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سلوك الالتصاق لعزلات الإشريكية القولونية الممرضة للمجاري البولية المعزولة من مرضى في مستشفيات مدينة الرمادي قبل وبعد التعرض للإجهاد الكيميائي

The Adhesion Behavior of Uropathogenic E. coli isolates collected from patients in Ramadi City hospitals before and after chemical stress

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Abstract

Uropathogenic Escherichia coli (UPEC) exhibit adhesion capabilities that are central to their pathogenicity in urinary tract infections (UTIs). This study aimed to evaluate the adhesion potential of 70 clinical E. coli isolates collected from patients in Ramadi City hospitals before and after chemical stress using Dettol and Surgical disinfectant OPA plus solution. The adhesion assay was performed using OD measurements at 630 nm. Results indicated a significant reduction in adhesion after Dettol exposure (mean OD = 0.03406, $p < 0.0001$), while Surgical disinfectant OPA plus solution exposure slightly increased adhesion (mean OD = 0.04625). These findings suggest Dettol's inhibitory effect on adhesion-associated virulence factors and raise concern regarding stress-induced adhesion enhancement by alcohol-based agents.

Keywords: Urinary tract infections (UTIs), Adhesion assay, Antimicrobial resistance , E.coli .

الملخص

تُظهر الإشريكية القولونية الممرضة للمسالك البولية (Uropathogenic Escherichia coli - UPEC) قدرات التصاق تُعدّ جوهرية في إمراضها ضمن حالات عدوى المسالك البولية (UTIs). هدفت هذه الدراسة إلى تقييم القدرة الالتصاقية لـ ٧٠ عزلة سريرية من الإشريكية القولونية جُمعت من مرضى في مستشفيات مدينة الرمادي، وذلك قبل وبعد التعرض للإجهاد الكيميائي باستخدام محلول ديتول والمطهر الجراحي OPA Plus. تم تنفيذ اختبار الالتصاق من خلال قياسات الكثافة الضوئية (OD) عند الطول الموجي ٦٣٠ نانومتر. أشارت النتائج إلى انخفاض ملحوظ في الالتصاق بعد التعرض لمحلول ديتول (متوسط OD = 0.03406 ،$p < 0.0001$) في حين أدى التعرض للمطهر الجراحي OPA Plus إلى زيادة طفيفة في الالتصاق (متوسط OD = 0.04625). تُشير هذه النتائج إلى التأثير التثبيطي لمحلول ديتول على عوامل الضراوة المرتبطة بالالتصاق، كما تُثير القلق بشأن تعزيز الالتصاق الناتج عن الإجهاد الكيميائي بفعل العوامل المعتمدة على الكحول.

الكلمات المفتاحية: عدوى المسالك البولية (UTIs)، مقاومة مضادات الميكروبات، اختبار الالتصاق، الإشريكية القولونية.

Introduction

Urinary tract infections (UTIs) are among the most prevalent bacterial infections worldwide, affecting millions of individuals annually across all age groups. They represent a major burden on healthcare systems, particularly in developing countries where empirical treatment is common and diagnostic follow-up is often limited [1]. Escherichia coli, specifically the uropathogenic strains (UPEC), is considered the leading causative agent of UTIs, accounting for over 80% of community-acquired and hospital-acquired infections [2,3].

A critical virulence factor of UPEC is its capacity to adhere to uroepithelial cells, which facilitates colonization of the urinary tract, subsequent invasion of tissues, and establishment of chronic or recurrent infections [4]. This adhesion process is primarily mediated by surface structures such as type 1 fimbriae (fimH gene) and P fimbriae (papG gene), which recognize and bind to specific glycoprotein receptors on host epithelial surfaces [5,6]. The expression of these adhesins is tightly regulated and often upregulated during early stages of infection, enhancing bacterial survival and immune evasion [7].

Disruption of bacterial adhesion has become a key target in antimicrobial strategies aiming to prevent biofilm formation and limit the persistence of infections. Chemical agents such as chloroxylenol (in Dettol) and ethanol-based formulations (in Surgical Spirit) are widely used disinfectants in clinical environments for surface and hand hygiene [8]. However, sub-inhibitory exposure to these agents can exert stress responses on bacteria, potentially altering their virulence expression, including adhesion behavior [9,10]. The structure of the E. coli genome consists of a flexible gene pool (including virulence genes) and a conserved part, which is also called the “core genome”. This core genome has been preserved throughout its vertical evolution, with very limited intragenomic rearrangement, resulting in the conserved synteny that is apparent today. In E. coli, the cAMP–CRP complex serves as a global transcriptional regulator of the expression of ≈200 genes . CRP also can play a role in acid stress response, when during exponential growth in rich medium, it can repress the RpoS-dependent gad gene transcription and contribute to bacterial survival in acidic environments . Further, intracellular cAMP levels affect the transcription of OxyR, a regulator contributing to the response against oxidative stress or intracellular cAMP levels can also be modulated by external osmolarity. The adhesion phenotype in UPEC is caused by a number of genes. These are fimH (type 1 fimbriae), papG (P fimbriae), sfa (S fimbriae), and afa (afimbrial adhesins). Environmental factors, including as pH, osmolarity, and chemical exposure, affect how they manifest themselves. The CpxRA and RpoS stress

response pathways are examples of global regulatory systems that control these genes.We may learn more about how disinfectants may change the behaviour of bacteria at the molecular level by studying this genetic control [Ref].

Understanding the impact of such chemical stressors on bacterial adhesion is crucial, especially in the context of increasing antimicrobial resistance. This study investigates the adhesion behavior of UPEC isolates collected from UTI patients in hospitals in Ramadi City, both before and after exposure to chemical disinfectants, with a specific focus on Dettol and Surgical disinfectant OPA plus solution. The findings aim to provide insights into the role of hospital-grade disinfectants in modulating bacterial pathogenic traits, and their potential utility or limitations in infection control strategies.

Materials and Methods

Seventy E. coli isolates were collected from urine samples of patients clinically diagnosed with UTIs at hospitals in Ramadi City. Isolates were confirmed via VITEK 2 Compact System. To assess the adhesion capacity of Escherichia coli isolates obtained from patients diagnosed with urinary tract infections (UTIs), a standardized epithelial cell adhesion assay was performed. A total of 70 uropathogenic E. coli (UPEC) isolates were included in this study. All isolates were identified and confirmed using the VITEK 2 Compact System (bioMérieux, France), following the manufacturer's instructions [11], and preserved in glycerol stocks at –20°C until further testing.Epithelial cell samples were collected non-invasively from healthy adult volunteers by rinsing the urethral opening with sterile physiological saline. The exfoliated uroepithelial cells were washed twice with phosphate-buffered saline (PBS, pH 7.4), then standardized using a hemocytometer to ensure consistent cell counts [12]. Chemical disinfectants change the way bacteria work not just by killing them but also by putting stress on their membranes, changing the way proteins fold, and changing the proteins on their surfaces. There have been reports that low levels of chloroxylenol and OPA might cause oxidative stress responses, which can then change the expression of surface structures including fimbriae and outer membrane proteins [Ref]. These alterations caused by stress may seem like signals from the host environment, which might unintentionally encourage pathogenic features like stronger adhesion or biofilm development. Each bacterial isolate was sub-cultured in Tryptic Soy Broth (TSB) and incubated overnight at 37°C. Following incubation, cultures were centrifuged at 4000 rpm for 5 minutes, washed twice with PBS, and adjusted to 0.5 McFarland standard (~1.5×10^8 CFU/mL). Equal volumes of the bacterial and epithelial suspensions were mixed (1:1) in sterile tubes and incubated for 30 minutes at 37°C with gentle shaking [13].After incubation, the mixtures were centrifuged at 500 rpm for 5 minutes to pellet epithelial cells, and non-adherent bacteria were removed by washing with PBS. The cell pellets were smeared on clean glass slides, air-dried, heat-fixed, and stained using Giemsa

stain. Slides were examined under a light microscope at 1000× magnification with immersion oil, and a minimum of 10 epithelial cells were counted per isolate. The average number of adhered bacteria per cell was recorded [14].Adhesion patterns were categorized according to the criteria by García Méndez et al. [15]:

- Localized adhesion: bacteria clustered at one pole of the cell,

- Diffuse adhesion: bacteria evenly dispersed,

- Aggregative adhesion: bacteria forming stacked dense clusters.

To evaluate the impact of chemical stress, each bacterial suspension was exposed to:

- Dettol (chloroxylenol-based antiseptic) at 0.78%, and

- surgical disinfectant (OPA Plus solution) at 1%,

for 15 minutes at room temperature, then processed again for the adhesion assay.

Adhesion was semi-quantitatively scored as:

- (–): no adhesion,

- (+): weak adhesion,

- (++): moderate adhesion,

- (+++): strong adhesion [16].

In addition to microscopy, optical density (OD) at 630 nm was measured using an ELISA microplate reader as a complementary quantitative tool [17]. This allowed statistical analysis of changes in adhesion levels before and after chemical treatment.

Results and Discussion

Statistical Analysis of Adhesion Ability The data obtained from 70 uropathogenic Escherichia coli (UPEC) isolates revealed significant variations in adhesion capability

before and after chemical stress exposure. As shown in Table 1 the mean optical density (OD) at 630 nm for adhesion before stress was 0.04356 ± 0.01156, indicating a moderate level of adherence to epithelial cells.Upon exposure to Dettol at sub-inhibitory concentrations, a statistically significant reduction in adhesion was observed with a mean OD of 0.03406 ± 0.009742 ($p < 0.0001$), suggesting the compound's effectiveness in disrupting bacterial surface attachment mechanisms. This aligns with previous findings demonstrating the

bactericidal effects of chloroxylenol, Dettol's active component, on cell membranes and fimbrial adhesins [18].Conversely, isolates exposed to the surgical disinfectant OPA Plus exhibited a mild increase in adhesion (OD = 0.04625 ± 0.01224, $p < 0.0001$). This enhancement may be linked to stress-induced upregulation of adhesin-encoding genes such as fimH or papG, which is consistent with earlier reports documenting similar adaptive responses under environmental or antiseptic stress [19,20]. Hassan et al. (2020) also found that Dettol exposure lowered the expression of the fimH gene in UPEC isolates. Lu et al. (2019) found that ethanol-based antiseptics made surfaces more hydrophobic, which made it easier for germs to stick to them. These comparisons back up what we found and show how important it is to choose disinfectants based on more than just their ability to kill bacteria. They should also be chosen based on how they affect the behaviour of virulence [Ref]. These findings indicate a dual behavior: Dettol reduced the adhesive capacity in most strains, while OPA Plus appeared to stimulate adhesion in certain strains.

Table 1: Adhesion Ability of *E. coli* Isolates Before and After Chemical Stress

Condition	Mean OD (630 nm)	Standard Deviation	Std. Error of Mean	P-value
Adhesion (Before Stress)	0.04356	0.01156	0.002043	—
After Stress with Dettol	0.03406	0.009742	0.001722	< 0.0001
After Stress with Surgical disinfectant OPA PLUS solution	0.04625	0.01224	0.002165	< 0.0001
** Highly significant at P < 0.0001				

Note: Highly significant at $P < 0.0001$ ** Microscopic Observation Microscopic analysis supported the quantitative findings.

Figure 1A shows strong adhesion (aggregative pattern) observed before treatment, while Figure 1B illustrates weak or absent adhesion following exposure to Dettol.

In Figure 1A, strong aggregative adhesion is evident, indicating intense colonization on epithelial surfaces. Following chemical treatment with Dettol, the adhesion pattern was notably diminished, as seen in Figure 1B. This visual evidence supports the OD findings and further validates the inhibitory effect of Dettol on UPEC adhesion behavior [21]. These findings align with the idea that certain disinfectants can exert varying degrees of selective pressure on bacterial populations, thereby modulating the expression of specific virulence factors such as adhesins. The reduction in adhesion observed after Dettol treatment supports the hypothesis that the active compound, chloroxylenol, not only has a bactericidal effect but also disrupts bacterial surface structures required for initial colonization. This is consistent with studies by McDonnell and Russell (1999), which showed that membrane-active disinfectants reduce fimbrial integrity and receptor-binding capacity [18]. Furthermore, the suppression of adhesive behavior could reflect damage to pili proteins such as FimH and PapG, both known to mediate tight binding to uroepithelial cells [19]. Conversely, the observed increase in adhesion following treatment with OPA Plus (surgical disinfectant) is a compelling indication that sub-lethal chemical exposure can trigger compensatory stress responses in bacteria. O'Toole et al. (2000) suggested that such responses often involve the upregulation of stress-related sigma factors and regulators, which may inadvertently activate genes associated with virulence, including fimbrial operons [20]. The increased OD and enhanced adhesion pattern post-treatment may represent an adaptive mechanism by which E. coli enhances its survival under unfavorable environmental conditions. This phenomenon is not unique to this study. Jahid and Ha (2012) reported similar

findings, noting that certain sanitizing agents at sub-inhibitory concentrations led to increased biofilm formation in foodborne pathogens [21]. They proposed that chemical exposure may act as a signaling cue, inducing the expression of genes associated with extracellular matrix production, stress tolerance, and attachment. Likewise, Bridier et al. (2011) demonstrated that ethanol and other alcohol-based disinfectants could paradoxically increase adhesion and biofilm formation when used at concentrations below MIC levels [22]. The microscopy results further reinforce these interpretations. Figure 1A, showing a strong aggregative adhesion pattern before chemical stress, reflects the inherent adhesive capability of untreated UPEC isolates. In contrast, Figure 1B, depicting a sparse adhesion pattern after exposure to Dettol, visually validates the observed statistical reductions. The visual disappearance or reduction in bacterial clusters confirms the inhibitory effect of Dettol on adhesion structures. However, OPA Plus-exposed isolates frequently exhibited increased bacterial clustering, especially in samples like 23 and 30, suggesting altered expression of aggregation-promoting surface molecules. Importantly, the dual nature of bacterial response – inhibition with Dettol and enhancement with OPA Plus – highlights the necessity for evaluating disinfectants not only for their bactericidal activity but also for their sub-lethal effects on bacterial physiology. For example, low-level exposure might result in increased expression of genes like papC, which encodes components of P fimbriae, or stress response regulators such as rpoS, contributing to adaptive phenotypes that enhance persistence and immune evasion [23, 24]. Moreover, the results bear clinical significance. In hospital environments where disinfectants are frequently used, particularly in suboptimal concentrations or improper application protocols, the potential for inducing bacterial adaptation should be critically assessed. The unintended consequence of enhanced adhesion may lead to increased biofilm formation on catheters, medical surfaces, or uroepithelial tissues, thereby complicating treatment and promoting antibiotic resistance. In conclusion, this study demonstrates the divergent effects of chemical stressors on the adhesive behavior of uropathogenic E. coli. While Dettol showed a clear reduction in adhesion, OPA Plus exposure led to a concerning increase in adherence levels. These findings underscore the complexity of microbial response to chemical agents and the importance of integrated antimicrobial stewardship approaches that consider both lethal and sub-lethal impacts. Results demonstrated a significant reduction in adhesion after Dettol treatment in most isolates, confirming its disruptive effect on surface adhesion factors [18]. Conversely, exposure to OPA Plus led to increased adhesion in some isolates, possibly indicating stress-induced upregulation of adhesin genes, such as fimH or papG, a phenomenon previously described under environmental stressors [19, 20]. Figure 1A shows strong adhesion (aggregative pattern) observed before treatment, while Figure 1B illustrates weak or absent adhesion following exposure to Dettol The present study investigated the adhesion behavior of uropathogenic Escherichia coli (UPEC) isolates collected from patients with urinary tract infections (UTIs)

in Ramadi City hospitals. One of the most important virulence mechanisms of UPEC is its capacity to adhere to uroepithelial cells, a key step for colonization, invasion, and biofilm formation [18]. The epithelial adhesion assay revealed that most isolates initially demonstrated weak to moderate adhesion patterns, consistent with prior reports indicating that UPEC commonly employs type 1 fimbriae (fimH) and P fimbriae (papG) to establish adherence to host epithelial surfaces [19,20]. After exposure to sub-inhibitory concentrations of Dettol (0.78%), the majority of isolates exhibited a significant reduction in adhesion (OD = 0.03406 vs. 0.04356 baseline, $p < 0.0001$), suggesting Dettol's effectiveness in disrupting bacterial cell wall structures and adhesin functionality. This is consistent with findings from Gant et al. (1996), who demonstrated that chloroxylenol, the active ingredient in Dettol, disrupts membrane integrity and protein conformation in Gram-negative bacteria [21]. Likewise, Okechukwu et al. (2020) confirmed the anti-adhesive action of Dettol against multidrug-resistant E. coli isolates [22]. In contrast, treatment with the surgical disinfectant OPA Plus resulted in a paradoxical increase in adhesion (OD = 0.04625), exceeding pre-treatment levels. This observation could be attributed to a bacterial stress response that leads to upregulation of adhesin-related genes, enabling bacteria to strengthen surface binding under sub-lethal conditions. This stress-induced hyper-adhesion has been documented in prior studies, such as by O'Toole and Kolter (2000), where nutrient stress enhanced fimbrial gene expression in UPEC [23], and by Jahid and Ha (2012), who noted increased biofilm formation in Salmonella enterica following alcohol-based sanitizer exposure [24]. Interestingly, Bridier et al. (2011) demonstrated that sub-inhibitory exposure to disinfectants could trigger increased expression of quorum sensing and adhesion genes in E. coli, further supporting our hypothesis [25]. These findings underscore the complex interplay between chemical stress and bacterial adaptation, especially within hospital environments where exposure to disinfectants is routine. The ability of UPEC to adapt and enhance adhesion under stress poses a critical challenge for infection control, especially in catheterized patients, where biofilm formation increases antimicrobial resistance [26]. Figures 1A and 1B provide visual evidence of these trends. Figure 1

A illustrates a classic aggregative adhesion pattern before treatment, characterized by dense bacterial clusters, while Figure 1B, post-Dettol treatment, shows a dispersed or absent adhesion profile, indicating disrupted interaction with host cells. Such microscopic observations mirror the statistical analysis and support Dettol's inhibitory role. Additionally, the variation in adhesion responses among isolates could be due to differential gene expression or strain-specific resistance mechanisms. For instance, studies by Sharma et al. (2018) and Meunier et al. (2021) revealed that fimH mutations can significantly alter bacterial affinity for host receptors, contributing to variable adhesion profiles among clinical isolates [27,28]. Further investigation using RT-qPCR could validate the upregulation of

adhesin genes following OPA Plus treatment. Similar molecular evidence was presented by Król et al. (2013), who documented that exposure to ethanol increased fimbrial gene expression in Enterobacteriaceae [29]. Such mechanisms underline the importance of understanding the molecular dynamics of stress responses in UPEC. The results of this study not only demonstrate the practical impact of disinfectants on bacterial adhesion but also align with WHO guidelines on preventing hospital-acquired infections (HAIs), which recommend regular rotation of antiseptic agents to prevent bacterial adaptation [30]. Our data may contribute to hospital policies on disinfection strategies, particularly in urology wards where UPEC prevalence is high.

To further contextualize our findings, we compared our results to other regional studies. For example, Al-Hadithi et al. (2020) found that Dettol significantly reduced adhesion in Klebsiella pneumoniae isolates from Baghdad hospitals, with patterns similar to those observed in our E. coli strains [31]. Moreover, a study conducted in Turkey by Aydin et al. (2021) noted increased adhesion after OPA exposure in Acinetobacter baumannii, paralleling our findings and reinforcing the need to re-evaluate reliance on certain disinfectants [32].In summary, this extended analysis underscores the dual nature of chemical disinfectants in influencing bacterial adhesion. While Dettol proves to be a potent agent for reducing adhesion, the unexpected stimulatory effect of OPA Plus necessitates cautious use and further exploration into its long-term impact on microbial virulence. These insights contribute to a broader understanding of microbial responses to hospital disinfectants and emphasize the need for molecular surveillance of virulence factors in high-risk environments.

The results of this study are consistent with those of 32] [who investigated the effect of ginger oil on plasmid content and biofilm formation in Staphylococcus aureus and Escherichia coli isolates isolated from teeth. This study demonstrated that ginger oil had the ability to reduce plasmid content and inhibit biofilm formation, suggesting that natural substances can influence bacterial virulence factors, including adhesion ability. This is consistent with our results, which showed a significant decrease in the adhesion ability of UPEC isolates after treatment with Dettol, demonstrating that the active ingredient (chloroxylenol) can disrupt surface proteins responsible for adhesion, such as FimH and PapG. On the other hand, the results are partially consistent with those of [33] who conducted molecular characterization of some virulence-related genes in E. coli O157:H7 isolates from bloody diarrhea and urinary tract infections. Their study focused on identifying genes such as stx1, stx2, and eae using PCR, and demonstrated that these genes are associated with increased virulence and adhesion ability. Although our study did not directly rely on molecular characterization techniques, the observed changes in adhesion pattern after exposure to disinfectants—particularly OPA Plus—may reflect the induction of

virulence genes associated with cell adhesion, such as fimH and papG, under chemical stress.

Conclusion

This study demonstrates that chemical stress can significantly alter the adhesion potential of UPEC isolates. Dettol effectively reduced adhesion, making it a potent agent against initial bacterial colonization. However, Surgical Spirit appeared to stimulate adhesion, possibly through stress-induced gene expression. Proper usage guidelines for disinfectants are crucial in clinical settings to minimize the risk of enhancing bacterial virulence.

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