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Intrafamilial Transmission of Periodontal Pathogens: A systematic Review

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Abstract Introduction: Periodontitis is a complex multifactorial disease resulting from a dysbiosis of the oral microbiota. Familial aggregation of this condition has been demonstrated, making intrafamilial transmission of periodontopathogenic bacteria possible, particularly Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis. Materials and Methods: A systematic literature search was conducted using three electronic databases (MEDLINE [PubMed], Scopus, and Google Scholar). The search included human studies published in English between January 2012 and September 2025 that investigated the intrafamilial transmission of periodontal pathogens, specifically Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis. The preparation of this literature review followed the guidelines of the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement. Results: Ten studies were included in this systematic review, comprising four observational studies, four case–control studies, one pilot study, and one case report. These studies suggest the existence of both horizontal and vertical transmission of periodontal pathogens (A. actinomycetemcomitans and P. gingivalis). Conclusion: Intra-familial transmission of periodontal pathogens has been discussed mainly in studies focusing on vertical transmission. To better address these issues, larger-scale studies are required. The mere presence of periodontopathogenic bacteria does not appear to be sufficient to trigger periodontal disease. Many questions remain unanswered regarding the exact relationship between this transmission and the development of periodontitis.

Keywords: *periodontitis; intrafamilial transmission; Aggregatibacter actinomycetemcomitans; Porphyromonas gingivalis; familial aggregation; vertical transmission; horizontal transmission; JP2 clone; systematic review*

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1. Introduction

Periodontitis is a chronic inflammatory disease that leads to the destruction of the tooth-supporting apparatus, resulting in discomfort, masticatory dysfunction, and a significant reduction in quality of life [1]. Beyond its local consequences, periodontitis contributes to systemic inflammation and has been associated with cardiovascular diseases, diabetes, and adverse pregnancy outcomes [2]. Its etiology is complex and multifactorial, with the subgingival biofilm playing a central role [3].

Although more than 700 microbial species have been identified in the oral cavity, only a limited subset exhibits high virulence and is strongly implicated in the onset and progression of periodontal destruction. Among these, Aggregatibacter actinomycetemcomitans (Aa) and Porphyromonas gingivalis [Pg] are recognized as major periodontal pathogens [4].

These microorganisms grow within structured biofilms embedded in an extracellular glycocalyx matrix, which provides protection and reduces their susceptibility to antimicrobial agents [5,6]. Recent epidemiological data also indicate that aggressive periodontitis is one of the most prevalent oral diseases worldwide. In Morocco, the situation is particularly concerning: a 2022 study conducted among adolescents aged 12–18 reported a periodontitis prevalence of 12.3%, including 5% presenting with aggressive forms [7].

Although periodontitis is mainly considered the result of a dysbiosis within the subgingival biofilm, certain species show a particularly strong association with the disease. This is the case for Aggregatibacter actinomycetemcomitans (Aa), which is notably involved in aggressive periodontitis in adolescents. Among its genotypes, the JP2 clone is distinguished by enhanced virulence [8]. This variant, characterized by a 530-base pair deletion in the promoter region of the leukotoxin gene, results in markedly increased leukotoxin production, enhancing its ability to evade host immune defenses. The JP2 clone is particularly prevalent in North and West Africa, where its presence constitutes a significant risk factor for early attachment loss in adolescents. Identification of this specific Aa subtype is therefore essential for risk assessment in populations where the clone is endemic [8,9].

Despite the well-established role of these pathogens, the

intrafamilial transmission of periodontal bacteria and the familial aggregation of periodontitis remain insufficiently understood [7]. Observations suggest that certain families may exhibit a predisposition to periodontal disease, possibly linked to the intergenerational transmission of periodontopathogenic species such as Aa and Pg [6,12]. Understanding these mechanisms is essential for improving prevention strategies.

The aim of this systematic review is to analyze the role of family in periodontitis by investigating the intrafamilial transmission of key pathogens, including Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis, examining the familial aggregation and predisposition to periodontal disease, and exploring the intergenerational transmission of periodontopathogenic bacteria. By clarifying how familial, genetic, and environmental factors contribute to the development and progression of periodontitis, this review aims to provide insights for early risk assessment and preventive strategies.

The synthesis of knowledge presented in this review aims to update our understanding of these phenomena and underscore the importance of family-related factors in the prevention and management of periodontal diseases.

2. Materials and Methods

The present manuscript was written according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) checklist.

Information sources and search strategy

Literature search was conducted purely electronically in three main databases: MEDLINE (via PubMed), Scopus, and Google Scholar. The search terms were selected using MeSH (Medical Subject Headings) and equivalent terms to cover three main concepts: Periodontal Pathogens (e.g., Aa., Pg.), Transmission and Familial Aggregation (e.g., "intrafamilial transmission," "parent-child transmission"), and Periodontal Diseases and Colonization. Boolean operators (AND) were used. Examples of search equations included: "Family transmission AND Aggregatibacter actinomycetemcomitans," and "Family aggregation AND periodontitis". A manual search of the reference lists of included articles was also performed.

The Boolean equation used to query PubMed was as follows:

- "Family transmission" [All Fields] AND "Aggregatibacter actinomycetemcomitans" [All Fields].
- "Family transmission" [All Fields] AND "Porphyromonas gingivalis" [All Fields].
- "Family transmission" [All Fields] AND "Periodontitis" [All Fields].
- "Family aggregation" [All Fields] AND "Periodontitis" [All Fields].

The search in Scopus was performed using the following Boolean equations:

- Family transmission AND Aggregatibacter actinomycetemcomitans
- Family transmission AND Porphyromonas gingivalis
- Family transmission AND periodontitis
- Family aggregation AND periodontitis

The search in Google Scholar was carried out using the following Boolean equations:

- Family transmission AND Aggregatibacter actinomycetemcomitans
- Family transmission AND Porphyromonas gingivalis
- Family transmission AND periodontitis
- Family aggregation AND periodontitis

Eligibility criteria

The eligibility criteria were defined using the PICO format:

Table 1. Summary of PICO Elements in Included Studies

PICO Criteria	Definition
P (Population)	Human families (children, adolescents, and adults) assessed for periodontopathogens or periodontal diagnosis, including individuals from families or households where intrafamilial relationships are documented.
I (Exposure)	Intrafamilial contact with carriers of *Aggregatibacter actinomycetemcomitans* (A.a.) or *Porphyromonas gingivalis* (Pg.), based on microbiological detection in one or more family members.
C (Comparison)	Families without documented carriers or general populations without documented familial exposure, or studies comparing intra-family members versus unrelated individuals.
O (Outcomes)	Microbiological outcomes (colonization, prevalence, concordance of A.a. and P.g. among family members) and clinical outcomes (periodontal diagnosis, periodontal indices, disease severity, familial aggregation).

Inclusion Criteria:

- Study Types: Only observational studies (cross-sectional, case–control, cohort), family or pedigree studies, and microbiological investigations with documented familial relationships were included.
- Language: English.
- Timeframe: from 2012 to now.

Exclusion Criteria:

- Experimental studies on models or animals.
- Articles published prior to 2012.
- Articles that did not meet the objectives of the review.

Study Selection and Data Abstraction

The selection process was conducted in multiple stages (duplicate removal, screening of titles and abstracts, full-text review). Two reviewers independently screened the articles, and any disagreements were resolved through discussion; if consensus was not reached, a third reviewer was consulted.

Data extraction was performed by two individuals independently using a standardized, pilot-tested form. The variables collected included: reference (author, year, country) and study design, population characteristics, microbiological detection method and sample type, family structure (relationship type, cohabitation), reported statistics (prevalence, OR, RR, concordance), confounder adjustments, and risk of bias information.

Assessment of quality and risk of bias

The quality assessment and evaluation of the risk of bias were conducted independently by two reviewers in accordance with the recommendations of the Cochrane Handbook for Systematic Reviews of Interventions. Any discrepancies were resolved through discussion or

consultation with a third reviewer.

Risk of Bias Tools Used:

Observational studies (cohort, case–control, and cross-sectional): the Newcastle–Ottawa Scale (NOS) was applied to evaluate methodological quality.

Diagnostic or molecular studies: an adapted STARD checklist or another appropriate methodological appraisal tool was used, depending on the nature of the study.

Domains of Bias Assessed:

- **Selection bias:** evaluated through the adequacy of participant selection methods, including sequence generation and allocation concealment when applicable
- **Performance bias:** assessed by determining whether participants and/or caregivers were blinded to the study conditions.
- **Detection bias:** evaluated based on the blinding of outcome assessors to minimize differential outcome assessment.
- **Attrition bias:** considered when there were withdrawals, exclusions, or incomplete outcome data that could affect study validity.
- **Selective reporting bias:** assessed by verifying the availability of study protocols and ensuring that all prespecified outcomes were reported.

Following the quality assessment, each study was classified into one of three categories:

Low risk of bias: when all relevant quality criteria were fulfilled.

High risk of bias: when several criteria were not met, indicating methodological limitations.

Unclear risk of bias: when one or more criteria lacked sufficient information for judgment.

3. Results

Study selection

Study selection proceeded as illustrated in the PRISMA flow diagram. Records identified through database searching included Google Scholar (n = 4,031), Scopus (n = 259), and Medline/PubMed (n = 175). After removal of duplicates, 8,503 records remained for screening.

Titles and abstracts of 102 records were screened, leading to the exclusion of 83 articles that did not meet the predefined eligibility criteria. The full texts of the remaining 19 articles were assessed for eligibility, and 9 were excluded with documented reasons. Ultimately, the studies that fulfilled all inclusion criteria were retained and incorporated into the qualitative synthesis. https://guides.temple.edu/c.php?g=769207&p=9747455

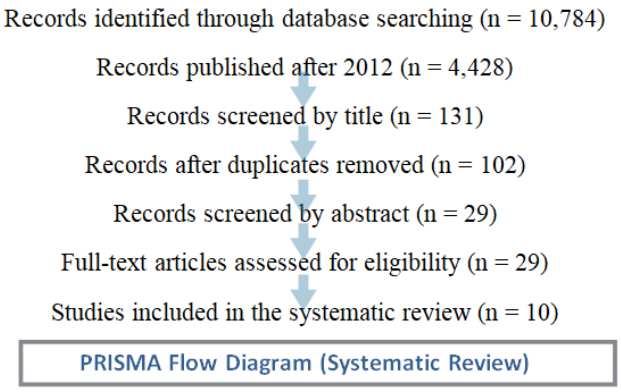


Figure 1. PRISMA Flow Diagram Showing the Study Selection Process

Study characteristics

A total of 10 studies were included. The types of studies included were: 4 Case-Control studies, 4 Observational studies (including cross-sectional), 1 Pilot study, and 1 Case report. The primary detection methods were DNA probes and the Polymerase Chain Reaction (PCR).

The characteristics of the included studies are detailed in the table below:

Risk of bias in included studies

The quality assessment of included studies showed that 8 studies were judged to be at Low risk of bias. Two studies (Díaz-Faes et al., 2022, and Al Yahfoufi Z., 2017) were judged to be at High risk of bias. The high risk of bias was often attributed to inadequate randomization and/or incomplete follow-up. The assessment focused on selection bias, performance bias, detection bias, attrition bias, and selective reporting bias.

Table 2. Synthesis of Results by PICO Elements

No	Author (Year)	Country	Study Type	P (Population)	I (Exposure)	C (Comparison)	O (Outcomes)	Principal Results
1	Díaz-Faes et al. (2022)	Spain	Pilot Study	Three families (12 participants) with aggressive/chronic periodontitis or periodontal health.	Subgingival microbial community analysis (16S rRNA sequencing).	Members affected vs. non-affected by periodontitis.	Structure and function of subgingival microbial communities; intrafamilial transmission.	**No obvious intrafamilial transmission** of periodontitis-related bacterial genera revealed.
2	Reis et al. (2021)	Brazil	Case Report	2 children (age 5 and 9) with early periodontal disease and their parents.	Assessment of familial aggregation factor; detection of Aa, Pg, Tf, Fn via PCR.	Parent's periodontal status.	Early diagnosis; relation with parental status; bacterial concentrations.	**Aa concentrations were comparable between parents and children**. Transmission of Aa and Pg can vary.
3	Adhikari K et al. (2018)	Nepal	Cross-sectional Observational Study	46 pregnant patients with chronic periodontitis and their newborns.	Vertical transmission via maternal saliva sampling.	Implicit: Healthy newborn status/no transmission.	Quantification of P.gingivali (Pg) DNA copies via real-time PCR.	**Significant correlation** between Pg in maternal saliva and newborn saliva. P.gingivali is transmitted vertically.

4	Al Yahfoufi Z. (2017)	Lebanon	Cross-sectional Observational Study	18 individuals from 5 Lebanese families (ages 5-55).	Presence of Pg, Pi, and Aa using DNA probe.	Implicit: Individuals without these pathogens.	Clinical destruction and microbiological similarity (Pi, Pg, Aa prevalence).	**Microbiological similarity** found between children and their mothers.
5	Monteiro et al. (2015)	Brazil	Case-Control Study	108 people from 30 families (30 couples, 48 children).	Clinical and subgingival microbial evaluation (Aa, Pg, Tf) using PCR.	Families with periodontally healthy parents (Témoins).	Frequency of detection and quantities of Aa, Pg, Tf.	**Higher detection frequency and quantities of Aa and Pg** in children from aggressive periodontitis families.
6	Feng et al. (2015)	China	Cross-sectional Observational Study	103 participants from 41 families (aggressive periodontitis patients and family members).	Transmission of 8 periodontal microorganisms; detection by PCR; Pg FimA genotype distribution.	Implicit: Non-aggressive periodontitis families.	Percentage of transmission; genotype sharing of Pg FimA.	**Aa detection highly coherent** among family members. Most patients shared the **same Pg genotype** with relatives.
7	Balasouli et al. (2015)	Greece	Cross-sectional Observational Study	108 Greek children and their 83 mothers.	Transmission of Aa from mothers to children; prevalence and serotype distribution via PCR.	Implicit: Children of Aa-negative mothers.	Prevalence and serotype distribution (JP2 clone exclusion).	Aa found in four mother/child pairs. In three pairs, isolates shared the **same genotype**. No JP2 clone detected.
8	Monteiro et al. (2014)	Brazil	Case-Control Study	100 participants: 15 Generalized Aggressive Periodontitis (GAP) families and 15 healthy families.	Evaluation of salivary microbial profile (Aa, Pg, Tf) via PCR.	Families with periodontally healthy parents (Témoins).	Salivary carriage/risk of colonization (Odds Ratio).	Children with an Aa-positive parent had **16.3 times higher risk** of infection by Aa. Aa detection higher in GAP children (90%) vs. healthy children (45%).
9	Martelli et al. (2012)	Italy	Cross-sectional Observational Study	9 couples married for 10 years.	Evaluation of horizontal transmission; PCR for Pg, Td, Tf, Fn, Pi, Aa.	Implicit: Unrelated individuals.	Comparison of periodontal microbiological patterns; Pg FimA genotype detection.	**Horizontal transmission was confirmed** by detecting the **same Pg FimA genotype in 55% of couples**.
10	Belcheva et al. (2012)	Bulgaria	Case-Control Study	62 parents (31 diseased, 31 healthy) and their 62 children.	Comparison of Pg prevalence between children of diseased parents and healthy parents via Culture/PCR.	Parents with healthy periodontal status (Groupe C).	Prevalence of P.gingivali .	**No statistically significant difference** (P>0.05) found in Pg prevalence between children of diseased parents and children of healthy parents.

Results of analysis

The synthesis of the 10 included studies established evidence for the intrafamilial transmission of key periodontal pathogens, Aa and Pg. Studies focusing on vertical transmission emphasized that the saliva is the most important vehicle for oral microorganism transfer.

- **Vertical Transmission (Parent-Child)**: The transmission of Aa is significant, with children having a risk 16.3 times higher of being colonized if a parent is positive for Aa. Children in generalized aggressive periodontitis (GAP) families showed a higher detection rate of Aa (90%) compared to healthy families (45%). Studies also showed that P.gingivalis DNA copies in newborns ‘saliva were equivalent to those in the mothers ‘saliva, providing clear evidence of vertical transmission of Pg.
- **Horizontal Transmission** (Spousal/Cohabitants): Horizontal transmission of periodontal pathogens was confirmed among spouses. The presence of periodontitis in one partner is considered a strong indicator of colonization risk for the spouse. Estimated horizontal transmission rates range between 14% and 60% for Aa and 30% and 75% for Pg.

Table 2. Articles included, classified according to author name, title, and PICO framework

4. Discussion

Summary of Evidence (PRISMA Item 24)

This systematic review aimed to examine the familial aggregation of periodontitis and evaluate the evidence for the intrafamilial transmission of Aa and Pg.

1. Microbiology of Periodontitis: Role of Key Pathogens

The etiology of periodontal disease is complex but is intrinsically linked to the presence of a dysbiotic subgingival biofilm [9]. The two major pathogens

highlighted in this review are Aggregatibacter actinomycetemcomitans (Aa) and Porphyromonas gingivalis (Pg), whose presence is strongly associated with the onset and progression of periodontitis [10,11,15].

A. actinomycetemcomitans: The Role of the JP2 Clone

Aa is a Gram-negative facultative anaerobic bacillus particularly implicated in localized aggressive periodontitis (LAP). One of the most significant findings regarding its virulence is the identification of a specific variant: the JP2 clone [10,12]. This clone, highly prevalent among Moroccan adolescents, is characterized by a 530-base pair deletion in the promoter region of the leukotoxin gene, resulting in markedly increased leukotoxin activity and an enhanced ability to interfere with innate immune defenses [35]. Longitudinal studies conducted in Morocco have demonstrated that individuals carrying the JP2 clone alone have a relative risk of clinical attachment loss of 18.0, markedly higher than those carrying non-JP2 clones (RR 3.0). The high pathogenic potential of the JP2 clone makes it a crucial marker for early risk assessment in populations where it is endemic [32].

Porphyromonas gingivalis: Virulence Factors and Host Response

Pg, a Gram-negative obligate anaerobe, is considered a primary etiologic agent of periodontitis. Its virulence factors, including fimbriae and proteases such as gingipains, play key roles in subverting host immune defenses [4]. Lipopolysaccharide (Pg-LPS] is another major component contributing to inflammation. The tissue-destructive mechanisms induced by Pg involve programmed cell death (PCD) in gingival epithelial cells, regulated by pathogen recognition receptors (PRRs) such as NLRP6. NLRP6 expression is upregulated in the epithelium of periodontitis patients, leading to pyroptosis [36,37]. Notably, this response requires the whole bacterium: stimulation of epithelial cells with Pg-LPS alone does not activate NLRP6 expression at the mRNA or protein levels, nor does it induce pyroptosis [38]. This suggests that the activation of this inflammatory pathway depends on additional virulence factors or the combined action of the intact bacterium rather than on LPS alone [40].

- Intrafamilial Transmission and Biogeographic Interactions

The review included ten studies, primarily using PCR and DNA probes, which confirmed both horizontal and vertical transmission of periodontal pathogens (Aa and Pg) [44].

➢ **Vertical Transmission (Parent-to-Child)**

Vertical transmission from mother to child is considered the main route of acquisition for these microorganisms [45]. Several studies support this concept:

A significant correlation was observed between the amount of Pg in the saliva of mothers with chronic periodontitis and that of their newborns, sampled within 48 hours after birth, excluding other early environmental factors [61,62].

For Aa, children with a parent carrying the bacterium had a 16.3-fold higher risk of infection. Saliva is identified as the primary vehicle for the transmission of oral microorganisms [63,64].

➢ **Horizontal Transmission (Spouses and Cohabitants)**

Horizontal transmission, particularly between spouses, has also been documented. Statistical analyses reveal a high correlation between the microbiological profiles of married couples [65,70]. The same FimA genotype of Pg was detected in 55% of couples studied. FimA, a fimbrial protein, is a virulence factor whose genotype is closely linked to the pathogenic potential and inter-spousal transmission of Pg [66]. Additionally, the sharing of food and utensils has been identified as a potential route for horizontal transmission of Aa within families.

Local Microbial Interactions: Spatial Competition

Beyond interpersonal transmission, the survival and pathogenic potential of Aa and Pg also depend on their spatial interactions within subgingival biofilms at the micro- and nanoscale [67]. Although Aa and Pg may co-occur at destructive periodontal sites, biochemical competition exists:

In vitro studies suggest that Pg gingipains can promote the detachment of Aa from hard surfaces and inhibit its aggregation within the biofilm, indicating that Pg may actively exclude Aa from specific ecological niches [78,81].

Conversely, Aa exhibits complex ”fight-and-flight” mechanisms in response to other commensals such as Streptococcus gordonii. Aa utilizes lactate produced by S. gordonii for growth while producing dispersin B and catalases to neutralize H_2O_2 generated by S. gordonii, maintaining a safe distance while maximizing nutritional benefits [70].

4.1. Limitations and Perspectives (PRISMA Item 25)

This systematic review, although limited by a final corpus of 10 studies over ten years, confirms the crucial role of familial factors in the etiology and prevention of periodontal diseases. Eight studies presented a low risk of bias, while two (Díaz-Faes et al. and Al Yahfoufi Z.) had a high risk due to inadequate randomization and/or incomplete follow-up [66].

In conclusion, the presence of periodontal pathogens (Aa and Pg) within families appears necessary but not sufficient to trigger periodontal disease. Genetic, environmental, and behavioral factors are essential co-factors. The complex pathogenic roles of Aa and Pg are further illustrated by highly virulent variants such as the JP2 clone and the requirement of host innate immune activation, which is not triggered by Pg-LPS alone [55]. Larger studies, particularly in Morocco—given the high prevalence of aggressive periodontitis and the Aa JP2 clone—are needed to better understand transmission mechanisms and to develop effective prevention strategies [54]. Studying the complex microbial interactions, including the antagonistic effect of Pg gingipains on Aa, is essential for decoding how these pathogens coexist, compete, or synergize to disrupt microbiota homeostasis and initiate disease [63].

Clinical implication & Future Research (PRISMA Item 26)

Clinical Implications:

- Given the proven vertical and horizontal transmission risks, prevention must target not only patients with periodontitis but also their entourage.
- Children whose parents suffer from periodontitis are considered high risk and must be evaluated and

monitored as early as possible. Early diagnosis is key to minimizing attachment loss and ensuring predictable treatment outcomes.

- The dental professional must conduct a detailed anamnesis (history taking) to identify potential risk factors, including family history and habits (e.g., sharing utensils). Measures such as rigorous oral hygiene and the use of individual cutlery should be recommended to minimize bacterial transmission.
- Saliva testing for periodontopathogenic bacteria (Aa and Pg) is particularly important in children, serving as a risk indicator for transmission and subsequent disease development.

Future Research: There remain many unanswered questions regarding the exact relationship between pathogen transmission and the development of periodontitis. It is essential to conduct multiple studies on larger populations to fully understand the transmission mechanisms and develop effective prevention and treatment strategies. Given the high prevalence of aggressive periodontitis and the highly leukotoxic JP2 clone of Aa in populations such as Moroccan adolescents, further large-scale studies should be conducted in these specific areas.

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