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RESEARCH ARTICLE

Molecular Characterization and Virulence Factors of *Streptococcus Mutans* Isolated From Dental Caries in Type 2 Diabetic Patients

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ABSTRACT

Individuals with type 2 diabetes are particularly susceptible to frequent and uncontrolled dental caries. *Streptococcus mutans*, a main cause of dental caries, impacts millions each year and presents significant oral health challenges. Although extensively studied, the bacterium's pathogenesis remains incompletely understood, underscoring the need for further research. This study aimed to isolate and analyze the molecular characteristics of *Streptococcus mutans* in patients with type 2 diabetes. It explored the bacterium's capacity to infect, survive, and endure acidic oral environments. Samples were taken from 50 individuals with both type 2 diabetes and dental caries, 50 with dental plaque, and 20 healthy donors. Bacterial identification used Gram staining and *16S rRNA* primer-based analysis with primers designed via Geneious Prime software. Virulence factors *dexA, ldh*, and *atpD* were also assessed. Thirty *Streptococcus mutans* samples were found, with 17 from individuals with both diabetes and caries, and 13 from those with caries alone. All isolates carried the *dexA* gene, 50% had the *atpD* gene, and 30% had the *ldh* gene. Four novel isolates from Baghdad, Iraq, were registered as PQ796766, PQ796767, PQ796768, and PQ796769. These findings reveal that *Streptococcus mutans* exhibits significant virulence and genetic diversity, with identified virulence genes providing insight into its pathogenic mechanisms and suggesting potential strategies for preventing and treating dental caries in diabetic individuals.

Keywords: Acid tolerance, Phylogenetic analysis, 16S rRNA, Streptococcus mutans, Type 2 diabetes

Introduction

Oral health depends on the balance of microbial communities, with oral diseases occurring when harmful species outgrow normal bacteria.[1] Saliva acts as a key defense by using its mix of proteins, cytokines, and immunoglobulins to support both innate and adaptive immune responses. Its diverse functions highlight its importance in promoting health through immune protection, digestion, and diagnostics. Saliva helps prevent bacterial adhesion, maintains microbial balance, and activates defenses when faced with disease.[2]

The oral microbiome can transition from a healthy to a diseased state through destructive host-pathogen interactions, as described by the "bacterial imbalance hypothesis". Although the underlying causes of this shift remain uncertain, research indicates that pH alterations may contribute to the imbalance.[3] This disruption has been associated with various systemic conditions, including diabetes, atherosclerosis, Alzheimer's disease, and head and neck cancers.[4] Additionally, the Inflammation-Mediated Multi-Microbiome Emergence and Exacerbation of Biotic Imbalance (IMPEDE) hypothesis, introduced by Van Dijk et al. in 2020, posits that the initial phase of caries development involves the host's innate immune response striving to maintain oral health symbiosis during the early stages of periodontitis. If this immune response fails to restore balance within

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the dental cavity, pathogenic microbes can progress to periodontitis. To date, clinical evidence linking periodontitis to systemic diseases remains limited.[5]

Type 2 diabetes mellitus (T2DM) is associated with various complications, including those arising from periodontitis. These complications include xerostomia (reduced saliva production), tooth decay, gum inflammation, periodontal disease, increased risk of oral infections, burning mouth syndrome, altered taste, and slower wound healing.[6] A 2023 study by Lee et al. found changes in the oral microbiota of patients with T2DM, showing diverse microbial patterns with gram-positive streptococci and staphylococci being most common.[7] Diabetes, a common endocrine disorder, can cause acute and chronic complications if left untreated.[8,9] Studies indicate that poorly controlled diabetes ($HbA_1c \geq 8.0\%$) is strongly linked to a higher prevalence of dental caries. Periodontal disorders are also recognized as a common complication of diabetes.[10–12] However, the specific mechanisms or combinations of factors directly contributing to complications in target tissues remain unclear.[13]

The Viridans group streptococci are part of the normal flora but can act as opportunistic pathogens under certain conditions. Immunocompromised individuals are particularly susceptible to infections caused by these bacteria, including infective endocarditis and bacteremia.[14] *S. mutans*, a key early colonizer of dental caries, is a Gram-positive, oval-shaped bacterium that often forms pairs or chains. Its thick cell wall, composed of peptidoglycan and teichoic acid, provides hardness and prevents the osmolality process, which leads to lysis. The bacterium also features a polysaccharide capsule with glucose dextran as its structural unit. *Streptococcus mutans* is a non-motile bacterium, devoid of flagella and cilia. It adheres firmly to the tooth surface by producing extracellular polysaccharides from sucrose, which are critical for biofilm formation, commonly known as dental plaque. This organism thrives at 37°C, and is a facultative anaerobe capable of thriving in both aerobic and anaerobic conditions. It is both acidophilic (growing well in acidic environments) and acidogenic (producing acid).[15–17]

The biofilm of *Streptococcus mutans*, composed of extracellular polysaccharides, proteins, and environmental DNA (eDNA), plays a significant role in the development of dental caries and contributes to its virulence, leading to infections and periodontitis.[18,19] Recent studies have confirmed that the most common conditions affecting cariogenic infections are factors linked to adhesion, acid production, and acid resistance.[20] As a result, highly acidogenic, industrialized short-chain acids soften the hard tissues of dental.[21] In addition, *Streptococcus mutans* has a high tolerance to acidic environments and thus affects other microorganisms in the presence of fermentable carbohydrates, which leads to a lower pH.[22] The ability of *Streptococcus mutans* to survive in low pH is due to the maintenance of the transmembrane pH gradient, i.e., ΔpH. Proton translocation of F^1F^0 ATPase can upregulate the extrudability of $H+$, which then causes the external environment to become more acidic.[23] The gene responsible for encoding the $F^{\circ}F^1$-ATPase system is the *atpD* gene.[24] It plays a role in the acid tolerance mechanism by secreting protons from the cytoplasm.[25]

Continuous exposure of fermentable carbohydrates by the host results in low pH fluctuations, which are the primary cause of dental caries caused by *Streptococcus mutans*. The acid produced as a final product promotes the demineralization of dental enamel and inhibits the remineralization process.[26]

The external sugar source through the diet may promote the possibility of a virulence mechanism.[22] Improving oral health in older people can play an effective role in better managing blood sugar levels among diabetic patients, a factor that has historically received little attention.[11] Periodontal disorders are also a common complication of diabetes. However, the specific mechanisms or combinations of factors directly contributing to these complications in the target tissues remain unclear. The unclear mechanisms underlying the relationship between diabetes and periodontal disorders underscore the necessity for additional research to elucidate how diabetes influences these disorders and their effects on target tissues. Further research is crucial to elucidate the mechanisms underlying the association between diabetes and periodontal disorders. The present study examined the molecular traits of *Streptococcus mutans* from diabetic patients with dental caries, focusing on how uncontrolled type 2 diabetes enhances the bacterium's ability to infect, survive, and resist acidity in the mouth.

Materials and methods

Samples collection

The study was approved by the Iraqi Ministry of Health and Environment (approval number: 2023178) and involved clinical samples from patients with carious anterior and posterior teeth, collected with sterile cotton swabs and transport media. Participants included 120 individuals aged 15 to 70, of both genders, who provided signed ethical consent (or parental consent for minors) under the supervision of the dentist. The study compared 50 type 2 diabetic patients with dental caries, 50 non-diabetic patients with dental caries, and 20 healthy donors. Samples were taken at Al-Yarmouk Teaching Hospital in

Baghdad from patients who were advised not to clean their teeth beforehand. Dental exams were conducted with a plain oral mirror under the dentist's supervision, and swabs were taken from decayed areas using sterile cotton swabs with transport medium to preserve bacteria. The study ran from October 2023 to July 2024 and excluded individuals with conditions like Type 1 diabetes, cancer, respiratory illnesses, or those undergoing temporary medical treatments to avoid interference conditions.

Morphological characterization

The isolation and characterization of *S. mutans* involved classical microbiological techniques. Tooth decay sites were swabbed with sterile cotton, and samples were cultured on Mitis Salivarius Agar (MSA), which is a semi-selective medium with 15% sucrose, 1% agar, and 1% potassium telluride solution. The plates were incubated anaerobically at 37˚C with 5% CO_2 for 48 hours. Initial identification was based on the appearance of colonies on MSA and blood agar, particularly their hemolysis patterns. To purify the isolates, samples were cultured on Mitis Salivarius Bacitracin Agar (MSBA) with 0.2 units/ml of bacitracin, while potassium tellurite was added to inhibit non-target bacteria. *S. mutans* was identified using Gram staining, and isolates were subsequently cultured on Brain Heart Infusion (BHI) agar to confirm the diagnosis. Additionally, the *16S rRNA* gene was employed to validate the detection of *S. mutans*.[27,28]

The identification of S. mutans by 16S rRNA

Genomic DNA was isolated following the manufacturer's protocol using a Mini Kit from Korea Favorgen (Thailand). The extracted DNA was subsequently stored at -40 ˚C for use in the polymerase chain reaction (PCR) step.[29] The concentration and purity of DNA were determined using the Qubit™ dsDNA HS Assay Kit from Thermo Fisher®(USA), following the manufacturer's instructions.[30] Using PCR, the 16S ribosomal RNA (rRNA) gene was detected with the primers designed via the National Center for Biotechnology Information (NCBI) primer design tool. PCR reactions were prepared using the OneTaq®2X Master Mix kit (NEB®, England). The study employed specific primers from Macrogen®(Korea) targeting the *16S rRNA* gene (gene sequence and temperature), which are given in Table 1.

The amplification of genes encoding the virulence factors of S. mutans by using a thermal cycler assay

Primers were designed to identify genes associated with biofilm formation, acid tolerance, and acid production, specifically targeting the *dexA*, *atpD*, and *ldh*, with their sequences detailed in Table 1. These primers were specifically designed and validated using Geneious Prime software to exclude false pairs, and the amplified *16S rRNA* DNA (PCR products) was sequenced and analyzed by Macrogen-Korea for Sanger sequencing of the targeted *16S rRNA* region. The phylogenetic analysis of four isolates was conducted through Sanger sequencing. Comparing local and reference isolates. The neighbor-joining method generated results, and sequence differences were identified via multiple alignments in Geneious Prime software.

Statistical analysis

SPSS 2027 and GraphPad Prism Version 9.5.1. Paired comparisons of taxa in samples were conducted using the Chi-squared test, with a significance level set at $P < 0.05$ and $P < 0.01$ for highly significant.

Results

In this study, isolates were obtained from 48 males (40%) and 72 females (60%). Initial identification was performed based on the morphological characteristics of the isolates on a selective medium. *S. mutans* exhibited distinct phenotypic features, including a glassy appearance, convex surface, pale-blue coloration, and spherical or oval morphology, with a frosted texture on MSBA medium. Out of 120 samples collected, 44 (36.67%) tested positive based on phenotypic and chemical properties. Notably, all samples were catalase-negative Fig. 1.

Detection of 16S rRNA gene using conventional PCR

The polymerase chain reaction (PCR) technique demonstrated the presence of the *16s rRNA* gene in 30 clinical samples. This study designed a new primer for the *16S rRNA* gene and tested by Geneious Prime software. Fig. 1 presents the detection results of *16S rRNA* gene across the three study groups: Group 1 (patients with type 2 diabetes and caries) exhibited the highest rate of gene presence (34%) (17/50), while Group 2 (patients with caries only) showed a lower rate (26%) (13/50). In contrast, Group 3 (healthy controls) demonstrated a much lower rate (0%) (0/20). A chi-squared test was conducted to compare the detection rates of *S. mutans* (*16S rRNA* gene) among the three study groups. The results showed a statistically significant difference between the groups (Chi-square = 8.857, $p < 0.001$), where the detection rate was higher in Group 1 (34%) and Group 2 (26%)

Table 1. The table shows the gene sequences, product size, annealing temperature, and the primer source.

Name of gene	Sequence	Product size	TM(°C)	Source
16S rRNA	F 5'-GGGGATAACTATTGGAAACGA -'3 R 5'-CTAGCCTTTTACTCCAGACT -'3	(479bp, 477bp)	(54°c)	Newly Designed
dexA	F 5'-TATGCTGCTATTGGAGGTTC-'3 R 5'-AAGGTTGAGCAATTGAATCG-'3	(1272 bp)	(48°c)	[30]
atpD Designed	F 5'-AGAATTGTTCTTGAAGTTGCC-'3 R 5'-AGTATTCAGCGATGGTAAGAC-'3	(583bp)	(48°c)	Newly
ldh Designed	F 5'- TCAGAATTTGCAGTATGGTCT-'3 R 5'- TCTTTTGTTTTTCAGCATCGT-'3	(355 bp)	(48°c)	Newly

TM=annealing Temperature, °= degree, C=Celsius, bp=base pair.

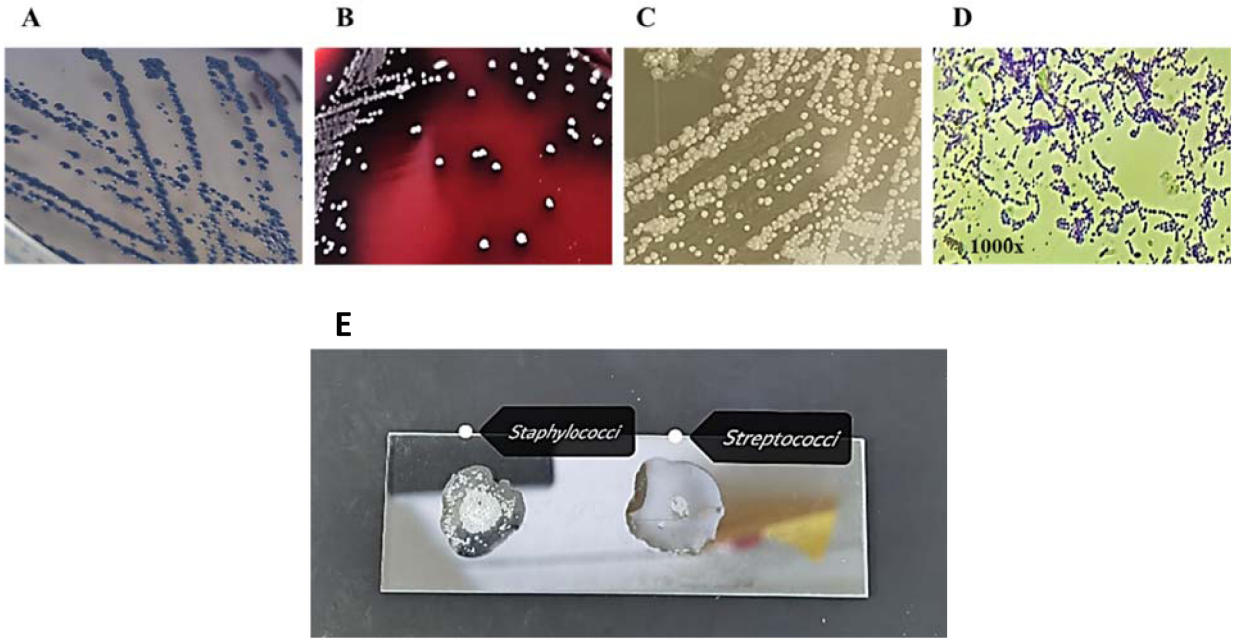

Fig. 1. Illustrates the colonies of *Streptococcus mutans* observed on different media: (A) Mitis Salivarius agar, **(B)** Blood agar showing hemolysis, **(C)** Brain heart infusion agar (BHIA), and**)D)** microscopic morphology at 1000x magnification, stained with Gram stain. **(E)** The catalase test is used to differentiate between Staphylococcus and Streptococcus species.

compared to Group 3 (0%). the PCR products of the *16S rRNA* is shown in Fig. 2.

Identification of virulence genes in S. mutans using PCR technique

Table 2 presents the detection frequencies of virulence-associated genes —*dexA*, *atpD*, and *ldh*—in *S. mutans* isolates. across the two study groups: G1 and G2. These genes are integral to the pathogenicity of *S. mutans*, particularly in its role in dental caries. The results show that *dexA* gene had the highest detection rate, being identified in 100% of the isolates (30 patients), whereas the *atpD* gene was identified in 50% of the isolates. Finally, the *ldh* gene was observed in 30% of the isolates. A chi-squared test was conducted to compare the detection rates among the three virulence genes in *S. mutans* isolates. The results showed a statistically significant difference between genes (Chi-square = 32.5, $p < 0.001$). Fig. 3 A, B, and C illustrated the Monoplex PCR products of *dexA*, *atpD,* and *ldh* virulence genes among *S. mutans* isolates.

The results of sanger sequencing

Sanger sequencing was conducted on four local isolates, and the isolates have been submitted to GenBank /NCBI under accession numbers PQ796766 to PQ796769 (https://www.ncbi.nlm.nih.gov/nuccore/PQ796769). Sequence similarity analyses, employing both local and global multiple alignment methods, revealed varying levels of similarity. Among the isolates, T2 16S.ab1 (PQ796767), which exhibited the highest similarity to other local isolates, was chosen for comparative analysis with international

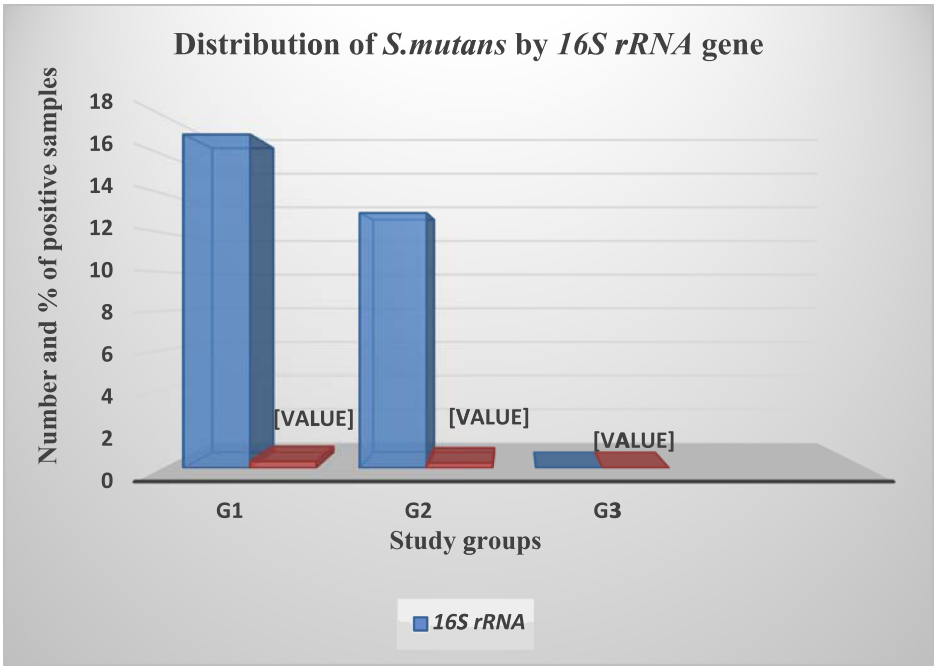

Fig. 2. Distribution of *S. mutans* according to molecular detection by *16S rRNA* gene.

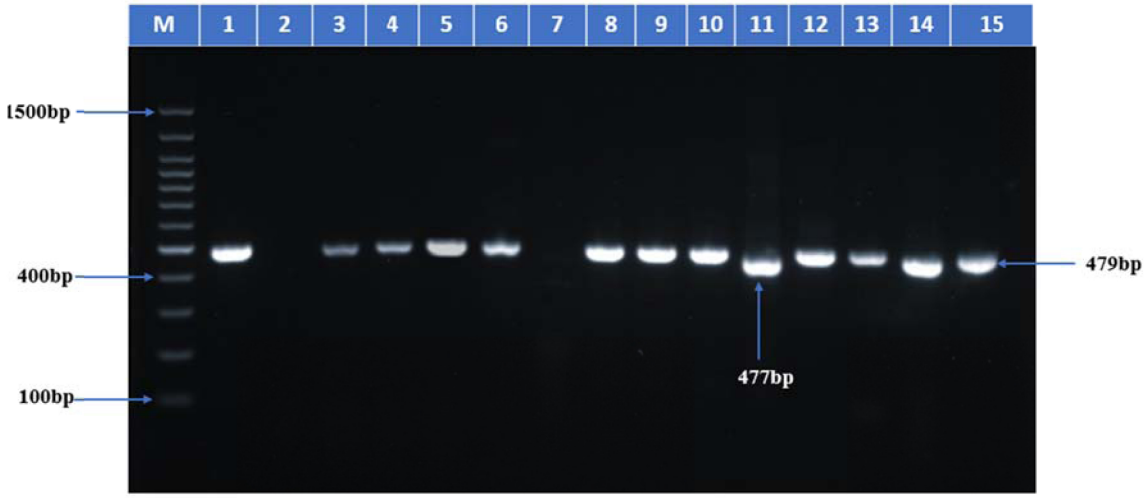

Fig. 3. Polymerase chain reaction (PCR) products of *16S rRNA* gene among *S. mutans* with an expected size (479bp,477bp). Lane M: DNA marker (100- 1500bp). Lane 2 and 7: negative for *S. mutans*. Lane 1,3,4,5,6,8,9,10,11,12,13,14,15: positive result for the *16S rRNA* gene.

Table 2. Detection rates of virulence-associated genes (*dexA*, *atpD*, *ldh*) in *Streptococcus mutans*.

Gene name	N	%
dexA	30	100%
atpD	15	50%
ldh	9	30%
Chi-squared test	32.5	
P value	$<0.0001^{S}$	
	Significant difference between genes (p value ≤ 0.05)	

strains from India, the United States, and other countries. Multiple sequence alignment, performed using Geneious Prime software, indicated a close genetic relationship between the local isolate and strains from other geographical regions.

Regional examination of genetic sequence variations in S. mutans isolates

Revealing a high degree of similarity among isolates T1, T2, and T4 in Table 3, with a similarity rate exceeding 99%. In contrast, isolate T3 showed significant sequence variation, with a 92% similarity to the other isolates. This difference was concentrated in certain parts of the gene sequence, where point mutations (substitutions) such as G to A or C to T were found, as illustrated in Fig. 4. These changes resulted in modifications to the expected amino acid sequence.

Table 3. Showing the percentage of similarity between isolates.

Name of isolates	T1_16S.ab1	T2_16S.ab1	T3_16S.ab1	T4_16S.ab1
T1_16S.ab1		100%	92.439%	99.756%
T2_16S.ab1	100%		92.439%	99.756%
T3_16S.ab1	92.439%	92.439%		92.176%
T4_16S.ab1	99.756%	99.756%	92.176%	

Multiple alignment of 16S rRNA for the local isolate and different from other countries

Through multiple alignment, the local isolate (PQ796767) was compared with other strains from different countries to determine the percentage of similarities and differences within the sequence of the studied region, as shown in Table 4. The results indicate that some nitrogenous bases were replaced by others, making it more heterogeneous than the rest of the international strains. This is indicated by the

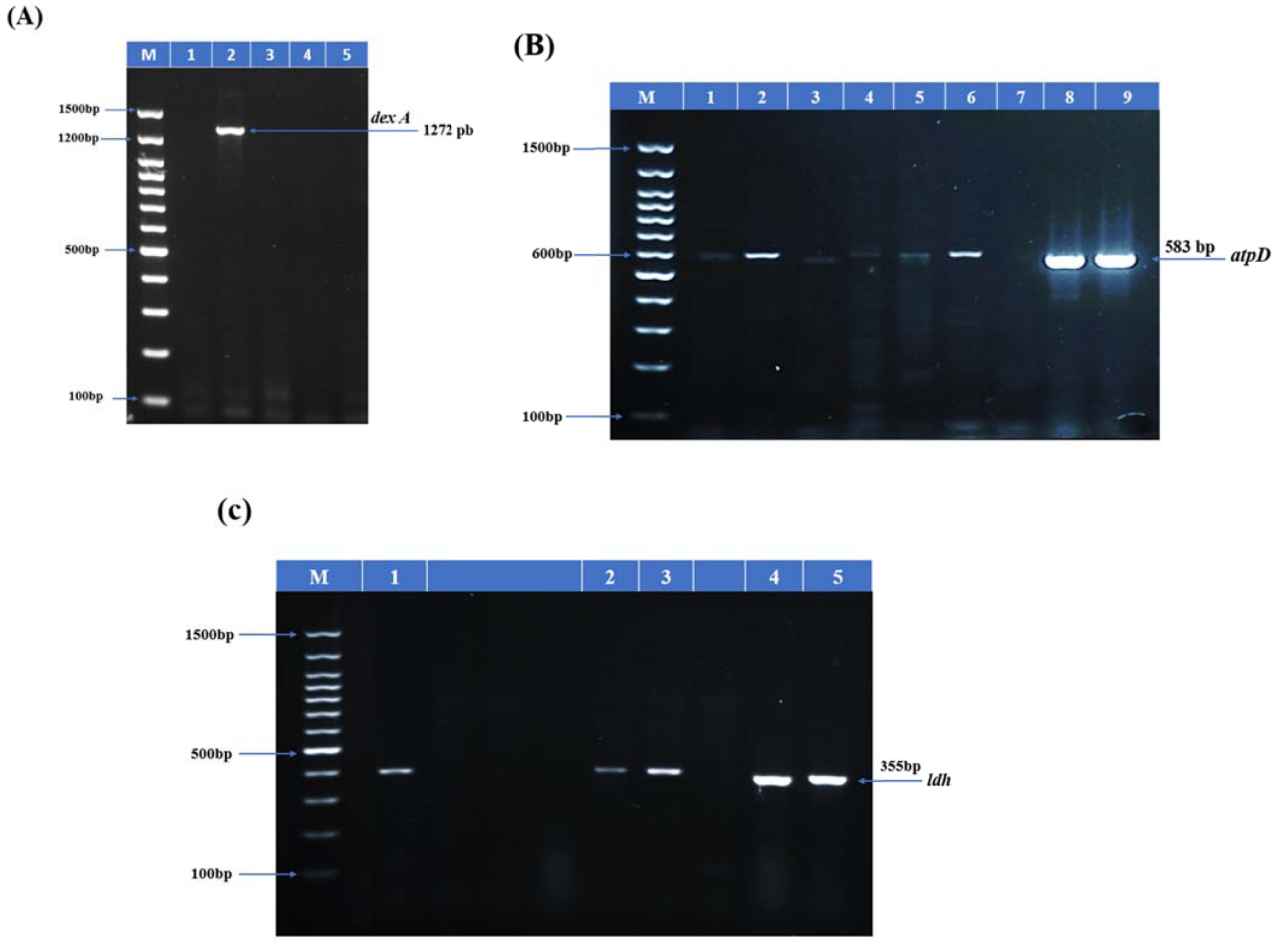

Fig. 4. Monoplex PCR products of *dexA*, *atpD,* and *ldh* virulence genes among *S. mutans* isolates. Lane M: marker (100- 1500bp), **(A)** Lane 2: positive result of *dex A* gene. Lane 1, 3-5: negative results of the *dex A* gene (1272bp). **(B)** Lane 1-6,8, and 9: positive detection of *atpD* gene with PCR product (583bp). Lane 7: negative result of the *atpD* gene. **(C)** Lane 1-5: positive result of the *ldh* gene(355bp). The electrophoresis was performed using a 2% agarose gel, 7v/cm for 90 min.

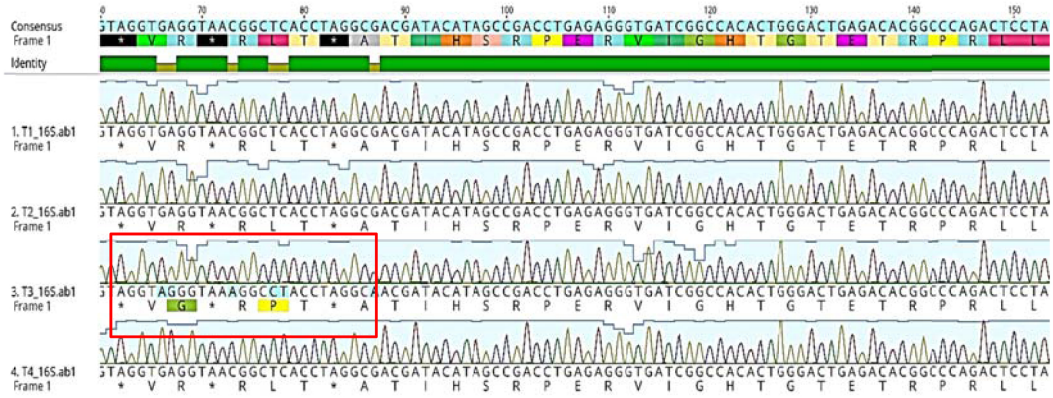

Fig. 5. Local alignment of the *16S rRNA* gene sequences from four distinct isolates. The analysis revealed variations in the local isolates, specifically in the 65A > G and 75T > C gene sequences.

Table 4. Illustrating the comparative similarity percentages between local isolates and strains from various countries.

Isolation code and country	PQ796767 Local	CP050271 Brazil	CP050272 USA	DQ677736 South Korea	GU907522 UK	MW263089 India	NR_114726 NCTC	OQ345820 Mosul
PQ796767 Local		92.548%	92.548%	92.788%	92.548%	90.909%	90.909%	92.548%
CP050271 Brazil	92.548%		100%	99.76%	100%	100%	100%	100%
CP050272 USA	92.548%	100%		99.76%	100%	100%	100%	100%
DQ677736 South Korea	92.788%	99.76%	99.76%		99.76%	99.76%	99.76%	99.76%
GU907522 UK	92.548%	100%	100%	99.76%		100%	100%	100%
MW263089 India	90.909%	100%	100%	99.76%	100%		99.8%	100%
NR_114726 NCTC	90.909%	100%	100%	99.76%	100%	99.8%		100%
OQ345820 Mosul	92.548%	100%	100%	99.76%	100%	100%	100%	

colors designated by the Geneious Prime program. The green color indicates identical sites across most or all isolates (highly conserved). The red color indicates a mutation or a non-identical site. Yellow indicates partial similarity or a base that is conserved in most of the isolates, but with some differences. Finally, the blue color indicates whether they are purine or pyrimidine bases, as presented in Fig. 5.

Phylogenetic analysis

In this study, the Neighbor-Joining (NJ) method was employed to construct an evolutionary tree that compares the genetic relationship of the local isolate PQ796767 with other isolates documented in the NCBI database Fig. 6.

This results indicate that the local isolate PQ796767 occupies a distinct branch, separated from other isolates by an evolutionary distance of 0.0784, suggesting significant genetic divergence, while the other isolates formed closely related groups: isolate DQ677736 from South Korea was positioned nearby, isolates CP050271 and CP050272 shared a cluster indicating genetic similarity, isolate GU907522 from the UK was linked to isolate OQ345820 from Mosul, and two isolates from India, MW263089 and NR-114726NCTC, were also identified.

The branch distances reflect the degree of genetic divergence or similarity, with lower values suggesting closer relationships. These findings emphasize a clear genetic distinction between the local isolate and global isolates, potentially shaped by environmental factors or selective pressures. Additionally, the tree shows close connections among certain isolates from different regions, suggesting globally distributed strains with shared genetic ancestry Fig. 7.

Discussion

Diabetes mellitus is regarded as one of the most serious chronic diseases, and it is also one of the fastest-growing in the 21st century. Indeed, it is estimated that within the next 20 years, the number of people with diabetes worldwide will exceed 643 million.[30] It has become a major public health concern in recent times. It is the most prevalent form of diabetes and one of the leading causes of premature death worldwide, according to the World Health Organization in 2021.[31] Periodontitis is a chronic inflammatory disease characterized by a multifactorial etiology. It is considered a major public health problem, with prevalence increasing since 1990 in both developed and developing countries. The global prevalence of

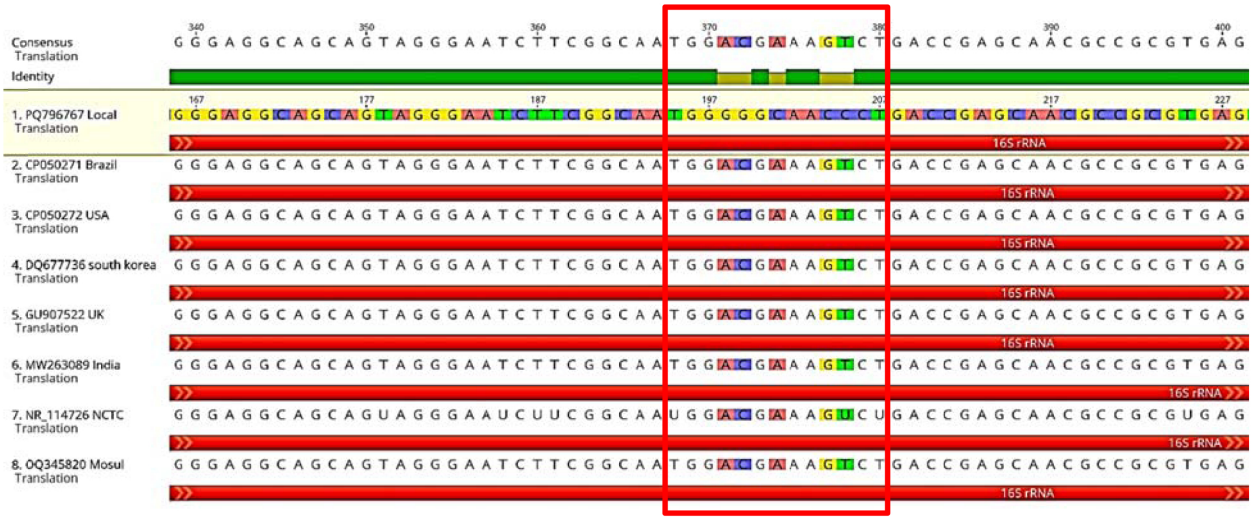

Fig. 6. Global alignment based on the *16S rRNA* gene for *S. mutans* isolates.

the condition is estimated to be approximately 45–50%, with 11% of the global population affected by its severe form. This makes it the sixth most common disease in the world.[32] Periodontitis is recognized as the sixth most prevalent form of dental disease. The presence of complications associated with diabetes indicates a greater prevalence of complications related to diabetes.[33] Chronic hyperglycemia is a significant risk factor for the development of periodontitis, contributing to its increased prevalence, severity, and progression. Similarly, periodontitis has been observed to exacerbate insulin resistance and compromise glycemic control, thereby leading to the development of diabetes-related complications. The presence of chronic hyperglycemia has been demonstrated to be associated with a higher incidence of diabetes-related complications.[34]

The study found that the presence of *Streptococcus mutans* bacteria was higher in patients with type 2 diabetes than in the control group (groups 2 and 3), despite the limited sample size. Our study is consistent with Al-Sudani and colleagues in Iraq, where a high frequency of oral bacteria, *S. mutans,* was found in type 2 diabetic patients compared to non-diabetic patients.[12] *Streptococcus mutans* appears on MSBA medium as a small, blue colony with viscous consistency resulting from sucrose metabolism to dextran. The type of hemolysis on blood agar medium is alpha-type, which shows partial breakdown of red blood cells, producing a greenish hue.[9,35] In another study, the hemolysis type appeared as gamma (with no hemolysis).[27] Despite the ongoing debate surrounding the correlation between the number of genotypes and the genotypic diversity of an individual, and their association with caries status, it is conceivable that the concurrent action of multiple genotypes, each exhibiting distinct phenotypic potentials, may culminate in disparate virulence attributes, thereby augmenting the risk of developing caries.[36]

One of the genes that plays a critical role in regulating biofilm and carbohydrate metabolism is the *dexA* gene, which codes for dextranase, an enzyme that catalyzes the breakdown of dextran molecules within the biofilm structure and regulates carbohydrate metabolism. Another gene called *atpD*, encoding the F1F0 ATPase synthase beta subunit, is an essential enzyme for proton translocation and ATP synthesis, especially under the acidic conditions prevalent in the oral cavity.[37] The *ldh* gene encodes lactate dehydrogenase, playing a key role in glycolytic fermentation by converting pyruvate into lactate, which is essential for acid production and cariogenic potential.[38] The moderate detection rate of this gene may suggest that some strains employ alternative metabolic pathways or rely on enzymatic redundancy.[39] While the study's limited sample size and focus on Baghdad, Iraq, limit its generalizability, broader insights into *Streptococcus mutans* impact on diabetic populations could emerge from future research with a larger and more diverse cohort. Monitoring oral health regularly in type 2 diabetes patients is advisable to address dental issues earlier. Efforts to counter *Streptococcus mutans* virulence factors through targeted approaches can enhance prevention, and collaboration between dental and medical professionals is crucial to integrating oral and systemic care for diabetic patients.

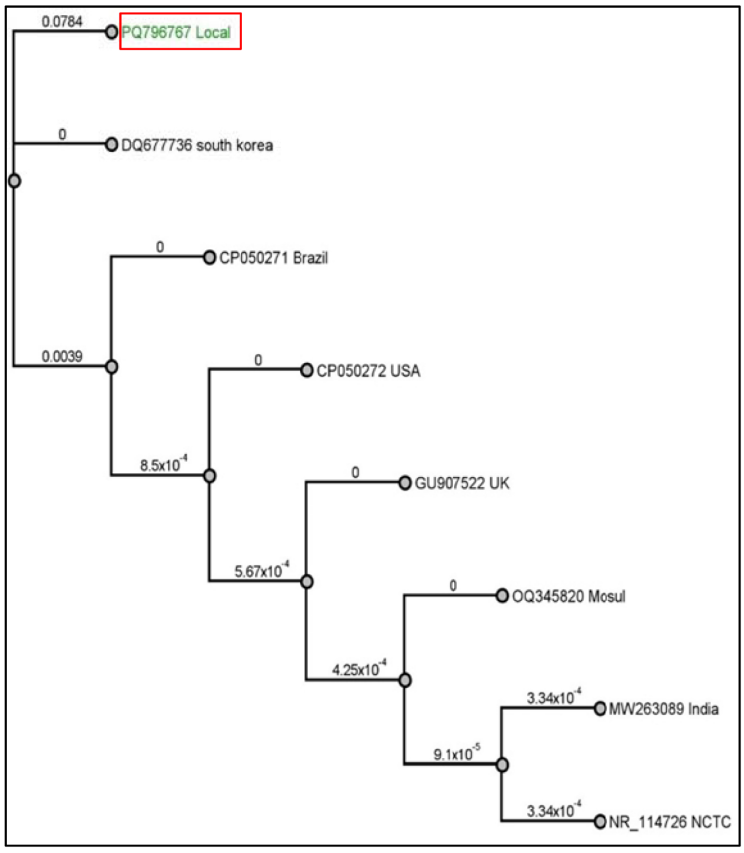

Fig. 7. Phylogenetic tree analysis demonstrates the relationship between the sequence of Iraqi isolates and various strains from other countries.

Conclusion

The study reveals the significant genetic diversity and virulence potential of *Streptococcus mutans,* especially in connection with type 2 diabetes. It identifies key virulence genes and documents new isolates, providing a valuable understanding of the bacterium's pathogenic mechanisms. This knowledge could support the creation of targeted prevention and treatment approaches for dental caries in individuals with diabetes. However, a small sample size, influenced by a narrow scope and focus on Baghdad city, may restrict the generalizability of the findings. Additional research should explore the *Streptococcus mutans* pathogenic mechanisms, including its potential spread through the bloodstream and role in systemic infections.

Acknowledgment

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Authors' declaration

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are ours. Furthermore, any Figures and images that are not ours have been included with the necessary permission for re-publication, which is attached to the manuscript.
- No animal studies are present in the manuscript.
- Authors signed on ethical consideration's approval.
- Ethical Clearance: The project was approved by the local ethical committee at University of Baghdad.

Authors' contribution statement

T.A. and S.I. designed the research plan and study design were developed collaboratively. The T.A. was actively involved in the collection and identification of samples, as well as in drafting the initial manuscript. Meanwhile, the S.I. provided continuous guidance throughout the study, contributing to data analysis and the review and editing of the article.

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التوصيف الجزيئي وعوامل الضراوة لبكتيريا المكورات العقدية الطافرة المعزولة من تسوس الأسنان لدى مرضى السكري من النوع الثاني

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الخلاصة

الخلفية: يُعدّ مرضى السكري من النوع الثاني هم من اكثر الفئات المجتمعية عُرضةً لتسوس الأسنان المتكرر. تعد بكتيريا العقدية الطافرة المسبب الرئيسي لتسوس الأسنان، والتي تُصيب ملايين الأشخاص سنويًا وتُشكّل مخاطر جسيمة على صحة الفم. على الرغم من الأبحاث المُكثّفة، لا تزال آلية تطوّر هذه البكتيريا غير مفهومة تمامًا، مما يُؤكّد ضرورة إجراء المزيد من الدراسات. الأهداف: هدفت هذه الدراسة إلى عزل وتشخيص السمات الجزيئية لبكتيريا العقدية الطافرة من مرضى السكري من النوع الثاني. يكشف البحث عن قدرة هذه البكتيريا على التسبب بالعدوى، والبقاء على قيد الحياة، في ظل الظروف الفموية الحمضية لمرضى السكري والتحديات التي تعيق نموها. حلّلت الدراسة التنوّع الجيني لبكتيريا العقدية الطافرة، وبناء شجرةً تطورية. المنهجية : تم جمع 120 عينة , 50 مريض مُشخّص على انه مصاب بالسكري من النوع الثاني بالاضافة الى تسوس الاسنان، و50 مريض يعانون من تسوس الاسنان. بالاضافة الى 20 عينة من متبرعين أصحاء . تم استخدام بادئات 16S rRNAمصممة باستخدام برنامج Geneious Prime كما فحصت الدراسة عوامل الضراوة dexAو ldh و atpD تم تحديد ثلاثين عينة من 17 ، Streptococcus mutans منها كانت من مرضى مصابين بالسكري من النوع الثاني ويعانون من تسوس الاسنان , و 13 من أولئك الذين يعانون من تسوس الأسنان. من بين العزلات، كانت نسبة 100٪ جين dexA، و 50٪ جين atpD، و 30٪ جين ldh تم تعيين أرقام تسجيل تسلسل النوكليوتيدات PQ796766 و PQ796767 و PQ796768 و PQ796769 لأربع عزلات جديدة من بغداد، العراق. الاستنتاج: توضح الدراسة أن Streptococcus mutans تُظهر لديها عوامل ضراوة دفاعية تمكنها من التاقلم في البيئات الفموية ذات الاجهاد التاكسدي العالي، قد يفيد ذلك في تطوير استراتيجيات مستهدفة للوقاية من تسوس الأسنان وعلاجه لدى الأفراد المصابين بمرض السكري.

الكلمات المفتاحية: تحمل الحامضية، تحليل الشجرة التطورية،16S rRNA ، العقدية الطافرة ، داء السكري النوع الثاني.