The vagina harbors a microbiota that is being increasingly recognized as protecting it from invading pathogens, including those that cause urinary tract infections and sexually transmitted diseases (1). The normal microbiota consists of a wide variety of genera and species, both anaerobic and aerobic, dominated by lactobacillus. Lactobacilli have been implicated as one factors controlling the growth of the other microorganisms in the vagina. Urinary infections constitute a worldwide problem that affects hundreds million women/year. These infections are a common reason for a woman's visit to a family practitioner or urologist, it has been estimated that each episode leads to 6 days of signs and symptoms (2). The main clinical outcome is morbidity and discomfort amongst a large percentage of the female population, in addition to enormous costs to the health care system for treatment (3). The incidence of UTI is higher among females, in whom it commonly occurs in an anatomically normal urinary tract. Conversely, in males and children, UTI generally reveals a urinary tract lesion that must be identified by imaging and must be treated to suppress the cause of infection and prevent recurrence. UTI can be restricted to the bladder (essentially in females) with only superficial mucosal involvement, or it can involve a solid organ (the kidneys in both genders, the prostate in males). Clinical signs and symptoms, hazards, imaging, and treatment of various types of UTIs differs. In addition, the patient’s background helps to further categorize UTIs according to age, type of urinary tract lesion(s), and occurrence in immunocompromised patients, especially with diabetes or pregnancy. Most UTIs are caused by bacteria, although fungi and viruses may occasionally be involved (4). UTIs may be classified by anatomic site of involvement or may be categorized as uncomplicated versus complicated. UTIs are divided into lower tract infections (cystitis, urethritis, prostatitis, and epididymitis) and upper tract infections (acute or chronic pyelonephritis). (5) The other method of classification divides UTIs into uncomplicated versus complicated infections. Uncomplicated UTIs generally occur in sexually active, non-pregnant adult women who do not have structural or functional abnormalities of the urinary tract. This is the more common type of UTI and is most responsive to antibiotics (5-6). Complicated UTIs, on the other hand, are harder to treat and occur in patients with structurally or functionally abnormal urinary tracts. This includes patients with renal stones, indwelling urinary catheters, prostatic hypertrophy, obstruction, or diabetes. Infections occurring in elderly, children, men, or pregnant women as well as hospital-acquired UTIs are also considered complicated (5-7).

Most pathogens responsible for UTI are enterobacteriaeae with a high predominance of Escherichia coli. This is especially true of spontaneous UTI in females (cystitis and pyelonephritis). Other strains are less common, including Proteus mirabilis and more rarely gram-positive microbes. Among the latter, Staphylococcus saprophyticus deserves special mention, as this gram-positive pathogen is responsible for 5% to 15% of such primary infections. Moreover, many patients have experienced a recurrence of symptoms, particularly within the first year of the original infection. Uropathogenic E. coli (UPEC) is the causative agent in 70%–95% of community acquired UTI and 50% of all cases of nosocomial infection (8). Vaginal colonization with E. coli appears to be an important determinant of UTI in women. Nevertheless, such colonization may be transient and not detectable in some women, even when they present with UTI. Compared with healthy controls, girls and women with a history of recurrent UTI have greater attachment of E. coli to their uroepithelial and vaginal epithelial cells and are more likely to be colonized with E. coli in the vagina between recurrences. In other women, however, the exogenous factors outlined below may predispose to recurrences. Some data suggest that cervicovaginal antibody may be deficient in women with recurrent UTI. E. coli strains vary in their abilities to adhere to uroepithelial and vaginal epithelial cells. Strains causing cystitis and especially pyelonephritis are more often P fimbriated than are strains causing asymptomatic bacteriuria or strains appearing in the faeces of healthy individuals. Thus, individuals harboring P-fimbriated strains in their faeces are presumably at much greater risk for UTI, especially pyelonephritis, than are those not harboring such strains. Several exogenous factors influence the adherence of E. coli to uroepithelial and vaginal epithelial cells and vaginal colonization with E. coli. Thus, antibiotic treatment frequently affects the vaginal microbiota, decreasing colonization with lactobacilli and increasing E. coli colonization. In fact, studies with primates and humans clearly show that certain antimicrobial agents, particularly amoxicillin and narrow-spectrum cephalosporins, facilitate vaginal colonization and/or UTI with E. coli, probably because they adversely affect anaerobic vaginal
flora more than other commonly used anti-UTI agents do. Moreover, hormonal status, sometimes, appears to facilitate E. coli adherence and colonization in premenopausal women, conversely, in postmenopausal women, replacement topical oestrogen clearly normalizes the vaginal flora and greatly reduces the risk of UTI. (9) In recent years, there has been an increasing recognition of the role of lactobacilli in the maintenance of the homeostasis within dynamic ecosystems such as the vagina and in the prevention of colonization and infection caused by pathogenic organisms (10). Has previous stated, Lactobacilli are important components of the normal vaginal microbiota, in fact, they help to repel invading pathogens and may also prevent urinary tract infections by interfering with the colonization of the periurethral epithelium by uropathogens such as E. coli. Thus, a loss of vaginal lactobacilli may predispose women to the acquisition of genitourinary infections (1, 6, 9).

In conclusion, several strategies can be used to decrease the risk of UTI by reducing vaginal colonization with uropathogens, but when antimicrobial agents are prescribed for the treatment, not only the antimicrobial spectrum, but also the presumed ecological disturbance on the anaerobic and aerobic vaginal and rectal microbiota, should be taken into a consideration (11). On the basis of these findings the use of fluroquinolones is appropriate and advisable (9, 11-12).

References