the known studies on the impact of a family history of periodontal disease on children. English-language articles, case reports, and case series published from 2013 to 2022, taken from the Cochrane Library, SCOPUS, and MEDLINE databases (through PubMed) in accordance with PRISMA criteria were reviewed to research children with a history of periodontal disease. researchers for relevant materials. Twenty-nine children from various case studies were included in the present systematic study. In nine of the 29 studies, no family members had Hereditary gingival fibromatosis, a kind of periodontal disease while a family member had in the other 20 studies. One person had Zimmemrman-Laband Syndrome, two had severe periodontal disease, one had juvenile hyaline fibromatosis, and the other two had non-syndromic Hereditary gingival fibromatosis in four independent tests. In three case studies, children whose parents have periodontitis are likely to have periodontal disease. Children who have periodontal disease are more likely to develop the illness and should be checked and treated very once. More research is needed, especially well-designed studies that avoid the flaws mentioned in the publications included in this review.

Keywords: Oral health, Periodontal diseases, Children, Gingivitis, Periodontitis, familial history.

INTRODUCTION

Children's oral health concerns, such as periodontal disease, may be influenced by variables such as the child's individual attributes, the family's traits, and the community's characteristics (Tadakamadla *et al*., 2020). According to a recent research, children's self-care behaviours and service use are impacted by family variables and their likelihood of getting periodontal disease. This is due to the similarity between the cariogenic and periodontal pathogenic micro biota of children and their parents. This might be the result of both hereditary and environmental influences that are transmitted through generations (Kilian *et al*., 2016). A broad array of dental care awareness activities are focused at families in terms of improving children's oral hygiene practices and dental health quality as a whole (Nath *et al*., 2021).

Periodontitis is the more severe form of periodontal disease whereas, gingivitis, is the milder kind. Periodontal disease belongs to the category of inflammatory diseases (Curtis *et al*., 2020). Gingivitis is a common facet of periodontitis that affects people of all ages, including adolescents and children (Noorul *et al*., 2021). Less common forms of periodontal disease take account of acute necrotizing ulcerative gingivitis, aggressive periodontitis, and infections of herpesvirus and fungoids (Balaji *et al*., 2021). It's imperative to perceive that although periodontitis and gingivitis are commonly considered as two phases of the similar inflammatory conditions, several gingival plaques do not progress to periodontitis. As soon as teeth will erupt, bacteria begin to colonize areas in close proximity to the gingival border that are exposed to oral microbes. (Periodontal disease and gingivitis in 2020: A Comprehensive Guide) The severity of periodontal disease is proportional to the amount of pathogenicity

of the bacteria in the biofilm and immunity against biofilm microbiota through intracellular and physiologic means (Sedghi *et al*., 2021).

In young people, gingivitis often continues for an extended length of time without producing damage to the periodontal ligament or bone. Even yet, periodontal attachment may be lost if the delicate equilibrium between biofilm and host is disturbed (Kononen *et al.*, 2019). Gingivitis is the beginning stage of chronic periodontitis and aggressive periodontitis, notwithstanding the difficulties in identifying the underlying biochemical mechanisms. Chronic or serious gum disease starts with gingivitis (Botero *et al*., 2015). The progression of gingivitis to periodontitis may have been influenced by variables such as microbial dysbiosis, a rise in the quantity of pathogenic bacteria, reactivation of the herpesvirus, immune system dysfunction, and acquired and/or hereditary vulnerability (Chen *et al*., 2020).

It is more common in adults, but adolescents and teenagers may also be affected; the extent of tissue damage is often proportional to the levels and risk factors of dental plaque and host defence. Even though periodontitis is more common in adults, it is possible for children and teenagers to acquire the condition (Kinane *et al.*, 2017). The development of periodontal pockets, as well as the related attachment and bone loss, is not consistent across the mouth. This is a characteristic sign of chronic and severe periodontitis (Arjunkumar, 2018).

It is assumed that gingivitis affects the majority of children and adolescents worldwide, although to varying degrees. This is despite the fact that there is a dearth of data on the prevalence of periodontal ailments in younger population because of restricted research and non-uniform evaluation procedures. In contrast, gingivitis may be present (Triantafyllia & Georgios, 2018).

Is a history of oral illness in one's family a cause for concern? Growing body of research shows that dental health transfer from parent to baby may be a potential risk for juvenile caries. This study's research was conducted in the United States (Shearer & Thomson 2010). Earlier studies on periodontal disease have looked at the potential that severe periodontitis might be handed down via family members (Nibali *et al*., 2008). Although signs of chronic periodontal disease frequently don't develop until the thirties, there is a lack of study on the idea that the illness might be handed down from generation to generation (Kinane & Hart 2003). Unlike other diseases, periodontal disease is thought to be brought on by changes in the phenotypic rather than vicissitudes in the primary array of DNA, making this a particularly serious shortcoming (Barros & Offenbacher 2009, Gomez *et al.*, 2009). This suggests, periodontal health may be passed down from one generation to the next, but no one knows exactly how this works or how far it can spread.

According to clinical studies, hereditary factors account for half of an individual's vulnerability to periodontal disease (Loos & Van Dyke, 2020). Early-onset periodontitis is more common in women, according to a study on disease risk (Freitag-Wolf *et al*., 2021). It's possible that it's passed on as an X-linked dominant trait since there was no evidence of a transfer from father to son. Identical twins were reported to have similar levels of adhesion attrition, cavity level, gingival indices and bacterial indices (Zheng *et al*., 2018). Gingival growth is characterized by an increase in the size of the gingiva as a consequence of the expansion and accumulation of connective tissue. Drug-related (phenytoin, cyclosporine, and nifedipine-induced) gingivitis and chronic gingival inflammation are two more possible causes of this illness (Strzelec *et al*., 2021). Hereditary gingival enlargement, also known as idiopathic gingival enlargement, may develop. The gingival tissues grow slowly and benignly in this kind of gingival hypertrophy. Hereditary gingival fibromatosis, which affects babies, is a rare yet serious condition. Normal colour and stiffness of the gingiva are the first signs of gingivitis. There are no symptoms and no bleeding associated with this illness. Diastema, misaligned teeth, and uncontrollably wide lips may all be signs of this disorder. It has been connected to two chromosomes; the two GNGF and GINGF3 loci on 2p 1-22 and 2p 22.3 - p23 may be identified on the second chromosome, although the two regions do not overlap. GINGF2 is exposed on the 5q13-q22 area of chromosome 5 (Muhammad and Watted, 2019).

Due to the lack of commulative evidence of effect of famililal history of periodontal disease in children, we aim to review the available evidence on this subject.

METHODOLOGY

Search Strategy

These studies were steered in compliance with PRISMA guidelines and all authors agreed upon the procedure. To locate papers that may be taken into account, researchers examined the Cochrane Library database, SCOPUS and the MEDLINE database (through PubMed) from 2013-2022. Key words like "Hereditary gingival fibromatosis", "Periodontal disease". ”Family history”, and “children” are employed to retrieve the articles of interest.

Children with periodontal disease whose family had a history of the condition were studied using articles, case reports, and case series originally published in English. We uncovered possibly relevant papers by manually reviewing the reference lists of previously thoroughly studied publications. The following were excluded from the scope of the study: studies that did not apply to pediatric patients diagnosed

with HGF and other periodontal diseases and studies that were limited to adult patients (over the age of 18) and scholarly articles that included both adult and pediatric patients but did not present the results individually for the patients under 18 years old. When appropriate, quality control contacts with a senior author were employed to identify and remedy mistakes.

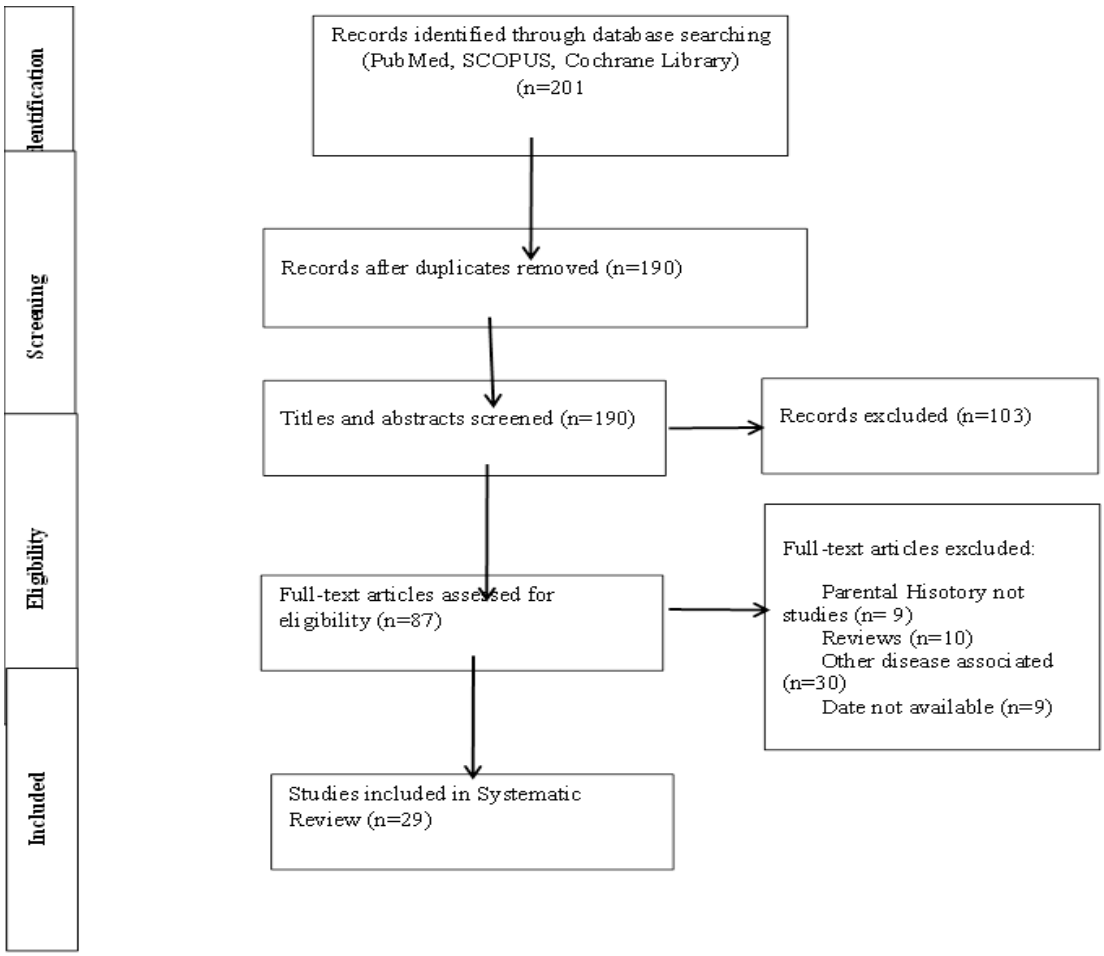
Data Tabulation and Extraction

A standardized and previously tested table was to be utilized to gather and present data from the included studies for evidence synthesis and assessment of study quality. For each research that matched the qualifying requirements, the following information was gathered: the study's first author, year of publication, country, study design, number of patients, gender, age, method of inheritance, association with syndromes, and family members afflicted by the condition. Data from all qualifying publications were sifted through to find relevant information from two studies that overlapped; however, only the biggest of those studies' populations were selected for inclusion. However, since they were new investigations on the same patient cohort, they were not included in the total. As a result, it wasn't included to the totals.

RESULT

Eligible Studies

Initial results from the search returned 201 possibly relevant records. After evaluating 87 papers based on their titles and abstracts, we downloaded their full-text versions and included 29 of them in our review. These comprise three case-control studies, while the other case studies satisfied the planned search criteria and were thus included in the literature evaluation. The technique of systematic literature extraction was documented in accordance with the PRISMA standards (Fig 1).



Patient characteristics and clinical manifestations

Study Characteristics

Case Studies

The current systematic review included a total of 29 pediatric patients from various case studies (Aghili & Goldani Moghadam, 2013; Baltacioglu *et al*., 2017; Dhadse *et al*., 2012; Duddu *et al*., 2012; Gao *et al*., 2020; Gawron *et al*., 2016; Gita *et al*., 2014; Japatti *et al*., 2013; Botero *et al*., 2015; Chaurasia, 2014; Chen *et al*., 2020; Cunha *et al*., 2020; Curtis *et al*., 2020; Kilian *et al*., 2016; Kinane *et al*., 2017; Könönen *et al*., 2019; Li *et al*., 2021; Manoj *et al*., 2017; Nath *et al*., 2021; Noorul *et al*., 2021; Peeran *et al*., 2013; Pego *et al*., 2015; Pol *et al*., 2016; Prasad *et al*., 2012; Saini *et*

al., 2013; Sharma *et al*., 2012; Tripathi *et al*., 2014; Umrania *et al*., 2016; Yadav *et al*., 2013). The ratio of girls to men was 1:1 (14 females, 15 males), and the mean age of the patients was 10.792 3.92 years (range: 3–15). The vast majority of people had global gingival expansion, whilst some exhibited only localized illness. The detailed study characteristic is tabulated in Table 1.

Table 1 Study charcterstics of included studies

Study	Country	Study design	No. of patient	Gender	Age	Disease	Family history
U KHAN 2012	Pakistan	Case study	1	Male	15	HGF	yes
Gita B 2014	India	Case study	1	Male	14	GF	No
Aghli 2013	Iran	Case study	1	female	9	nonsyndromic HGF	Yes
Yadav2013	India	Case study	1	Male	15	nonsyndromic HGF	no
Saini A 2013	India	Case study	1	Male	13	Isolated IGF	no
Japatti S 2013	India	Case study	1	female	15	IGF with localized aggressive periodontitis	no
Prasad 2012	India	Case study	1	female	13	‘HGF with unusual facies	yes
Dhadse 2012	India	Case study	1	female	12	HGF	yes (no PH)
Sharma 2012	India	Case study	1	female	8	familial gingival fibromatosis.	yes
Duddu 2012	India	Case study	1	Male	5	IGF.	no
Pol 2016	India	Case study	1	female	14	idiopathic GF	no
Pedro 2016	Spain	Case study	2	Male	8,5	HGF	N/A
Manoj 2017	India	Case study	3	male	15, 8,1	HGF	no
Umrania 2016	India	Case study	1	female	15	HGF	yes
Peeran 2013	Libya	Case study	1	female	13	HGF	yrs
Garon 2016	USA	Case study	1	female	15	HGF	yes
charausia 2014	India	Case study	1	female	10	HGF	n
Tripahi 2014	india	Case study	1	m	14	non syndromic HGF	yes
Esra 2017	Turkey	Case study	1	male	14	JHF	N/A
Gso 2020	China	Case study	1	male	8	HGF	yes
Kunha 2020	Brazil	Case study	1	female	10	HGF	yes
Ning Li 2021	China	Case study	1	female	7	HGF	yes
Gughemi	Italy	Case study	1	male	3	ZLS-1	no
Pego 2015	Brazil	Case study	1	female	3	HGF	yes
Reis 2020	Brazil	Case study	1	male	5	APD	yes
Reis 2020	Brazil	Case study	1	female	9	APD	yes
Monteiro 2021	USA	case-control	8	female	9	PD	yes
Pakhla 2009	Estonoa	casecontrol	20	female	19	PD	yes
Seebacher 2021	Austria	case-control	19	F/M	32	pEDS	yes

The main prevalent HGF-induced symptoms were face discomfort and deformity, deformed jaws, and difficulty with eating or speaking. Typical oral clinical findings comprised gingival fibrosis, nodularity, and hyperpigmentation, as well as atypical teeth (Table 2).

Table 2: Clinical and Oral Findings associated with HGF

Clinical and Oral manifestations
Oral and facial discomfort
Disfigurement of the facial profile
Jaws that have been distorted.
Lip closure was averted.
Lips that are everted, protruded, or both.
There was a noticeable tenseness in the facial muscles.
Cupid's lips is bent in submission.
Difficulties with eating or motor function
Communication and language problems
Ingesting
Gingiva with a thick, leathery, or fibrotic appearance.
Tuberosity/nodularity of the gingival tissue
A darkening of the gingiva
An ulcer in the gingival tissue
Hemorrhaging from the gums

Stippling of the gums
Periodontitis
Pseudo pockets
Calculus releases
The gingiva covers the teeth completely or partly.
Inter teeth/diastema spacing
A suffocating mass of teeth
Teeth erupting from the gums
Teeth partially submerged
Movement of the teeth
Teeth aberrant growth or dystocia
The emergence of permanent or primary teeth that are delayed.
Delays in the resorption or loss of the first teeth
Misaligned teeth
Occlusions with an open bite.
A little mouthful
A constrained range of motion for the mouth
Oral and facial discomfort
Disfigurement of the facial profile

The most common radiologic techniques included intraoral and extraoral X-rays. Preserved, affected, supernumerary, or dental problems, as well as postponed primary tooth root erosion, were all frequent radiographic observations. Bone attachment or degeneration was also prevalent in childhood with HGF.

There was no family history of HGF in 9 of the 29 investigations, but there was a family history of HGF in the other 20 studies. IGF was discovered in four distinct studies: two had severe periodontal disease, one had Zimmemrman-Laband Syndrome (ZLS-1), one had juvenile hyaline fibromatosis (JHF) and the other two had non-syndromic HGF.

In two case studies, recurrence after surgical therapy was found. The duration of the follow-up period for one case study was ten years (Baltacioglu *et al*., 2017), whereas the duration of the follow-up period for the second case study was seven years (Li *et al*., 2021). While one reported two year duration (Tripathi *et al*., 2014).

Case Control Study

The data retrieved 3 case/control studies with 47 mothers and more than 50 children with mean age of 10 year (Kapferer-Seebacher *et al*., 2021; Monteiro *et al*., 2021; Pähkla *et al*., 2010). According to the findings of one investigation, the gingival phenotype was characterised by a broad absence of connected gingiva. All of the children that had this gingival morphology (n = 12) had received the family pathologic variation, while the gum phenotype did not appear in any of the kids who did not possess the parental pathogenic variant (n = 7). Eight of the children who were impacted and none of the children who were not affected stated having trouble bleeding easily. In youngsters, the signs and symptoms of periodontal Ehlers–Danlos syndrome (pEDS) were very sometimes seen. Only two out of twelve children between the ages of eight and thirteen who were afflicted met the clinical criteria for pEDS. A generalised lack of linked gingiva is the one and only clinical finding that is consistent across all affected individuals, including both adults and children. This pathognomonic hallmark of pEDS is also the only finding that is always present. This is significant because getting a timely detection may lead to improved oral hygiene practises throughout infancy, which may be necessary to forestall the premature loss of baby

According to the findings of research, children whose mothers had a condition were more likely to have periodontal illnesses, particularly gingivitis. Additionally, clinical markers of gingival inflammation were found to be more pronounced in this group of children, and oral hygiene was shown to be worse. The percent of ill and well moms were considerably different from one another. Oral pathogens A. actinomycetemcomitans and P. intermedia/nigrescens were the most often detected. It was shown that children who had healthy moms were less likely to harbor germs than children who had mothers who had diseases. Five times as many families shared P. intermedia/nigrescens, between which two families shared A. actinomycetemcomitans. Indicators of periodontal disease in mothers, including as periodontitis, hygiene practices, and dental micro biome, are associated with an increased risk of periodontal disease in children and adolescents.

One more research demonstrated that offspring of parents who have deprived periodontal wellbeing are more probable to have pitiable periodontal wellbeing themselves. The oral health history of an individual's family or parents is an accurate picture of the cumulative hereditary and ecological variables that

underwrite an entity's periodontal eminence and might assist to forecast enduring prospects and the need for preventative therapy. However, there was no correlation found between the presence of periodontitis and factors such as gender, length of time spent at the center for follow-up, education level, and degree of participants' reliance, flossing, or previous periodontal disease in the family in this study (Botero *et al*., 2015).

DISCUSSION

Hereditary gingival fibromatosis, often known as HGF, is a condition that, in the majority of cases, strikes younger juveniles. We carried out a comprehensive literature analysis in order to provide a comprehensive description of the demographics, clinicopathological features, symptomatic and parental linkages, and outcomes of paediatric individuals diagnosed with HGF and other periodontal conditions. During the course of our investigation, we came across a total of 26 papers that included information on 29 HGF children who were under the age of 18 years old. Sixty per cent of patients had extraoral indications of the condition, according to their reports. The vast majority of individuals were found to have severe dental illness. However, around one-third of children are affected by HGF in a form that is considered syndromic. This is because autosomal dominant inheritance was the mode of transmission in the great majority of HGF cases.

Histologically, HGF is defined by a rise in sub mucosal connective tissue that results in gingival fibrous growth. This symptom may be seen in persons with the disease. The connective tissue seems to be highly collagenized, avascular, and devoid of differentiated fibroblasts and infiltrates, the epithelium seems to be thick and hyperkeratosis, with elongated rete ridges. In some areas, acanthosis may result in epithelial hyperplasia, which can be seen in association with chronic inflammation. It is probable that each kind of HGF has a distinct set of histological characteristics.

HGF has been characterised in both syndromic and nonsyndromic types. In about two of the individuals examined, HGF was part of a syndrome juvenile hyaline fibromatosis and the Zimmermann-Laband syndrome were among these disorders. However, we did not notice any of other disorders in any of the individuals whose medical records we studied thoroughly. Though one patient have reported hearing loss.

To diagnose HGF, a clinical examination of the head, neck, and especially the oral cavity is necessary in the vast majority of cases. The gingiva in either the maxillary or mandibular region may become enlarged, and there may also be some deformity of the jaw and teeth. These are the most noticeable signs. Radiographic evidence, such as anomalies in some teeth or bone loss, might provide support to a diagnosis. It is essential to have an accurate diagnosis as soon as possible in order to reduce the risk of gingival irritation and possible attachment loss, both of which may be brought on by difficulties in practising good oral hygiene. In addition, the detection of extraoral symptoms in 61 percent of patients and the hereditary accumulation of HGF may be able to assist in the earlier detection, management, and mitigation of gum disease in children who come from HGF-positive parents.

Gingivectomy, followed by gingivoplasty, was the treatment that was performed on the vast majority of the patients in this comprehensive research. Two of our research revealed the gingival expansion recurrence rate after surgical therapy was administered. Even when HGF has been surgically removed, we believe it is very important to perform long-term follow-up on all of the children who have the condition. This is because less than half of the youngsters were clear of relapse three years after the procedure was performed.

The impact of a parent's micro biome exclusively of women, on the structure of a child's micro biome has been widely examined and discussed in the scientific literature. The impact may be felt in several places of the body, including the skin, stomach, and mouth. In addition, there is a strong association between the similarity of two environments and the level of interaction between the various microbial communities. All of this gives support to the notion that vertical transmission of microbes, i.e., transfer from parents to their offspring, is a crucial way of obtaining oral micro biota from family members who have comparable hygiene, eating, and social interaction practises.

An alteration in the parental microbiome, such as that found in certain infectious disorders, is most likely to be a driving force behind the development of symbiotic relationships between pathogens and their children. In fact, research on the micro biome of the gut has shown that women with particular illnesses, such as diabetes or obesity, pass on a micro biome that is suggestive of these conditions. Using a family-based approach and a longitudinal study design, researchers found that parental periodontitis had a significant impact on the microbiota of their children. Another interesting finding was the microbiota's ability to adapt to environmental changes. This indicates a high degree of heredity.

Both the vulnerability to periodontitis and the family clustering of the disease are connected to the early acquisition of pathogenic microbiota as well as the occurrence of symbiosis in infancy. A longitudinal cohort study with an experimental component discovered that parents who have periodontal disease are more likely to pass on oral germs to their children. The study was conducted to investigate this hypothesis. Compared to parents who did not have periodontitis,

those who had been diagnosed with the gum disease periodontitis were more likely to have children who were infected with bacteria belonging to the genera Filifactor alocis, Porphyromona spp., Aggregatibacter actinomycetemcomitans, Streptococcus parasanguinis, and Fusobacterium nucleatum. These species were in charge of moderating the interactions between the different species. Using these pathogenic pathogens, which have been shown to be reliable, it is possible to properly anticipate the microbial fingerprints of children whose parents have periodontitis. These children are more likely to develop periodontitis themselves. Plaque management has no influence on this pathogenic pattern because the microbiome is so resistant to change once it has been created. Because the condition of the parents has a significant influence on the microbial colonisation patterns of their children and the early acquisition of periodontitis-related species, there is an increased need for increased surveillance and preventative efforts in families of periodontitis patients.

Periodontal disease-related genes and polymorphisms are the focus of a lot of study right present. People with chronic periodontitis may have different genotypes depending on their origin and personal attributes, which may affect their risk of developing the disease. The variations of genes implicated in cytokine production have received much attention, although single-nucleotide polymorphisms have yet to be reliably discovered. While family studies may provide light on the dynamics of extended families, they are unable to separate the effects of genetics from those of the surroundings. Because the expression of genes may be influenced by a variety of external stimuli. DNA methylation or acetylation, as well as chromatin alterations that alter the way the genetic code is read, are all examples of epigenetic modifications that may alter gene expression patterns. Despite this, little is known about the epigenetic processes that regulate the expression of pro- and anti-inflammatory genes, respectively. To help comprehend the variables that influence vulnerability and demographic diversity, epigenetics is a relatively recent idea. It has the ability to offer the critical connection between genotypes, disease manifestations, and external conditions.

LIMITATIONS

According to our knowledge, this is the first comprehensive research undertaken on HGF patients less than 18 years old. When evaluating the findings of our research, it is crucial to keep in mind a number of its limitations. To begin with, we have presented solely English-language content. Due to this, it was unable to properly combine the data for the HGF-specific outcomes; therefore, they could only be presented in terms of their qualitative components.. Instead of objectively quantifying the radiographic assessment rates, we opted to adopt a subjective way to summarise the most often seen radiographic abnormalities in paediatric HGF patients.. Although some research did not cover all key criteria, our conclusions are based on published literature. Histologically, HGF is distinguished from other types of growth factors by the increase in sub mucosal connective tissue it presents. Patients with the disease may display this trait. Connective tissue is collagenized and avascular, but contains few differentiated fibroblasts and a negligible number of inflammatory infiltrates despite the fact that connective tissue is avascular, this is the case. It is possible to see hyperkeratosis characteristics and extended rete ridges in the epithelium. In addition to a prolonged inflammatory response, it is possible that certain instances of anthosis will also be accompanied with epithelial hyperplasia. It is likely that the histological qualities of different types of HGF vary.

CONCLUSION

In both primary and permanent teeth, periodontitis is a condition that produces extensive damage of the periodontal tissues and progresses rapidly. Children of parents with periodontal are thought to be at greater risk of developing the illness due to the aforementioned incidence, and as a result, should be checked and monitored as soon as feasible.

REFERENCES

- Aghili, H., & Goldani Moghadam, M. (2013). Hereditary gingival fibromatosis: a review and a report of a rare case. *Case Reports in Dentistry*, *2013*, 930972. https://doi.org/10.1155/2013/930972
- Arjunkumar, R. (2018). Nanomaterials for the management of periodontal diseases. In *Dental Applications of Nanotechnology* (pp. 203–215). Springer International Publishing.
- Balaji, T. M., Varadarajan, S., Sujatha, G., Muruganandhan, J., Shanmugapriya, R., Raj, A. T., Patil, S., Fageeh, H. I., Fageeh, H. N., Malik, N. H., & Awan, K. H. (2021). Necrotizing periodontal diseases in human immunodeficiency virus-infected patients receiving highly active antiretroviral therapy: A review. *Disease-a-Month: DM*, *67*(9), 101168. https://doi.org/10.1016/j.disamonth.2021.101168
- Baltacioglu, E., Guzeldemir, E., Sukuroglu, E., Yildiz, K., Yuva, P., Aydin, G., & Karacal, N. (2017). Juvenile Hyaline Fibromatosis: A 10-year Follow-up. *Indian Journal of Dermatology*, *62*(2), 210–212. https://doi.org/10.4103/ijd.IJD_166_16
- Botero, J. E., Rösing, C. K., Duque, A., Jaramillo, A., & Contreras, A. (2015). Periodontal disease in children and adolescents of Latin America. *Periodontology 2000*, *67*(1), 34–57. https://doi.org/10.1111/prd.12072
- Chaurasia, A. (2014). Hereditary gingival fibromatosis. *National Journal of Maxillofacial Surgery*, *5*(1), 42–46. https://doi.org/10.4103/0975-5950.140171

- Chen, C., Feng, P., & Slots, J. (2020). Herpesvirus-bacteria synergistic interaction in periodontitis. *Periodontology 2000*, *82*(1), 42–64. https://doi.org/10.1111/prd.12311
- Cunha, J. L. S., Ramos, M. A. C. da C., Regis, D. M., Sanchéz-Romero, C., de Andrade, M. E., Bezerra, B. T., & de Albuquerque-Júnior, R. L. C. (2020). Generalized hereditary gingival fibromatosis in a child: clinical, histopathological and therapeutic aspects. *Autopsy & Case Reports*, *10*(1), e2020140. https://doi.org/10.4322/acr.2020.140
- Curtis, M. A., Diaz, P. I., & Van Dyke, T. E. (2020). The role of the microbiota in periodontal disease. *Periodontology 2000*, *83*(1), 14–25. https://doi.org/10.1111/prd.12296
- Dhadse, P. V., Yeltiwar, R. K., Pandilwar, P. K., & Gosavi, S. R. (2012). Hereditary gingival fibromatosis. *Journal of Indian Society of Periodontology*, *16*(4), 606–609. https://doi.org/10.4103/0972-124X.106930
- Duddu, M. K., Muppa, R., Reddy, G. S. P., & Reddy, P. V. N. (2012). Non syndromic gingival fibromatosis in a mild mental retardation child. *Contemporary Clinical Dentistry*, *3*(Suppl 2), S206-9. https://doi.org/10.4103/0976-237X.101096
- Freitag-Wolf, S., Munz, M., Junge, O., Graetz, C., Jockel-Schneider, Y., Staufenbiel, I., Bruckmann, C., Lieb, W., Franke, A., Loos, B. G., Jepsen, S., Dommisch, H., & Schaefer, A. S. (2021). Sex-specific genetic factors affect the risk of early-onset periodontitis in Europeans. *Journal of Clinical Periodontology*, *48*(11), 1404–1413. https://doi.org/10.1111/jcpe.13538
- Gao, H., Liang, J., Xia, X., Deng, Z., & Zhang, Z. (2020). Unusual clinical and histologic findings in a child with mixed dentition with hereditary gingival fibromatosis: a case report. *Translational Pediatrics*, *9*(1), 74–80. https://doi.org/10.21037/tp.2019.12.01
- Gawron, K., Łazarz-Bartyzel, K., Fertala, A., Plakwicz, P., Potempa, J., & Chomyszyn-Gajewska, M. (2016). Gingival fibromatosis with significant DE Novo formation of fibrotic tissue and a high rate of recurrence. *The American Journal of Case Reports*, *17*, 655–659. https://doi.org/10.12659/ajcr.899997
- *Gingivitis and periodontitis: Overview*. (2020). Institute for Quality and Efficiency in Health Care (IQWiG).
- Gita, B., Chandrasekaran, S., Manoharan, P., & Dembla, G. (2014). Idiopathic gingival fibromatosis associated with progressive hearing loss: A nonfamilial variant of Jones syndrome. *Contemporary Clinical Dentistry*, *5*(2), 260–263. https://doi.org/10.4103/0976-237X.132387
- Japatti, S., Bhatsange, A., Reddy, M., Patil, S., Chidambar, & Waghmare, A. (2013). Bimaxillary unilateral gingival fibromatosis with localized aggressive periodontitis (eating the tooth at the same table). *Contemporary Clinical Dentistry*, *4*(3), 366–370. https://doi.org/10.4103/0976-237X.118348
- Kapferer-Seebacher, I., Oakley-Hannibal, E., Lepperdinger, U., Johnson, D., Ghali, N., Brady, A. F., Sobey, G., Zschocke, J., & van Dijk, F. S. (2021). Prospective clinical investigations of children with periodontal Ehlers-Danlos syndrome identify generalized lack of attached gingiva as a pathognomonic feature. *Genetics in Medicine: Official Journal of the American College of Medical Genetics*, *23*(2), 316–322. https://doi.org/10.1038/s41436-020-00985-y
- Kilian, M., Chapple, I. L. C., Hannig, M., Marsh, P. D., Meuric, V., Pedersen, A. M. L., Tonetti, M. S., Wade, W. G., & Zaura, E. (2016). The oral microbiome - an update for oral healthcare professionals. *British Dental Journal*, *221*(10), 657–666. https://doi.org/10.1038/sj.bdj.2016.865
- Kinane, D. F., Stathopoulou, P. G., & Papapanou, P. N. (2017). Periodontal diseases. *Nature Reviews. Disease Primers*, *3*(1), 17038. https://doi.org/10.1038/nrdp.2017.38
- Könönen, E., Gursoy, M., & Gursoy, U. K. (2019). Periodontitis: A multifaceted disease of tooth-supporting tissues. *Journal of Clinical Medicine*, *8*(8), 1135. https://doi.org/10.3390/jcm8081135
- Li, N., Wang, W., Sun, Y., Wang, H., & Wang, T. (2021). Seven-year follow-up of a patient with hereditary gingival fibromatosis treated with a multidisciplinary approach: case report. *BMC Oral Health*, *21*(1), 473. https://doi.org/10.1186/s12903-021-01830-7
- Loos, B. G., & Van Dyke, T. E. (2020). The role of inflammation and genetics in periodontal disease. *Periodontology 2000*, *83*(1), 26–39. https://doi.org/10.1111/prd.12297
- Manoj, M., Garg, R., & Babu, K. (2017). Unusual presentation of familial gingival fibromatosis among male siblings. *Journal of Indian Society of Periodontology*, *21*(2), 152. https://doi.org/10.4103/jisp.jisp_207_16
- Monteiro, M. F., Altabtbaei, K., Kumar, P. S., Casati, M. Z., Ruiz, K. G. S., Sallum, E. A., Nociti-Junior, F. H., & Casarin, R. C. V. (2021). Parents with periodontitis impact the subgingival colonization of their offspring. *Scientific Reports*, *11*(1), 1357. https://doi.org/10.1038/s41598-020-80372-4
- Muhamad, A.-H., & Watted, N. (n.d.). *Genetics in pediatric dentistry: A review*. Oraljournal.Com. Retrieved 17 June 2022, from http://www.oraljournal.com/pdf/2019/vol5issue3/PartF/5-3-60-557.pdf
- Nath, S., Zilm, P., Jamieson, L., Kapellas, K., Goswami, N., Ketagoda, K., & Weyrich, L. S. (2021). Development and characterization of an oral microbiome transplant among Australians for

the treatment of dental caries and periodontal disease: A study protocol. *PloS One*, *16*(11), e0260433. https://doi.org/10.1371/journal.pone.0260433

- Noorul, A. N., Rajasekar, A., & Ganapathy, D. (2021). Periodontal health in children and adolescents-A comparitive study. *Annals of the Romanian Society for Cell Biology*, 15728–15736. https://www.annalsofrscb.ro/index.php/journal/article/view/5224
- Pähkla, E.-R., Jõgi, E., Nurk, A., Pisarev, H., Koppel, T., Naaber, P., Saag, M., & Lõivukene, K. (2010). Periodontal disease in mothers indicates risk in their children: Risk for children of mothers with periodontitis. *International Journal of Paediatric Dentistry*, *20*(1), 24–30. https://doi.org/10.1111/j.1365-263X.2009.01027.x
- Peeran, S. W., Ramalingam, K., Peeran, S. A., Mugrabi, M. H., & Abdulla, K. A. (2013). Hereditary nonsyndromic gingival fibromatosis: report of family case series. *Case Reports in Dentistry*, *2013*, 835989. https://doi.org/10.1155/2013/835989
- Pego, S.-P.-B., Coletta, R. D., Mendes, D.-C., de Faria, P.-R., Melo-Filho, M. R., Alves, L.-R., & Martelli-Júnior, H. (2015). Hereditary gingival fibromatosis: clinical and ultrastructural features of a new family. *Medicina Oral, Patologia Oral y Cirugia Bucal*, *20*(2), e150-5. https://doi.org/10.4317/medoral.20170
- Pol, D. G., Lobo, T. M., & Pol, S. D. (2016). Idiopathic gingival fibromatosis with asymmetrical presentation and electrosurgical management. *Journal of Indian Society of Periodontology*, *20*(1), 98–102. https://doi.org/10.4103/0972-124X.170831
- Prasad, S. S. R., Radharani, C., Sinha, S., & Kumar, S. K. (2012). Hereditary gingival fibromatosis with distinctive facies. *The Journal of Contemporary Dental Practice*, *13*(6), 892–896. https://doi.org/10.5005/jp-journals-10024-1248
- Saini, A., Singh, M., & Singh, S. C. (2013). A rare case of isolated idiopathic gingival fibromatosis. *Indian Journal of Dental Research: Official Publication of Indian Society for Dental Research*, *24*(1), 139–141. https://doi.org/10.4103/0970-9290.114925
- Sedghi, L. M., Bacino, M., & Kapila, Y. L. (2021). Periodontal disease: The good, the bad, and the unknown. *Frontiers in Cellular and Infection Microbiology*, *11*, 766944. https://doi.org/10.3389/fcimb.2021.766944
- Sharma, S., Goyal, D., Shah, G., & Ray, A. (2012). Familial gingival fibromatosis: A rare case report. *Contemporary Clinical Dentistry*, *3*(Suppl 1), S63-6. https://doi.org/10.4103/0976-237X.95108
- Strzelec, K., Dziedzic, A., Łazarz-Bartyzel, K., Grabiec, A. M., Gutmajster, E., Kaczmarzyk, T., Plakwicz, P., & Gawron, K. (2021). Clinics and genetic background of hereditary gingival fibromatosis. *Orphanet Journal of Rare Diseases*, *16*(1), 492. https://doi.org/10.1186/s13023-021-02104-9
- Tadakamadla, S. K., Tadakamadla, J., Kroon, J., Lalloo, R., & Johnson, N. W. (2020). Effect of family characteristics on periodontal diseases in children and adolescents-A systematic review. *International Journal of Dental Hygiene*, *18*(1), 3–16. https://doi.org/10.1111/idh.12398
- Triantafyllia, V., & Georgios, T. (2018). Periodontal diseases in children and adolescents affected by systemic disorders - A literature review. *International Journal of Oral and Dental Health*, *4*(1). https://doi.org/10.23937/2469-5734/1510055
- Tripathi, A. K., Upadhaya, V., Kumar, V., & Saimbi, C. S. (2014). Hereditary gingival fibromatosis and its management: 2-year follow-up. *Contemporary Clinical Dentistry*, *5*(4), 569–571. https://doi.org/10.4103/0976-237X.142835
- Umrania, V., Reddy, N., Chandrashekhara Rao, D., & Hegde, U. (2016). Hereditary gingival fibromatosis: A report of two cases in the same family. *Journal of the International Clinical Dental Research Organization*, *8*(2), 129. https://doi.org/10.4103/2231-0754.186423
- Yadav, V. S., Chakraborty, S., Tewari, S., & Sharma, R. K. (2013). An unusual case of idiopathic gingival fibromatosis. *Contemporary Clinical Dentistry*, *4*(1), 102–104. https://doi.org/10.4103/0976-237X.111623
- Zheng, Y., Zhang, M., Li, J., Li, Y., Teng, F., Jiang, H., & Du, M. (2018). Comparative analysis of the microbial profiles in supragingival plaque samples obtained from twins with discordant caries phenotypes and their mothers. *Frontiers in Cellular and Infection Microbiology*, *8*, 361. https://doi.org/10.3389/fcimb.2018.00361