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Molecular Description of Gene Associated with Biofilms in Oral Klebsiella Isolated from Obese People.

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الوصف الجزيئي للجينات المرتبطة بالاغشية الحيوية في بكتيريا الكليبسيلا الفموية المعزولة من الاشخاص البدينين

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ABSTRACT

Background: Obesity is a chronic health condition resulting from excessive accumulation of body fat. It is related to an increased risk of chronic diseases such as diabetes, heart disease, and high blood pressure, and is often caused by an imbalance between calories consumed and expended.

Materials and Methods: This study aims to find relation between the presence of *Klebsiella* bacteria in the oral microbiota and obesity and the possibility of its association with other chronic diseases; it was conducted between November and December 2025, targeting obese individuals. Oral swab samples were collected to analyze the relationship between obesity and oral bacterial composition, with a focus on the presence of Klebsiella bacteria and genotypes associated with biofilm formation.

Results: The study included 65 participants, divided according to their body mass index (BMI): 2 participants had normal weight (3%), 17 were overweight (26%), 26 were obese (40%), and 20 were morbidly obese (31%). Diagnostic results showed that 10 of the 65 samples (13.38%) were positive for Klebsiella spp., indicating a relatively moderate prevalence of these bacteria in the mouths of obese individuals. To understand the mechanisms of pathogenesis, the presence of three key genes responsible for biofilm formation in these bacteria was analyzed: Only four isolates were positive for the rmpA gene, while only three isolates had the mrkD gene, and only two isolates had the mrkA gene.

Conclusion: The results of this study suggested a potential association between obesity and the presence of *K. pneumoniae* in the oral cavity, as well as differences in the presence of genes associated with biofilm formation, which may reflect variation in the pathogenicity of these bacteria.

Key words: Obesity, Klebsiella spp., biofilm genes, Oral swabs.

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INTRODUCTION

The microbiota is the human body's host to a diverse and dynamic community of microorganisms, which play an important roles in maintaining health and contributing to disease. Among these microbial ecosystems, the oral cavity represents one of the most complex and heavily colonized sites, second only to the gut. The oral microorganism includes hundreds of bacterial species that co-exist in a precise balance, affecting both local and systemic health. Defects in this balance, known as dysbiosis, have been implicated in various diseases ranging from periodontal disease to systemic conditions such as cardiovascular disease and obesity [1].

Klebsiella, a genus of Gram-negative, facultatively anaerobic rods within the family *Enterobacteriaceae*, is an opportunistic pathogen that is commonly found in the gastrointestinal tract, environment, and increasingly, in the mouth. The species *K. pneumoniae* is of particular concern due to its related to hospital-acquired infections, antibiotic resistance, and pathogenicity. Although not traditionally considered a core member of the healthy oral microorganisms, *Klebsiella* can colonize the oral cavity under certain conditions, particularly in immune-compromised individuals or those with poor oral hygiene Its presence in the mouth may be a sign of a shift toward a more pathogenic microbiome, potentially contributing to local inflammation and systemic disease[2].

The oral microorganisms have a pivotal role in shaping host metabolic and immune responses. Recent studies have revealed important links between the composition of the oral microorganisms and obesity, a global health epidemic characterized by increased fat accumulation and associated with increased risk for diabetes, cardiovascular disease, and certain cancers [3]. Dysbiosis in the oral microorganisms may influence obesity through several mechanisms, including low-grade systemic inflammation, modulation of appetite-regulating hormones, and interactions with the gut microbiome [4]. The migration of oral bacteria into the digestive system, especially in individuals with poor oral health, may alter gut microbial composition, leading to metabolic changes that promote weight gain and insulin resistance [5].

Adhesives attach bacteria to host cells or shells. Caustic glues. For new host colonization, germs stick. We treat and prevent infections by attacking bacterial adhesions. By fimbriae pili, bacteria assault kerchief shells. K has 3 fimbriae. 1, 3, K pneumoniae. KPC pneumoniae carbapenemase. Thin, stiff, hair-like type I fimbriae are bacteria. The fim gene cluster decrypts them after chaperones/intercellular pathways aggregate them [6]. Small-terminal adhesin is encoded by fimH, structural subunit by fimA. pneumoniae but not E. coli possesses fimK. Type I fimbriae need fimK [7]. The type I fimbriae gene is expressed in the urinary system but not the GI tract or lungs for bladder cell biofilming. The chromosomal or plasmid-derived mrkABCD gene cluster spirally paraphrases type three fimbria. The fimH and mrkD genes of type I and type III fimbriae attach to host cells separately [8].

MATERIALS AND METHODS

Collection of samples

Sixty five oral cavity swabs and 79 were collected from persons who were suffering from obesity, by using sterile swabs for both sexes with ages ranging from (5-65) years. All health information was taken, including weight, height, waist circumference, and hip circumference. This study was conducted from November and December 2025; and then, all swabs were cultivated in different culture media, MacConkey agar, mannitol salt agar, chromogenic agar, and Eosin methylene blue, and incubated aerobically, at the temperature of 37°C for (24-48) hours.

The media agar was prepared according to manufactural guides. The swab was immediately streaked on plates of media and incubated at 37°C for 24 h. The plates were then observed for growth and a colonial description of the isolates. After pure colonies were obtained, they were taken from each primary positive

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culture on MacConkey, chromogenic, and Mannitol salt agar medium and they were identified depending on its morphology and then it was examined by light microscope after being stained with Gram's stain.

DNA extraction

The genetic material (DNA) was then extracted using a DNA extraction kit from Favorgen(cat NO. FABGK 001-1), according to the manufacturer's instructions.

PCR and electrophoresis

The mixture of PCR was 2µl of DNA sample , 1.5µl of each forward and reverse primer, and 15µl of green master mix . agarose was prepared by dissolving 1gm of agarose in 100 ml TBE buffer. primers used in this research are in table 1.

Table 1. primer sequences used in the study

Genes	Primers	Sequences (5'→3')	Tm ̊C 35cycles	Product size (bp)
16sRNA K.p	F	5'CCTGGACAAAGACTGACGCT3'	60	584
	R	5'AGTTGCAGACTCCAATCCGG3'		
Rmpa	F	ACTGGGCTACCTCTGCTTCA	55	535
	R	CTTGCATGAGCCATCTTTCA		
Mrkd	F	GTCTTTTCGTCCCGGGTATATAAC	58	244
	R	CCACATCGACATTCATATTTTTCC		
Mrka	F	ATGCGAACGTTTACCTGTCTCC	58	298
	R	CCCGGGATGATTTTGTTGG		

RESULTS AND DISCUSSION

A sixty five cotton swab sample obese individuals ; body mass index was calculated according to BMI equation. After applying the equation, it was possible to divide the samples under study according to the type of obesity based on the levels shown in the figure (1),

Figure (1) BMI ranges

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Depending on the ranges in the above figure, it was found that the samples were divided as follows: normal weight 2(3%), overweight 17 (26%), obese 26 (40%) and extremely obese 20 (31%) as shown in figure (2).

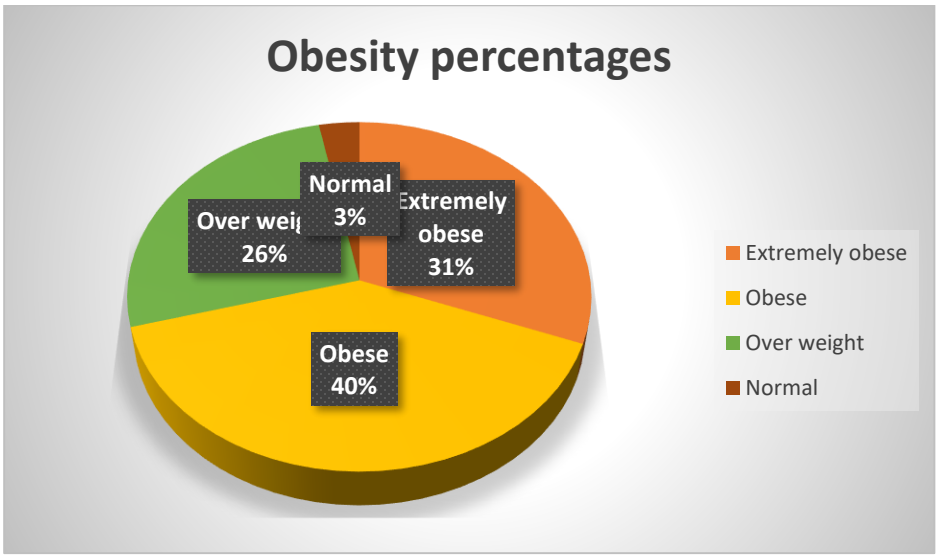

Figure (2) Obesity percentages

Cotton swabs were cultured on more than one culture medium to determine the bacterial species present in the mouth. A number of bacterial species were found among them and then diagnosed based on microscopic examination (Gram stain) . Additionally, Klebsiella sp. Was identified using molecular diagnosis 16SrRNA .figure (4),

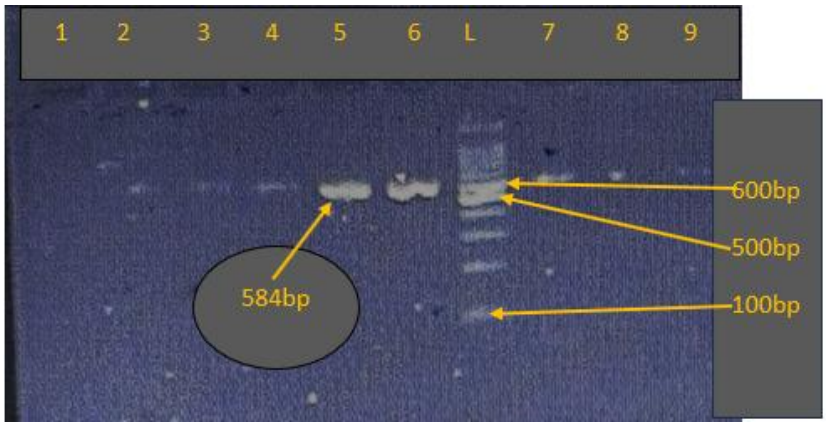

Figure (4) 16SrRNA of *Klebsiella* PCR product (584bp). PCR products were separated by electrophoresis in an 1.5% agarose gel, at 80 V\Cm for 60 min. M: Marker DNA ladder size (100bp)

The results show that out of 65 samples only 10 samples (13.38%) gave *Klebsiella* sp. Three genes that responsible for biofilm forming in Klebsiella bacteria were studied, and the results for the ten samples were as follows: for rmpA gene there were only 4 samples that gave the positive results figure (5) ,for mrkD gene only three isolates possessed it Figure (6) and for mrkA two isolates were positive figure (7)

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In comparison to antibiotic-sponsored studies, the Balaky research comes out on top. A total of 92 different bacterial species were isolated from the mouths of 252 individuals; the most common of these were *S. aureus* (47-83%), followed by *P. aeruginosa* (23.91%), *B. subtilis* (5.43%), *E. faecalis* (4.34%), *E. aerogenes* (2.17%), and *K. pneumonia* (0.85%) [9]

Ectopic oral bacteria, which normally appear innocuous in the mouth, have recently been shown to have a harmful function, according to research. Commensal bacteria in the mouth and digestive tract are Klebsiella spp. Klebsiella pneumoniae and Klebsiella aeromobilis are two of the most notorious bacteria that can cause infections in hospitals (CASIs). Researchers found that certain Klebsiella pneumoniae and Klebsiella aeromobillis bacteria often found in the saliva of people with Crohn's disease (CD) and ulcerative colitis (UC) can effectively stimulate the production of T helper I cells[10].

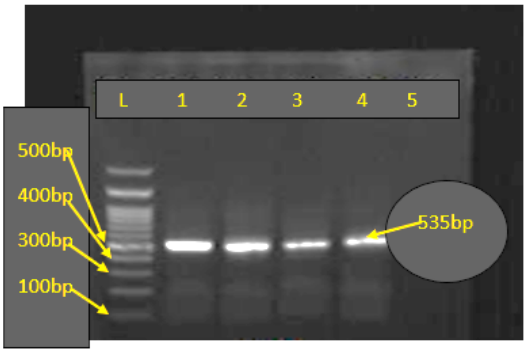

Figure (5) **PCR product of** rmpA **(535bp). The product of PCR were separated by electrophoresis in an 1.5% agarose gel, at 80 V\Cm for 60 min. M: Marker DNA ladder size (100bp)**

Seven (6.4% of the total) isolates were found in urine (2), blood (3), and wounds (2), and were all related with the hypercapsule (hypermucoviscous phenotype) gene (2). All rmpA-positive isolates carried the entB and irp-1 siderophore genes, while a small number of rmpA-positive isolates also had the iroD, iroN, and clbA and clbB genes encoding colibactin. The kfu and wcaG genes were detected in 19% of the K1 isolates. Other isolates that had the wcaG gene included K2 (sixteen percentage), K57 (five percentage), and K54 (fifty percentage). Isolates of various K serotypes carried the most prevalent virulotypes: entB, irp-1, fimH, and mrkD. As diagnostic biomarkers for hvKP, the rmpA, iroD, or iroN genes were found in 18 (16.5%) of the KP isolates studied. Seven specimens (6.4%) from blood, wounds, and urine were positive for the rmpA gene, which is responsible for regulating mucoid phenotype A, according to the research by Guo et al., [11]. The rmpA and iroB genes were shown to be highly accurate laboratory markers for detecting hvKp, according to the results reported by Russo et al. [12]. After looking at KP isolates from hospitalized patients for rmpA, iroD, and iroN, we found that 16.5% of the isolates had at least one hvKp strain-related genetic trait. Some rmpA isolates have also been found to encode colibactin genes. This non-ribosomal peptide-polyketide can trigger host DNA damage and cause colorectal cancer [13,14]. Although most cases of antibiotic resistance are due to cKP, reports have emerged of hvKP strains that contain ESBLs [15,16]. Also found were the siderophore genes that encode salmochelin, diagnostic markers for hvKP, and the rmpA gene, which is linked to the hyper-mucoviscous phenotype. Among hvKP, nearly 50% were MDR and developed ESBLs. The healthcare system is facing a grave danger from the rising tide of hypervirulent and antibiotic-resistant KP isolates.

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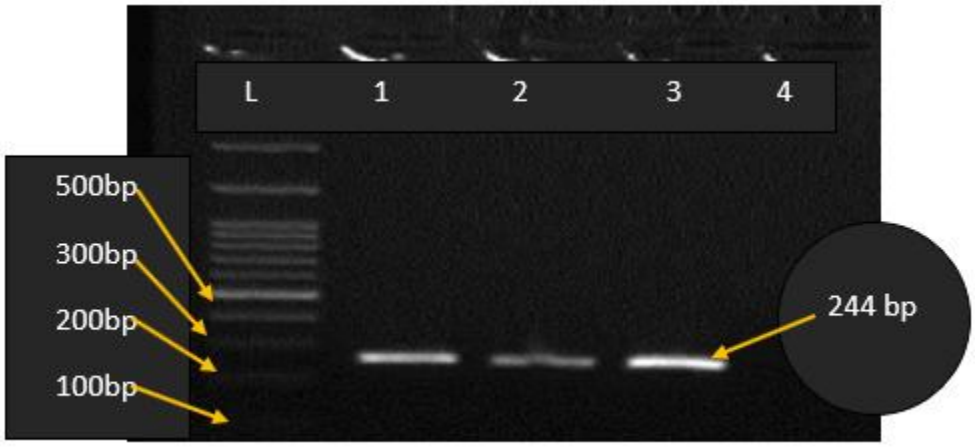

Figure (6) **PCR product of** mrkD **(535bp).The product of PCR were separated by electrophoresis in an 1.5% agarose gel, at 80 V\Cm for 60 min. M: Marker DNA ladder size (100bp)**

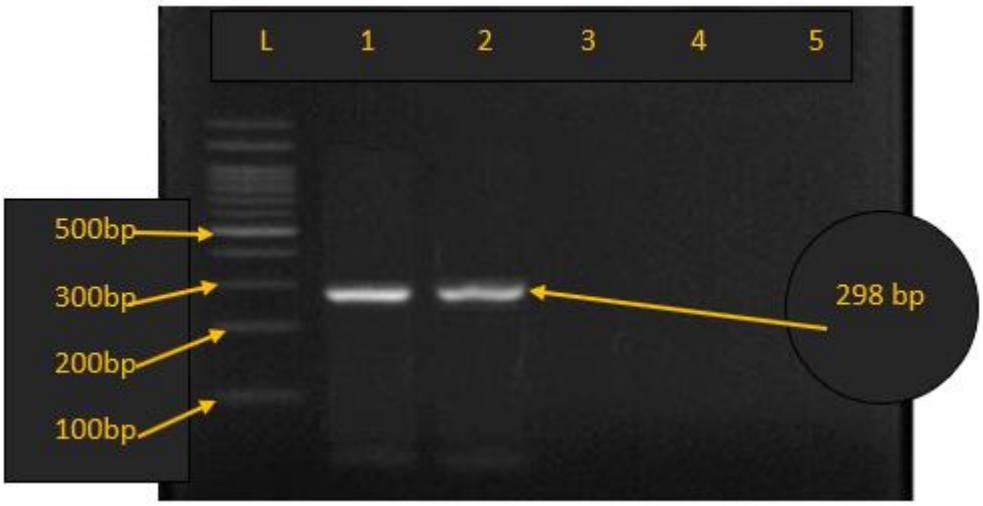

Figure (7) **PCR product of** mrkA **(535bp). The products of PCR were separated by electrophoresis in an 1.5% agarose gel, at 80 V\Cm for 60 min. M: Marker DNA ladder size (100bp)**

Several clusters of fimbrial genes allow Klebsiella pneumonia to attach to surfaces and produce biofilms. Bacterial fimbriae, which resemble hair and are protein structures, enable attachment to both biotic and abiotic surfaces; this property is useful in medical contexts, such as catheters. Type Iand type three fimbriae, which have been extensively studied, are encoded by the fim and mrk gene clusters, respectively. Furthermore, adhesion and biofilm development are aided by the ecp and kpa-kpg fimbrial gene clusters[16].

When growing biofilms on inanimate surfaces, type 3 fimbriae, primarily MrkA, are essential. In a chaperone-usher route, MrkB and MrkC build these fimbriae, while MrkD provides binding specificity.

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The growth of biofilms is significantly reduced upon deletion of mrkA or mrkD. The expression of type three fimbriae is regulated by the (mrk,H,I,J) gene cluster. As a "biofilm switch," the PilZ domain-containing transcriptional activator MrkH responds to c-di-GMP by activating the mrkABCDF operon. Additional regulators that enhance mrkHI expression and fimbriae production are Fur, a global regulator, and MrkI, a LuxR-like regulator. Environmental factors, such as phosphatidylcholine and cholesterol derived from lung surfactant, have the ability to enhance the expression of mrk genes and the production of biofilms [17].

Bacteria can transition from a planktonic to a biofilm lifestyle, propelled by c-di-GMP, when they attach to inanimate objects or damaged epithelial tissues via type 3 fimbriae. Enzymes such as DGC and PDE control the concentrations of c-di-GMP. Inhibiting fimbriae formation and degrading c-di-GMP, MrkJ is the PDE found in the mrkHIJ cluster. In its absence, biofilm formation and fimbriae are accelerated by high c-di-GMP. In response to oxidative stress, YfiN, a DGC, promotes fimbriae synthesis whereas YjcC, another PDE, inhibits it. OmpR/EnvZ osmotic stress-sensing also impacts fimbriae regulation and C-di-GMP [18].

Infections in the urinary tract can be caused by type I fimbriae, which are encoded by the fim gene cluster (fimAICDFGHK). These fimbriae attach to mannose residues on the surfaces of host epithelial cells. Host binding requires the apical adhesin FimH. The fim gene cluster is controlled by transcriptional repressors such as PDE-active FimK and DNA inversion (fimS). Deletion of fimK leads to an increase in hyperfimbriation and colonization. When it comes to catheters, the two kinds of fimbrial algae work together to promote biofilm formation. Although type I fimbriae can cause a decrease in type III expression, type III fimbriae are more influential [19]

Additionally, adhesion and biofilm growth are assisted by ECP (ecpRABCDE) and kpa-kpg gene cluster fimbriae. Microcolonies and long-lasting biofilms are produced by ECP in strains that do not possess type III fimbriae. Mutations in kpaC, kpeC, and kpjC decrease adhesion and biofilm formation. The kpfR gene cluster controls the expression of type 1-like fimbriae in a negative feedback loop. Eliminating it enhances adhesion between epithelial cells and biofilms[20]

CONCLUSION:

The results of this study suggested a potential association between obesity and the presence of *K. pneumoniae* in the oral cavity, as well as differences in the presence of genes associated with biofilm formation, which may reflect variation in the pathogenicity of these bacteria.

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Conflict of interests.

There are non-conflicts of interest.

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الخلاصة

السمنة حالة صحية مزمنة ناتجة عن تراكم مفرط للدهون في الجسم. وهي مرتبطة بزيادة خطر الإصابة بأمراض مزمنة مثل السكري وأمراض القلب وارتفاع ضغط الدم، وغالبًا ما تنتج عن اختلال التوازن بين السعرات الحرارية المستهلكة والمحروقة. تهدف هذه الدراسة إلى تحديد العلاقة بين وجود بكتيريا الكلبسيلا في الميكروبات الفموية والسمنة. أُجريت الدراسة بين نوفمبر وديسمبر 2025 واستهدفت الأفراد المصابين بالسمنة. جُمعت عينات مسحات فموية لتحليل العلاقة بين السمنة وتكوين البكتيريا الفموية، مع التركيز على وجود بكتيريا الكلبسيلا والأنماط الجينية المرتبطة بتكوين الأغشية الحيوية. شملت الدراسة 65 مشاركًا، تم تقسيمهم وفقًا لمؤشر كتلة الجسم (BMI): مشاركان بوزن طبيعي (3%)، و17 يعانون من زيادة الوزن (26%)، و26 يعانون من السمنة (40%)، و20 يعانون من السمنة المفرطة (31%). أظهرت نتائج التشخيص أن 10 من أصل 65 عينة (13.38%) كانت إيجابية لبكتيريا الكلبسيلا، مما يشير إلى انتشار متوسط نسبيًا لهذه البكتيريا في أفواه الأفراد المصابين بالسمنة. لفهم آليات الإمراض، تم تحليل وجود ثلاثة جينات رئيسية مسؤولة عن تكوين الأغشية الحيوية في هذه البكتيريا: أربع عزلات فقط كانت إيجابية لجين rmpA، بينما ثلاث عزلات فقط كانت تحمل جين mrkD، وعزلتان فقط كانتا تحملان جين mrkA. أشارت نتائج هذه الدراسة إلى وجود ارتباط محتمل بين السمنة ووجود بكتيريا *Klebsiella pneumoniae* في تجويف الفم، بالإضافة إلى اختلافات في وجود الجينات المرتبطة بتكوين الأغشية الحيوية، والتي قد تعكس اختلافًا في قدرة هذه البكتيريا على إحداث المرض.