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Inflammation: A Potential Association Between Cervical Cancer and UTI

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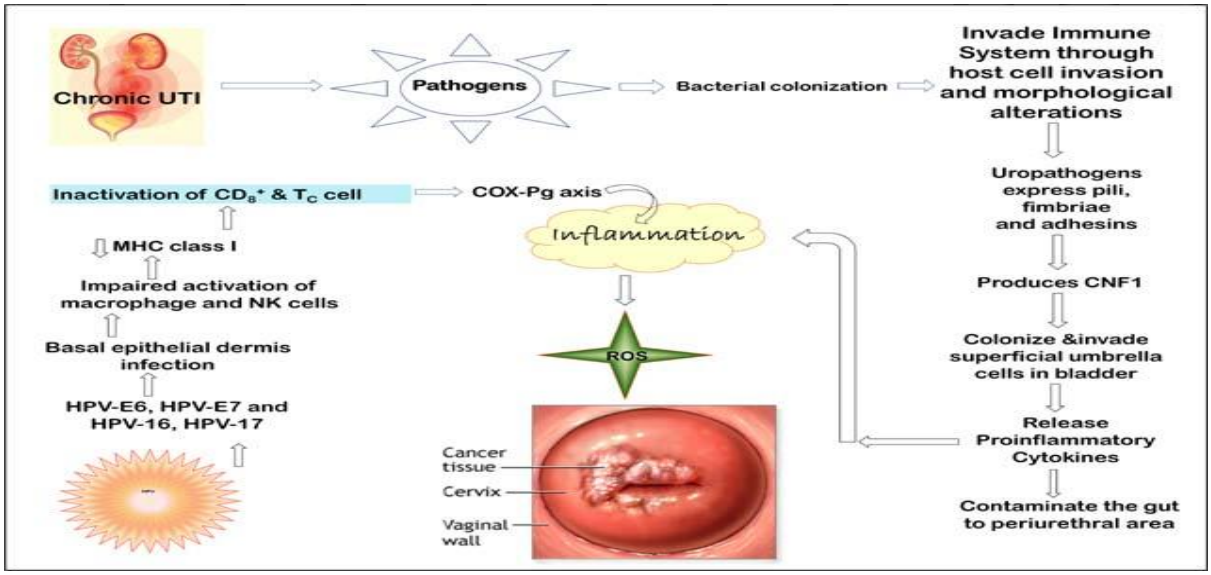
ABSTRACT

One of the most frequently diagnosed malignancies among females is cervical cancer. An escalation of cells in the cervix, located in the lower part of the uterus, connects to the vagina. Cervical cancer, a major cause of mortality and morbidity in young-adults and older-women, develops from a carcinogenic human-papillomavirus (HPV) in almost all patients. Several risk factors of HPV infection are shared with common urogenital-infective conditions, such as cystitis (lower urinary tract infection), vaginosis and vulvovaginitis, e.g., sexual behavior and certain vaginal microbiome structures. Urinary tract infections (UTIs), which are mainly due to bacterial infections such as those caused by *Escherichia coli*, are common in females and characterized by symptoms including lower abdominal soreness, frequent impulses to urinate and pain during urination. Cervical cancer and UTIs are two different diseases, but new research points to a possible connection in a retrospective cohort between chronic UTIs and a greater risk of cervical cancer. According to concurrent investigations, continual inflammation linked to repeat UTIs may produce CNF1, resulting in colonization and invasion of superficial umbrella cells in the bladder and significantly enhancing inflammation via proinflammatory cytokines. Concurrently, the COX-prostaglandin inflammatory axis provokes the inflammatory cascade mechanism in HPV infections. These two phenomena generate ROS to increase the risk of cancer-causing alterations in cervical cells. This review aims to explore the correlation between cervical cancer and UTIs as well as between HPV and *E. coli* which may abet the combined screening program of cervical cancer and UTIs in prevention and therapy.

Keywords: UTI, Inflammation, UPEC, Cervical cancer, HPV, ROS



INTRODUCTION

Microorganisms are crucial environmental factors in the progression of several vulnerable infections. Awareness of the multifactorial processes eventually results in combination with tumors and other bacteraemia which has increased considerably in prevalence in the recent years. Among them, the coinfection of cervical cancer, due to the persistent contagion of human-papillomavirus (HPV) and urinary tract infections (UTI), a probable cause of the emergence of antimicrobial resistance in *E. coli*, potentially boosts immunomodulation toward the generation of carcinoma. In such cases, inflammation may play a vital role in promoting immunotolerance against HPV and *E. coli* as well as against cervical cancer and UTIs.

Cervical Cancer

Cervical cancer, a commonly diagnosed cervical neoplasm, is caused mostly by persistent infection with a virus namely, human papilloma virus (HPV) and is the fourth leading cause of cancer mortality in women globally [1,2]. According to Singh et al. [3] cervical cancer in India, was the 2nd most common cancer in terms of both incidence (18.3%) and cancer mortality (18.7%) among women in 2020, with a 5-year prevalence of 18.8%. India accounts for approximately 1/5 of new cases and nearly 1/4 of deaths due to cervical cancer, making it a major contributor to the global burden of cervical cancer [2].

The cervix is anatomically the 1/3rd lower portion of the uterus and is cylindrical, protruding and linked with the vagina through the external orifice of the uterus [4]. The uterine cervix is divided into three regions, the ectocervix (nonkeratinized stratified squamous epithelium, containing stem-like cells and differentiated cells), the endocervix (columnar epithelium) and the squamo-columnar junction (metaplastic epithelium). The presence of a microwound in nonkeratinized stratified squamous epithelium is necessary for virions to access the basal layer and specifically contaminate stem-like cells. The infected stem-like cells now form a reservoir of contagion where the viral genome is maintained in an episomal state [5]. As the cells divide, the daughter cells are pushed toward the epithelial surface, giving rise to transient productive infections possibly progressing to high-grade neoplasia or squamous cell carcinoma [5,6,7].

HPV

Human papilloma viruses (HPVs) are small icosahedral viruses (~ 50-60 nm in diameter), that are nonenveloped with a circular double-stranded DNA genome, infect mucosal and skin epithelia in a specific manner and induce cell proliferation [7,8]. The cellular tropism of HPV is associated with cervical carcinogenesis [9] in the nonkeratinized stratified squamous epithelium of the ectocervix which expresses the epidermal growth factor receptor (EGFR) and the keratinocyte growth factor receptor [10], where the endocervix and the squamo-columnar junction have received less attention [10,11,12,13].

Genital HPV infection can be divided into two categories: ‘high-risk’ HPV types causing cervical intraepithelial neoplasia as well as cervical cancer and ‘low-risk’ HPV types causing genital warts [14]. The HPV genome comprises three functional regions: the long control region (LCR), the early region (E) encoding E1, E2 and E4-E8 and the late region (L) encoding L1 and L2 genes, in which the HPV-E6 and -E7 proteins are crucial to chronic inflammation as well as malignant transformation [15,16]. High-risk HPV genotypes, especially HPV 16 and HPV 18, are also well-established oncogenic factors in cervical carcinogenesis [17,18].

Persistent HPV infection leads to modifications in the release of proinflammatory cytokines, which in turn may alter the infiltration of immune cells, causing inflammation [19]. HPV-E6 and-E7, as well as HPV-16 and-18, impede the

interferon pathway, which in turn decreases the amount of major histocompatibility complex (MHC) class I molecules on the cell surface, resulting in inactivation of CD8+ cytotoxic T-cell responses [20]. Neoplastic epithelial cells of the cervical mucosa, are thus able to induce the cyclooxygenase (COX)-prostaglandin inflammatory axis [21], resulting in a direct link between HPV oncogenes and the activation of potent inflammatory cascades with well-known roles in cancer promotion via impaired activation of the IFN-mediated response and NF-κB [14,22,23]. Finally, the potential for HPV is to interfere with the migration and adhesion of innate immune cells. Thus, the functions of antigen-presenting cells (APCs), macrophages and natural killer (NK) cells can be downregulated by HPV infection [24,25,26,27,28]. This phenomenon results in an abnormal imbalance in immune responses which in turn causes cell transformation, inflammation and tumor progression initiated by HPV infection [29,30,31].

UTI

Urinary tract infections (UTIs) are potential causes of antimicrobial resistance, disturbing 150 million people each year worldwide in both healthcare and community settings and causing significant morbidity and mortality [32,33,34,35]. Among the various clinical manifestations of UTI (cystitis, pyelonephritis, asymptomatic bacteriuria and chronic and recurrent UTIs), cystitis is the most frequent presentation affecting the urinary bladder [33]. The bacteria may further accumulate in the urinary tract causing infection of the kidney [36]. The majority of cases of complicated UTIs are community-acquired by uropathogenic *E. coli* (UPEC) and *Klebsiella spp*., accounting for approximately 75–95% of the total cases [35,37].

E. coli

Escherichia coli (*E. coli*) is an important urinary tract pathogen and is responsible for considerable healthcare-associated problems worldwide [38,39]. *E. coli* possesses a wide variety of virulence factors, which help the organism infect and damage the host. Among them, *Escherichia coli* sequence type 131 (ST131) has recently emerged as an imperative UTI public health pathogen owing to its rapid spread worldwide and multidrug resistance [39]. The UPEC, a common pathogenic pathogen for UTIs, has the ability to adhere to, move, produce toxins, acquire metals, from intracellular bacterial communities (IBCs) and evade immune defenses [40].

The strong pathogenicity of bacteria imbalances the internal aspects of the urethral mucosa and epithelial cells and the defense function of the body is compromised [41,42]. Chronic UTIs are instigated when UPEC persuades periurethral invasion and colonization. Subsequent UPEC ascension into the bladder and expression of pili, fimbriae (type 1 fimbriae, P fimbriae, and other fimbriae) and adhesions (nonfimbrial adhesins) result in colonization and invasion of superficial umbrella cells in the bladder [40,41,43,44,45,46,47]. UPEC thus produces toxins (cytotoxic necrotizing factor 1: CNF1) that provoke host cell damage, release essential nutrients that promote bacterial survival, significantly increase in proinflammatory cytokines and contaminate the gut to periurethral area [48]. Subsequently, a series of inflammatory reactions, such as urethritis, cystitis and pyelonephritis [49,50,51], occur in the urinary tract.

Coinfection/the Bridge

Several reports suggest that insights into the coinfection of viruses and bacteria results in disease exacerbation [18,52,53,54,55]. Evidence from retrospective cohort studies is emerging that *E. coli*, the most common microorganism, dominates the pathogens isolated from the cervical discharge of patients diagnosed with cervical cancer [18,56]. The proportions of HPV 16 and *E.*

coli, amplified with increasing the severity of intraepithelial cervical neoplasia, indicating that there is a possible link or bridge in between cervical cancer and UTIs. In this context, Zou et al [18] reported that the largest proportion of *E. coli* isolated from HPV16 is phylotype B2, which more frequently carries virulence genes and induces cytotoxicity.

HPV oncogenes activate potent inflammatory cascades by impairing the activation of IFN-mediated responses and NF-κB which in turn interferes with the migration and adhesion of innate immune cells [14, 20, 21, 28, 31]. In these cases, the function of APCs, macrophages and NK cells are downregulated. This phenomenon in turn rationalizes the amount of MHC class I, resulting in the inactivation of CD8+ cytotoxic T-cell responses. The COX-prostaglandin inflammatory axis is then able to provoke the inflammatory cascade mechanism.

Potential overgrowth of *E. coli* also induces inflammation and enhances carcinogenesis. UPEC expresses pili, fimbria and adhesions and produces CNF1 which results in colonization and invasion of the superficial umbrella cells in the bladder and significantly enhances inflammation via proinflammatory cytokines to contaminate the gut to the periurethral area [40, 48, 51].

These data revealed that inflammation is a potent factor for coinfection or a bridge between cervical cancer or HPV and UTI or *E. coli*. Chronic inflammation triggers the production of reactive oxygen species (ROS) [57, 58,59,60], which accordingly promotes endothelial dysfunction via the oxidation of crucial cellular signaling proteins [61,62,63] and can damage DNA, leading to mutations and genomic instability to promote tumor growth and metastasis [64,65]. An imbalance between oxidants and antioxidant systems in cervical cancer is the predominant factor in its etiopathogenesis. Uterine cervical intraepithelial abnormalities and the development of cancer consequently influence the vulnerable metaplastic cervical epithelium resulting from complex molecular disturbances. The generation of free radicals as well as ROS causes genetic damage to the cervical epithelium, leading to its transformation into malignant cells and the initiation of cervical cancer [66,67].

CONCLUSION

The molecular imbalance plays an important role in the pathogenesis and progression of the coinfection between neoplasia and bacteremia. This review describes explanations for how cervical cancer and UTI is associated in relation to inflammation and becomes more pronounced because of the involvement of ROS. This study provides strong insight into HPV and *E. coli*, which may assist in the development of combined screening programs for the prevention and treatment of cervical cancer and UTI.

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Author contributions

Ritika Seth: conceptualization, writing - original draft. Sougata Sarkar: conceptualization, writing - original draft, Supervision. Sarmishtha Chatterjee: conceptualization, visualization, writing - review and editing.

Declarations

Ethics approval and consent to participate: Not applicable.

Consent for publication: Not applicable.

Competing interests: The authors declare that they have no competing interests.

REFERENCES

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, Bray F. Global Cancer Statistics 2020: GLOBOCAN Estimates of Incidence and Mortality Worldwide for 36 Cancers in 185 Countries. CA Cancer J. Clin. 2021; 71:209–249.

2. Ramamoorthy T, Kulothungan V, Sathishkumar K, Tomy N, Mohan R, BAlan S, Mathur P. Burden of cervical cancer in India: estimates of years of life lost, years lived with disability and disability adjusted life years at national and subnational levels using the National Cancer Registry Programme data. Reprod Health. 2024; 21:111. https://doi.org/10.1186/s12978-024-01837-7.
3. Singh D, Vignat J, Lorenzoni V, Eslahi M, Ginsburg O, Lauby-Secretan B, Arbyn M, Basu P, Bray F, Vaccarella S. Global estimates of incidence and mortality of cervical cancer in 2020: a baseline analysis of the WHO Global Cervical Cancer Elimination Initiative. Lancet Glob Health. 2023;11(2): e197–206.
4. Kurnia I, Rauf S, Hatta M, Arifuddin S, Hidayat YM, Natzir R, Kaelan C, Bukhari A, Pelupessy NU, Patelonggi IJ. Molecular Patho-mechanisms of cervical cancer (MMP1), Ann. Med Sur. 2022:77;103415. https://doi.org/10.1016/j.amsu.2022.103415.
5. Doorbar J, Quint W, Banks L, Bravo IG, Stoler M, Broker TR, Stanley MA. The biology and life-cycle of human papillomaviruses. Vac. 2012;30(5):F55–F70.
6. Griffin H, Soneji Y, Van Baars R, Arora R, Jenkins D, van de Sandt M, Wu Z, Quint W, Jach R, Okon K, Huras H, Singer A, Doorbar J. Stratification of HPV-induced cervical pathology using the virally encoded molecular marker E4 in combination with p16 or MCM. Mod Pathol. 2015 Jul;28(7):977–993. doi: 10.1038/modpathol.2015.52.
7. Rosendo-Chalma P, Antonio-Véjar V, Ortiz Tejedor JG, Ortiz Segarra J, Vega Crespo B, Bigoni-Ordóñez GD. The Hallmarks of Cervical Cancer: Molecular Mechanisms Induced by Human Papillomavirus. Biol. 2024;13(2):77. https://doi.org/10.3390/biology13020077.
8. WHO. IARC Working Group on the Evaluation of Carcinogenic Risks to Humans. In Human Papillomaviruses; WHO: Lyon, France, 1995; Volume 64. Available online: https://www.ncbi.nlm.nih.gov/books/NBK424408.
9. Walboomers JM, Jacobs MV, Manos MM, Bosch FX, Kummer JA, Shah KV, Snijders PJ, Peto J, Meijer CJ, Muñoz N. Human papillomavirus is a necessary cause of invasive cervical cancer worldwide. J Pathol.1999 Sep;189(1):12-9.
10. Ozbun MA, Campos SK. The long and winding road: Human papillomavirus entry and subcellular trafficking. Curr. Opin. Virol. 2021; 50:76–86.
11. Martens JE, Arends J, Van der Linden PJ, De Boer BA, Helmerhorst TJ. Cytokeratin 17 and p63 are markers of the HPV target cell, the cervical stem cell. Anticancer Res. 2004 Mar-Apr;24(2B):771-5. PMID: 15161025.
12. Herfs M, Yamamoto Y, Laury A, Wang X, Nucci MR, McLaughlin-Drubin ME, Münger K, Feldman S, McKeon FD, Xian W, Crum CP. A discrete population of squamocolumnar junction cells implicated in the pathogenesis of cervical cancer. Proc. Natl. Acad. Sci. U.S.A. 2012:109(26);10516-10521. https://doi.org/10.1073/pnas.1202684109.
13. Kines RC, Schiller JT. Harnessing Human Papillomavirus' Natural Tropism to Target Tumors. Vir. 2022;14(8):1656. https://doi.org/10.3390/v14081656
14. Nahand JS, Esghaei M, Monavari SH, Moghoofei M, Kiani SJ, Mostafaei S, Mirzaei H, Bokharaei-Salim F. The assessment of a possible link between HPV-mediated inflammation, apoptosis, and angiogenesis in Prostate cancer. Inter Immunopharmacol. 2020. 88. https://doi.org/10.1016/j.intimp.2020.106913.
15. Li G, He L, Zhang E, Shi J, Zhang Q, Le AD, Zhou K, Tang X. Overexpression of human papillomavirus (HPV) type 16 oncoproteins promotes angiogenesis via enhancing HIF-1α and VEGF expression in non-small cell lung cancer cells. Can Let. 2011:311(2);160-170. https://doi.org/10.1016/j.canlet.2011.07.012.
16. Ferlay J, Ervik M, Lam F, Laversanne M, Colombet M, Mery L, Piñeros M, Znaor A, Soerjomataram I, Bray F. Global Cancer Observatory: Cancer Today (version 1.1). Lyon, France: International Agency for Research on Cancer. 2024. https://gco.iarc.who.int/today.
17. Crosbie EJ, Einstein MH, Franceschi S, Kitchener HC. Human papillomavirus and cervical cancer. Lancet. 2013 Sep 7;382(9895):889-99. doi: 10.1016/S0140-6736(13)60022-7
18. Zou Q, Wu Y, Zhang S, Li S, Li S, Su Y, Zhang L, Li Q, Zou H, Zhang X, Wang T, Liang S, Yang J, Li C. Escherichia coli and HPV16 coinfection may contribute to the development of cervical cancer. Virulence. 2024 Dec;15(1):2319962. doi: 10.1080/21505594.2024.2319962. Epub 2024 Feb 21. PMID: 38380669; PMCID: PMC10883084.
19. Broccardo E, Lepique AP, Villa LL. The role of inflammation in HPV carcinogenesis.

Carcinogenesis. 2010; 31:1905–1912. doi: 10.1093/carcin/bgq176.
20. Senba M, Mori N. Mechanisms of virus immune evasion lead to development from chronic inflammation to cancer formation associated with human papillomavirus infection. Oncol Rev. 2012:5;6(2):e17. doi: 10.4081/oncol. 2012.e17.
21. Adefuye A, Sales K. Regulation of inflammatory pathways in cancer and infectious disease of the cervix. Scientifica (Cairo) 2012; 2012:548150. doi: 10.6064/2012/548150.
22. Trinchieri G. Innate inflammation and cancer: is it time for cancer prevention? F1000 Med Rep. 2011; 3:11. doi: 10.3410/M3-11.
23. Scott ME, Shvetsov YB, Thompson PJ, et al. Cervical cytokines and clearance of incident human papillomavirus infection: Hawaii HPV cohort study. Int J Cancer. 2013; 133:1187–1196. doi: 10.1002/ijc.28119.
24. Mota FF, Rayment NB, Kanan JH, Singer A, Chain BM. Differential regulation of HLA-DQ expression by keratinocytes and Langerhans cells in normal and premalignant cervical epithelium. Tissue Antigens. 1998;52((3)):286–93. doi: 10.1111/j.1399-0039. 1998.tb03046. x.
25. Hammes LS, Tekmal RR, Naud P, Edelweiss MI, Kirma N, Valente PT, et al. Macrophages, inflammation and risk of cervical intraepithelial neoplasia (CIN) progression--clinicopathological correlation. Gynecol Oncol. 2007;105((1)):157–65. doi: 10.1016/j.ygyno.2006.11.023.
26. Lepique AP, Daghastanli KRP, Cuccovia IM, Villa LL. HPV16 Tumor Associated Macrophages Suppress Antitumor T Cell Responses. Clin Cancer Res. 2009;15(13): 4391–4400.
27. Garcia TA, Ventura CL, Smith MA, Merrell DS, O'Brien AD. Cytotoxic Necrotizing Factor 1 and Hemolysin from Uropathogenic Escherichia coli Elicit Different Host Responses in the Murine Bladder. Infect Immun. 2013:81. https://doi.org/10.1128/iai.00605-12.
28. Nunes RAL, Morale MG, Silva GÁF, Villa LL, Termini L. Innate immunity and HPV: friends or foes. Clinics (Sao Paulo). 2018:11;73(suppl 1): e549s. doi: 10.6061/clinics/2018/e549s.
29. Bais AG, Beckmann I, Lindemans J, Ewing PC, Meijer CJ, Snijders PJ, Helmerhorst TJ. A shift to a peripheral Th2-type cytokine pattern during the carcinogenesis of cervical cancer becomes manifest in CIN III lesions. J Clin Pathol. 2005;58(10):1096-100. doi: 10.1136/jcp.2004.025072.
30. Kobayashi A, Weinberg V, Darragh T, Smith-McCune K. Evolving immunosuppressive microenvironment during human cervical carcinogenesis. Mucosal Immunol. 2008;1(5):412-20. doi: 10.1038/mi.2008.33.
31. Song D, Li H, Li H, Dai J. Effect of human papillomavirus infection on the immune system and its role in the course of cervical cancer. Oncol Lett. 2015;10(2):600-606. doi: 10.3892/ol.2015.3295.
32. Saha S, Nayak S, Bhattacharyya I, Saha S, Mandal AK, et al. Understanding the patterns of antibiotic susceptibility of bacteria causing urinary tract infection in West Bengal, India. Front Microbiol. 2014; 5:1–7. doi: 10.3389/fmicb.2014.00463.
33. Flores-Mireles AL, Walker JN, Caparon M, Hultgren SJ. Urinary tract infections: epidemiology, mechanisms of infection and treatment options. Nat Rev Microbiol. 2015;13(5):269-84. doi: 10.1038/nrmicro3432.
34. Borah VV, Saikia KK, Chandra P, Hazarika NK, Chakravarty R. New Delhi metallo-β-lactamase and extended spectrum β-lactamases co-producing isolates are high in community-acquired urinary infections in Assam as detected by a novel multiplex polymerase chain reaction assay. Indian J Med Microbiol. 2016; 34:173–182. doi: 10.4103/0255-0857.176853.
35. Mohapatra S, Panigrahy R, Tak V, J V S, K C S, Chaudhuri S, Pundir S, Kocher D, Gautam H, Sood S, Das BK, Kapil A, Hari P, Kumar A, Kumari R, Kalaivani M, R A, Salve HR, Malhotra S, Kant S. Prevalence and resistance pattern of uropathogens from community settings of different regions: an experience from India. Access Microbiol. 2022;4(2):000321. doi: 10.1099/acmi.0.000321.
36. Alós JI. Epidemiología y etiología de la infección urinaria comunitaria. Sensibilidad antimicrobiana de los principales patógenosy significado clínico de la resistencia. Enfermedades Infecciosasy Microbiología Clínica 2005; 23:3–8.
37. Kolawole AS, Kolawole OM, Kandaki-Olukemi YT, Babatunde SK, Durowade K, Kolawole CF. Prevalence of urinary tract infections (utiuti) among patients attending dalhatu araf specialist hospital, llafia, nnasarawa state, nnigeria. Int J Med Sciences 2009; 1:163–167.
38. Medina M, Castillo-Pino E. An introduction to

the epidemiology and burden of urinary tract infections. Ther Adv Urol. 2019; 11:3–7.
39. Kudinha T, Kong F. Possible step-up in prevalence for Escherichia coli ST131 from fecal to clinical isolates: inferred virulence potential comparative studies within phylogenetic group B2. J Biomed Sci. 2022; 29:78. https://doi.org/10.1186/s12929-022-00862-7.
40. Zhou Y, Zhou Z, Zheng L, Gong Z, Li Y, Jin Y, Huang Y, Chi M. Urinary Tract Infections Caused by Uropathogenic Escherichia coli: Mechanisms of Infection and Treatment Options. Inter J Mol Sci. 2023;24(13):10537. https://doi.org/10.3390/ijms241310537.
41. Totsika M, Moriel DG, Idris A, Rogers BA, Wurpel DJ, Phan MD, Paterson DL, Schembri MA. Uropathogenic Escherichia coli mediated urinary tract infection. Curr. Drug Targets. 2012; 13:1386–1399. doi: 10.2174/138945012803530206.
42. Terlizzi ME, Gribaudo G, Maffei ME. UroPathogenic Escherichia coli (UPEC) Infections: Virulence Factors, Bladder Responses, Antibiotic, and Non-antibiotic Antimicrobial Strategies. Front. Microbiol. 2017; 8:1566. doi: 10.3389/fmicb.2017.01566.
43. Bäckhed F, Alsén B, Roche N, Angström J, von Euler A, Breimer ME, Westerlund-Wikström B, Teneberg S, Richter-Dahlfors A. Identification of target tissue glycosphingolipid receptors for uropathogenic, F1C-fimbriated Escherichia coli and its role in mucosal inflammation. J. Biol. Chem. 2002; 277:18198–18205. doi: 10.1074/jbc.M111640200.
44. Nielubowicz GR, Mobley HL. Host-pathogen interactions in urinary tract infection. Nat. Rev. Urol. 2010; 7:430–441. doi: 10.1038/nrurol.2010.101.
45. Agarwal J, Srivastava S, Singh M. Pathogenomics of uropathogenic Escherichia coli. Indian J. Med. Microbiol. 2012; 30:141–149. doi: 10.4103/0255-0857.96657.
46. Melican K, Sandoval RM, Kader A, Josefsson L, Tanner GA, Molitoris BA, Richter-Dahlfors A. Uropathogenic Escherichia coli P and Type 1 fimbriae act in synergy in a living host to facilitate renal colonization leading to nephron obstruction. PLoS Pathog. 2011;7(2):e1001298. doi: 10.1371/journal.ppat.1001298.
47. Biggel M, Xavier BB, Johnson JR, Nielsen KL, Frimodt-Møller N, Matheeussen V, Goossens H, Moons P, Van Puyvelde S. Horizontally acquired papGII-containing pathogenicity islands underlie the emergence of invasive uropathogenic Escherichia coli lineages. Nat. Commun. 2020; 11:5968. doi: 10.1038/s41467-020-19714-9.
48. Chakraborty A, Adhikari P, Shenoy S, Saralaya V. Molecular characterisation of uropathogenic Escherichia coli isolates at a tertiary care hospital in South India. Indian J Med Microbiol. 2017;35(2):305-310. doi: 10.4103/ijmm.IJMM_14_291.
49. Garcia EC, Brumbaugh AR, Mobley HL. Redundancy and specificity of Escherichia coli iron acquisition systems during urinary tract infection. Infect. Immun. 2011; 79:1225–1235. doi: 10.1128/IAI.01222-10.
50. Conover MS, Ruer S, Taganna J, Kalas V, De Greve H, Pinkner JS, Dodson KW, Remaut H, Hultgren SJ. Inflammation-Induced Adhesin-Receptor Interaction Provides a Fitness Advantage to Uropathogenic, E. coli during Chronic Infection. Cell Host Microbe. 2016; 20:482–492. doi: 10.1016/j.chom.2016.08.013.
51. Pokharel P, Dhakal S, Dozois CM. The Diversity of Escherichia coli Pathotypes and Vaccination Strategies against This Versatile Bacterial Pathogen. Microorganisms. 2023; 11:344. doi: 10.3390/microorganisms11020344.
52. Griffiths EC, Pedersen AB, Fenton A, Petchey OL. The nature and consequences of coinfection in humans. J Infect. 2011;63(3):200-6. doi: 10.1016/j.jinf.2011.06.005.
53. Cougnoux A, Dalmasso G, Martinez R, Buc E, Delmas J, Gibold L, Sauvanet P, Darcha C, Déchelotte P, Bonnet M, Pezet D, Wodrich H, Darfeuille-Michaud A, Bonnet R. Bacterial genotoxin colibactin promotes colon tumour growth by inducing a senescence-associated secretory phenotype. Gut. 2014;63(12):1932-42. doi: 10.1136/gutjnl-2013-305257.
54. Pleguezuelos-Manzano C, Puschhof J, Rosendahl Huber A, van Hoeck A, Wood HM, Nomburg J, Gurjao C, Manders F, Dalmasso G, Stege PB, Paganelli FL, Geurts MH, Beumer J, Mizutani T, Miao Y, van der Linden R, van der Elst S; Genomics England Research Consortium; Garcia KC, Top J, Willems RJL, Giannakis M, Bonnet R, Quirke P, Meyerson M, Cuppen E, van Boxtel R, Clevers H. Mutational signature in colorectal cancer caused by genotoxic pks+ E. coli. Nature. 2020;580(7802):269-273. doi: 10.1038/s41586-020-2080-8.
55. Kaul D, Rathnasinghe R, Ferres M, Tan GS, Barrera A, Pickett BE, Methe BA, Das SR, Budnik I, Halpin RA, Wentworth D,

Schmolke M, Mena I, Albrecht RA, Singh I, Nelson KE, García-Sastre A, Dupont CL, Medina RA. Author Correction: Microbiome disturbance and resilience dynamics of the upper respiratory tract during influenza A virus infection. Nat Commun. 2020:16;11(1):3132. doi: 10.1038/s41467-020-17020-y. Erratum for: Nat Commun. 2020:21;11(1):2537. doi: 10.1038/s41467-020-16429-9.

56. Hou Y, Pan J, Kuerban G. Pathogenic bacterium and drug resistance in cervical cancer patients complicated with reproductive tract infection. Zhong Nan Da Xue Xue Bao Yi Xue Ban. 2016. Jul;41(7):721–728. doi: 10.11817/j.issn.1672-7347.2016.07.011.
57. Poyton R.O., Ball K.A., Castello P.R. Mitochondrial generation of free radicals and hypoxic signaling. Trends Endocrinol. Metab. 2009; 20:332–340. doi: 10.1016/j.tem.2009.04.001.
58. Reuter S, Gupta SC, Chaturvedi MM, Aggarwal BB. Oxidative stress, inflammation, and cancer: how are they linked. Free Radic Biol Med. 2010; 49:1603–16. 10.1016/j.freeradbiomed.2010.09.006.
59. Robb CT, Regan KH, Dorward DA, Rossi AG. Key mechanisms governing resolution of lung inflammation. Semin Immunopathol. 2016; 38:425–448.
60. Lennicke C, Cochemé HM. Redox metabolism: ROS as specific molecular regulators of cell signaling and function. Mol Cell. 2021;81(18):3691–3707. doi: 10.1016/j.molcel.2021.08.018.
61. Beckman JS. Oxidative damage and tyrosine nitration from peroxynitrite. Chem Res Toxicol 1996;9: 836–844.
62. Mittal M, Siddiqui MR, Tran K, Reddy SP, Malik AB. Reactive oxygen species in inflammation and tissue injury. Antioxid Redox Signal. 2014. 20:1126–67. 10.1089/ars.2012.5149.
63. de Almeida AJPO, de Oliveira JCPL, da Silva Pontes LV, de Souza Júnior JF, Gonçalves TAF, Dantas SH, de Almeida Feitosa MS, Silva AO, de Medeiros IA. ROS: Basic Concepts, Sources, Cellular Signaling, and its Implications in Aging Pathways. Oxid Med Cell Longev. 2022:19; 2022:1225578. doi:10.1155/2022/1225578.
64. Storz P. Reactive oxygen species in tumor progression. Front Biosci. 2005. 1;10:1881-96. doi: 10.2741/1667.
65. Chen H, Li Y, Li H, Chen X, Fu H, Mao D, Chen W, Lan L, Wang C, Hu K, Li J, Zhu C, Evans I, Cheung E, Lu D, He Y, Behrens A, Yin D, Zhang C. NBS1 lactylation is required for efficient DNA repair and chemotherapy resistance. Nat. 2024;631(8021):663-669. doi: 10.1038/s41586-024-07620-9.
66. Zahra K, Patel S, Dey T, Pandey U, Mishra SP. A study of oxidative stress in cervical cancer an institutional study. Biochem Biophy Rep. 2021:25. https://doi.org/10.1016/j.bbrep.2020.100881.
67. Despot A, Fureš R, Despot AM, Mikuš M, Zlopaša G, D'Amato A, Chiantera V, Serra P, Etrusco A, Laganà AS. Reactive oxygen species within the vaginal space: An additional promoter of cervical intraepithelial neoplasia and uterine cervical cancer development? Open Med (Wars). 2023:21;18(1):20230826. doi: 10.1515/med-2023-0826.

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