International Journal of Research and Review
www.ijrrjournal.com E-ISSN: 2349-9788; P-ISSN: 2454-2237

Review Article

Pregnancy and Hormonal Effects on Urinary Tract Infections in Women: A Scoping Review

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ABSTRACT

Urinary tract infections (UTI) are among the most common bacterial infections in humans usually caused by Escherichia coli. In our body, estrogen and progesterone hormones play a very important role in the urinary tract. Cyclical variations in these levels during the menstrual cycle changes urodynamics and cause lower urinary tract symptoms. The aim of the review was to know the effect of hormones in women and in pregnant ladies on UTI. Many publications were searched using the keywords in particular databases like PubMed, Trip database etc. Suitable articles were explored by three-stage screening. Decreased estrogen levels cause changes in the vaginal flora and increase the colonization of bacteria. While increased progesterone levels antagonize estrogen actions and cause the ureters muscle tone to relax. This decreases the voiding of urine and its flow leading to the infection. In pregnancy, ureteral dilatation starts at the beginning of the first trimester and develop hydronephrosis gradually. This is due to hormonal changes, primarily by progesterone. As the uterus starts growing, mechanical compression of the urinary tract begins and has more incidence of infections. Unresolved UTI's may cause many complications in pregnant women as well as in neonates. A decrease in estrogen and an increase in progesterone levels can cause urinary tract infections. Pregnancy can enhance the risk of infections from the end of the first trimester through the mechanical compression and through progesterone release.

Keywords: urinary tract infections, estrogen, progesterone, pregnancy, bacteriuria

INTRODUCTION

After the kidney filters the blood plasma, they return most of the water and solutes to the bloodstream. The remaining water and solutes constitute urine**,** which passes through the ureters and is stored in the urinary bladder until it is excreted from the body through the urethra. [1, 2]

EFFECTS OF HORMONES ON THE URINARY TRACT IN WOMEN

The female genital and lower urinary tracts share a common embryological origin, arising from the urogenital sinus and both are sensitive to the effects of the female sex steroid hormones throughout life. The female lower urinary tract is thought to be a target organ for the action of the sex steroid hormones estrogen and progesterone.

Estrogen

Connolly et al [3] found that estrogen was linked to decreased peristalsis of the ureters, thus allowing urine to pool in the already dilated ureters. Estrogen is known to have an important role in the function of the lower urinary tract and estrogen and progesterone receptors have been demonstrated in the vagina, urethra, bladder and pelvic floor musculature. [4-7] In addition, estrogen deficiency occurring following the menopause is known to cause atrophic change and may be associated with lower urinary tract symptoms such as

frequency, urgency, nocturia, urgency incontinence and recurrent infection. [4, 6, 7] This may also co-exist with symptoms of urogenital atrophy such as dyspareunia, itching, vaginal burning, and dryness. [4, 7]

Progesterone

Progesterone brings about relaxation of smooth muscle in the urinary system. [4] The well-recognized physiologic hydroureter, an increased bladder capacity, and an increased incidence of genuine stress incontinence during pregnancy are all felt to be due to the effects of progesterone. [5] These changes in structure and function are attributed to the relaxative effect of progesterone on the smooth muscle of the urinary system. [4, 6] It has also been demonstrated that in adult females there is an increase in bladder tone during the follicular phase and a decrease in tone during the luteal phase when progesterone is the predominant hormone. [7] Batra et al [8] noted that estrogen caused an increase in blood flow to the urethra over controls. However, when progestational agents were added along with the estrogen, the increase was significantly less, although still greater than controls. Hence, they proved that the estrogen acts as an antagonist to progesterone.

Effects of Pregnancy on Urinary Tract

The pathophysiology of this condition is ambiguous. [9] Patterson and Andriole [10] attribute the development of UTIs in pregnancy to physical and hormonal changes that occur in the urinary tract. They reported that hydroureter (the dilation of the renal pelvis and ureters to accommodate increased circulatory volume) can begin as early as 7 weeks gestation and progressively worsen until term. These dilated ureters can hold as much as 200 cc of urine, contributing significantly to the development of bacteriuria. [9, 10] Increased bladder volume and decreased bladder tone, along with decreased ureteral tone, contribute to increased urinary stasis and ureterovesical reflux. [11]

Factors predisposing to bacteriuria and UTI comprise hormonal changes and influence of mechanical factors during pregnancy which result in:

1. Relative stasis of urine in ureters;
2. Impaired emptying of the urinary bladder;
3. Increased bladder residual volume and increased prevalence of vesicoureteral reflux;
4. The increase of urine pH. [12]

Urinary tract infections (UTI) are among the most common bacterial infections in humans. UTI is commonly diagnosed based on clinical findings of bacteriuria (bacteria in midstream urine) counts of > 105 colony forming units (CFU)/mL along with patient-reported symptoms. Lower bacterial counts are considered clinically significant when urine is collected by catheterization. Cystitis, or infection of the bladder, is typically accompanied by painful urination (dysuria), urgency, and frequent urination. A more severe infection of one or both kidneys, called pyelonephritis, is often accompanied by fever and flank pain, often in addition to symptoms of cystitis. [11, 13, 14]

METHODOLOGY

Search strategy and databases:

We conducted a systematic literature review, with eligibility criteria and search strategy based on the Cochrane library. The databases searched are PubMed, Trip database, Science direct and Sage Journal. A two-phase search strategy was performed with an initial search to establish primary search terms followed by a second systematic search in all relevant databases using the search terms. The search included the following keywords: urinary tract infection, pregnancy, hormones, management. This search was conducted to identify relevant primary articles and studies on UTI. Studies which were published from 2013(last 5 years) are searched and collected. A Systematic search of the literature was also conducted to identify relevant systematic reviews (only recent reviews of potential relevance were considered).

Inclusion criteria

Type of study: Randomized control trials, observational studies, and systematic reviews were considered.

Study population: All Pregnant women irrespective of trimesters and post-menopausal women.

Type of interventions: Interventions related to the diet and lifestyle changes in UTI included. We also included studies which reported data on the effects of hormones on the urinary tract structure, function and also the reasons for the prevalence of UTI in post-menopausal women. Reports related to the effects of UTI on neonates were also included.

Exclusion criteria

Exclusion criteria include Pregnant women with co-morbidities including diabetes mellitus, hypertension, renal failure, placental hemorrhage and studies on surgical treatments, reviews like narrative reviews, opinions or editorials, reports published as meeting abstracts and studies based on the diagnosis of UTI. We also excluded studies related to recurrent urinary tract infections in men and younger/older women. Papers focused on UTI along with other co-morbid conditions like a polycystic ovarian disease, sepsis, and other cardiovascular conditions were also excluded.

Data extraction

Data extraction included Information about 1) Study information (demographic details and year), 2) Type of study (Systematic review, RCTs and Meta-analysis), 3) Intervention (diet and lifestyle changes), 4) Participants (pregnant and post-menopausal women), 5) Search strategy (search terms, inclusion and exclusion criteria).

DISCUSSION

EFFECTS OF ESTROGEN

Green et al [15] stated that the classic estrogen receptor (ER a) was first discovered in 1958 and was not cloned from uterine tissue until 1986 whilst Kuiper et al [16] stated that the second estrogen receptor (ER b) was identified in 1996. Warner et al [17] stated that ER a appears to play a major role in the regulation of reproduction while ER b has a more minor role. Chen GD et al [18] reported that the distribution of estrogen receptors throughout the urogenital tract with both a and b receptors being found in the vaginal walls and uterosacral ligaments of premenopausal women although the latter was absent in the vaginal walls of postmenopausal women. Estrogen receptors have also been demonstrated throughout the lower urinary tract and are expressed in the squamous epithelium of the proximal and distal urethra, vagina and trigone of the bladder. [19, 20] Jones et al [21] conducted a research study on 90 women undergoing surgery for genuine stress incontinence by cystoscopy and biopsy of the lower urinary tract at the time of their surgery. Six 3 mm cystoscopic punch biopsies were obtained from each woman from the bladder dome, trigone, proximal urethra, distal urethra, vagina, and vesicovaginal fascia at the level of the bladder neck. These tissues were analyzed for estrogen and progesterone receptor expression using histochemical scoring (H-score) system. Differences in overall tissue positivity were assessed using Fisher's exact test, while differences in H-scores were assessed using the nonparametric Mann Whitney U test. He concluded that in the subepithelial tissues of the vagina ER positivity was significantly higher in post-menopausal women not receiving HRT than in both pre-menopausal women and women receiving estrogen supplementation ($P < 0.05$). There was no receptor expression in the deeper tissues. There was significantly higher PGR positivity in the squamous epithelia of the premenopausal and HRT groups than in the postmenopausal group ($P< 0.01$);($P< 0.05$ urethra, $P < 0.01$vagina). PGR is an estrogen-dependent protein and has cyclical variation in expression in the endometrium. Estrogen is known to have an important role in the function of the lower urinary tract throughout adult life. [22, 23] Estrogen is also known to have a direct effect on detrusor function through modifications in

muscarinic receptors and by inhibition of movement of extracellular calcium ions into muscle cells. [23-25]

Neurologic control

Maggi et al [26] stated that the Sex hormones influence central neurologic control of micturition, although their exact role in the micturition pathway is not known. ERs have been shown to be present in the cerebral cortex, limbic system, hippocampus, and cerebellum.

Bladder function

ERs, though absent in the transitional epithelium at the dome of the bladder, are present in areas of the trigone that have undergone squamous metaplasia. Estrogen directly affects detrusor function through modifications in muscarinic receptors and by inhibition of the movement of extracellular calcium ions into muscle cells. [24, 25] Consequently, Shenfield OZ et al [27] Fantl et al [28] stated that estradiol reduces the amplitude and frequency of spontaneous rhythmic detrusor contractions, and there is evidence that it may increase the sensory threshold of the bladder in some women.

Urethra

Rud T [29] through his experiments suggested that estrogen increases urethral closure pressure and improves pressure transmission to the proximal urethra actions that promote continence. Estrogens cause vasodilatation in the systemic and cerebral circulation, and these changes also occur in the urethra. [30, 31] Versi et al [32] conducted a research study and concluded that the urethral pressure profilometry shows vascular pulsations, secondary to blood flow in the urethral submucosa and urethral sphincter that increase in size after estrogen administration but disappear after menopausal estrogen withdrawal. The urethral vascular bed is thought to account for about 33% of the urethral closure pressure, and HRT in postmenopausal women with SUI has been shown to increase the number of periurethral vessels.

Collagen

Jackson et al [33] through his study reported that the estrogen influences collagen synthesis and directly affects the collagen metabolism in the lower genital tract. Urogenital atrophic changes in women may result from an alteration in systemic collagenase activity, and SUI as well as urogenital prolapse are associated with reduced vaginal and periurethral collagen. Skin collagen content is reduced after menopause: rectus muscle fascia becomes less elastic with increasing age so that less energy is required to cause irreversible damage. Collagen composition also changes; hydroxyproline content in connective tissue from women with SUI is 40% lower than incontinent control subjects. [34]

Estrogen Effects on Urinary Tract Infections

Estrogens may affect continence by increasing urethral resistance, raising the sensory threshold of the bladder or increasing an adrenoreceptor sensitivity in the urethral smooth muscle. [35, 36] Robinson et al [4] in his research reported that the Changes in the vaginal flora due to estrogen depletion lead to colonization with Gram-negative bacilli which in addition cause local irritative symptoms. These microbiological changes may be reversed with estrogen replacement following the menopause, offering a rationale for treatment and prophylaxis. Estrogens play an important role in the continence mechanism with bladder and urethral function becoming less efficient with age. [37] In his study, Malone lee [38] found that Elderly women have a reduced flow rate, increased urinary residuals, higher filling pressures, reduced bladder capacity, and lower maximum voiding pressures. These all changes in the urinary tract cumulatively increase the chances of urinary tract infections.

EFFECTS OF PROGESTERONE

Progesterone receptors

Batra et al [23] conducted experiments and demonstrated that progesterone receptors are expressed in the

lower urinary tract although their role is less clear. These are demonstrated to be in the vagina, urethra, bladder, and pelvic floor musculature and the binding sites in the bladder had a broader hormonal specificity than those in the urethra or vagina. The concentration and the effect of progesterone in the urethra were four-fold higher than that in the bladder. It was also reported in the same study that the concentration of progesterone receptors in the vagina is similar to that of the concentration and specificity in the urethra. Peck et al [39-41] in the study stated that Progesterone receptors have been found in almost all estrogen target tissues, and progesterone is generally looked upon as an antagonist to estrogen. The antagonistic effect of progesterone in estrogen-induced responses is thought to be mediated by a reduction in estrogen receptors caused by progesterone. This reduction in estrogen receptor is in turn probably mediated by the genomic effects of progesterone exerted through its own receptor. In a research study, Smith et al [42] reported that Progesterone receptors have also been identified in the levator ani and pelvic ligaments, but their role has yet to be elucidated.

Anatomical and functional effects on urinary tract

Progesterone also inhibits estrogen action, including cell proliferation in the endometrium. [43] Clayton et al [44] evaluated the effects of progesterone on the bladder in rhesus monkeys over a 90 day period. During cystometry, they found an increase in the bladder volume to pressure ratio during filling, suggesting an increase in compliance. It has also been demonstrated that in adult females when progesterone is the predominant hormone, there is an increase in bladder tone during the follicular phase and a decrease in tone during the luteal phase. [45] Therefore, it appears that progesterone may cause a relaxation of vesical smooth muscle as noted by increased capacity and compliance. This has been demonstrated in pregnancy, under the influence of exogenous progesterone, and during the luteal phase of the menstrual cycle. [6, 8] Fuchs et al [46] reported that Progesterone produces relaxation of the uterine Smooth muscle by inducing beta-adrenergic receptor formation.

PROGESTERONE EFFECTS ON URINARY TRACT INFECTIONS

Francis [47] and Beck et al [48] exclaimed that up to 60% of pregnant women report stress incontinence symptoms at some time during pregnancy, and it has been suggested that this may be due to elevated levels of progesterone. It is also demonstrated that high levels of progesterone hormone decrease the muscle tone of ureters. This causes the ureters to dilate thus reducing the flow of urine and void. In patients with PCOS, there is a relative decrease in progesterone secondary to their anovulatory status, and they postulated this as the cause for their findings. [6] This increase in progesterone may cause urinary incontinence and urinary tract infections.

EFFECTS OF PREGNANCY ON URINARY TRACT INFECTIONS

Physiological changes occur to a varying extent in the urinary tract of pregnant women from the seventh week of gestation. These changes progress to delivery and resolve by the second postpartum month. [49] Carol et al [50] conducted a comprehensive review and reported that the five most common complications in pregnancy were anemia, hypertensive disorders of pregnancy, urinary tract infections, pelvic and perineal trauma occurring at delivery, and mental health conditions. Goto et al [51] in his research study stated that acute pyelonephritis is one of the most serious complications among urinary tract infections in pregnancy. Acute pyelonephritis is an upper urinary tract infection involving the renal pelvis, calcies, and parenchyma. Clinical manifestations include fever, nausea/ vomiting, or flank pain with or without cystitis symptoms. During pregnancy, anatomical and

physiological changes occur in the urinary tract including dilation of renal pelvis and ureter, displacement of the bladder, and mechanical compression of the ureters by the uterus. As a result, urinary stasis and vesicoureteral reflux predispose the patient to acute pyelonephritis.

ASB during pregnancy is influenced by a range of physiological and anatomical factors, including mechanical compression and changes in the immune and renal systems. [11] The incidence of uncomplicated recurrent UTIs increases with age. [53] Nicolle et al [54] through his research concluded that pregnant women are at increased risk of bacterial ascension to the kidneys and pyelonephritis, due partly to dilation of the renal pelvis and ureters by as early as the eighth week of pregnancy. [55] Schnarr et al [56] in his study explained that bacteriuria that progresses to pyelonephritis during pregnancy is associated with poor outcomes for both the mother and child, including maternal sepsis and anemia, preterm birth (PTB) low birth weight (LBW), and perinatal death. Even without progression to pyelonephritis, bladder infection during pregnancy is associated with increased risk of maternal hypertension, anemia, amnionitis, and premature labor, as well as PTB, and LBW. Recently, Bolton et al [57] reported in his studies that using a mouse model have provided even more compelling evidence for a causal relationship between UTI and adverse pregnancy outcomes. Experimental UTI in pregnant mice was sufficient to cause intrauterine growth restriction and resulted in significantly reduced litter size.

According to the studies, perinatal mortality due to urinary tract infections is influenced by metabolic and physiological events occurring in the mother. [58-60] Andriole [61] and Marchant [62] stated that effective management of bacteriuria during pregnancy reduces this risk; however, in approximately 1 percent of women, bacteriuria develops later in pregnancy and may be missed by screening cultures. Serial urine cultures during the third trimester may be useful in detecting these cases. [53] Batra et al [63] and Khan-Dawood et al [64] reported that the levels of both progesterone and estrogen in blood increase very substantially during pregnancy, the concentration of estrogen in uterine tissue remains low and estrogen receptors are not even detectable. This suggests that during pregnancy progesterone action predominates at cellular and receptor level resulting in suppression of estrogen receptor. Provided that this kind of antagonism between estrogen and progesterone at the receptor and genomic levels are also operative in the lower urinary tract, the estrogen receptor concentration in the urethra during pregnancy would, as in the myometrium, be greatly depressed. *Escherichia coli* is responsible for approximately 80 percent of all community-acquired UTIs in pregnancy, although other pathogens, such as *Klebsiella pneumoniae, Proteus mirabilis,* and *Enterococcus faecalis*, are commonly seen, because of *E. coli's* susceptibility to amoxicillin, 63 percent of studies involved the use of ampicillin or amoxicillin. [65]

EFFECTS OF UTI ON NEONATES (COMPLICATIONS)

The maternal and neonatal complications of a UTI during pregnancy can be devastating. Thirty percent of patients with untreated asymptomatic bacteriuria develop symptomatic cystitis and up to fifty percent develop pyelonephritis. [66] Asymptomatic bacteriuria is also associated with intrauterine growth retardation and low birth weight infants. [67] Schieve and associates conducted a study involving 25,746 pregnant women and found that the presence of UTI was associated with premature labor (labor onset before 37 weeks of gestation), hypertensive disorders of pregnancy (such as pregnancy-induced hypertension and preeclampsia), anemia (hematocrit level less than 30 percent) and amnionitis. [68] While this does not prove a cause and effect relationship, randomized trials have demonstrated that antibiotic treatment decreases the incidence of preterm birth and low birth weight

infants. [69] A risk of urosepsis and chronic pyelonephritis was also found. [70] In addition, acute pyelonephritis has been greatly associated with anemia. [71] Pfau A and Sacks TG [92] have outlined the outcome of UTI complications in neonates and in mother as:

- **Perinatal:**
  - Low birth weight (weight less than 2,500 g [5 lb, 8 oz])
  - Prematurity (less than 37 weeks of gestation at delivery)
  - Preterm low birth weight (weight less than 2,500 g and less than 37 weeks of gestation at delivery)
- **Maternal:**
  - Premature labor (less than 37 weeks of gestation at delivery)
  - Hypertension/preeclampsia
  - Anemia (hematocrit level less than 30%)

UTI in pregnancy, whether symptomatic or asymptomatic, requires treatment. The potential adverse effects of bacteriuria in the mother, such as persistent bacteriuria, symptomatic UTI, and acute and chronic pyelonephritis, as well as fetal adverse effects, such as increased frequency of premature delivery, low birth weight, and fetal infection, must be minimized with appropriate antibiotic treatment. [11, 65]

MANAGEMENT

Pharmacological treatment

Brumfitt et al [72] stated that each patient management strategy branch should be modeled by splitting the symptomatic population according to the presence of UTI, using information from prevalence studies. Gleckman [13] reported that, for about 30% of women in whom an infectious agent cannot be identified, antibiotic treatment is of no proven value.

Antibiotic treatment for UTIs in pregnancy is essential. Up to 30% of pregnant women with UTIs can develop acute pyelonephritis if not treated. [73] In a randomized, placebo-controlled trial, Kass [74] demonstrated that antibiotic treatment of ASB successfully eliminated bacteriuria and completely prevented acute pyelonephritis in pregnant women, while untreated ASB led to pyelonephritis in 40% of women receiving placebo. Prolonged antimicrobial prophylaxis effectively reduces the number of symptomatic recurrences of urinary tract infection but does not correct the predisposing defect. Nitrofurantoin is the most effective antimicrobial for the treatment of ASB. [52] All individuals presenting with symptoms of UTI receive a three-day course of general antibiotics called empiric treatment. [75] C-reactive protein is an acute phase protein widely used as an indicator of infectious or inflammatory conditions. Currently, C-reactive protein is used in the management of chorioamnionitis, preterm premature rupture of membranes, pelvic inflammatory disease, and urinary tract infection. [76] Many single-dose regimens exist to treat uncomplicated UTIs in pregnancy. Although Patterson et al [10] have reported that single-dose regimens are less effective (cure rate 50%-60%) when compared to 3 to 7 day regimens (cure rate 80%-90%), Krcmery et al [12] in a review of multiple studies, suggest that single-dose therapy can be as effective as 3-5, and 7-day courses of treatment during pregnancy in women with acute uncomplicated cystitis; however, women with recurrent UTI, women with pyelonephritis, or women who have resistant uropathogens should be given 7 to 10 days of treatment.

All pregnant women should be screened for bacteriuria and subsequently treated with antibiotics such as nitrofurantoin, sulfisoxazole or cephalexin. Ampicillin should no longer be used in the treatment of asymptomatic bacteriuria because of high rates of resistance. Alternatively, cephalosporins are well tolerated and adequately treat the UTI. Fosfomycin is a new antibiotic that is taken as a single dose. Sulfonamides can be taken during the first and second trimesters but, during the third trimester, the use of sulfonamides carries a risk that the infant will develop kernicterus,

especially the preterm infants.[77,78] Other common antibiotics (e.g., fluoroquinolones and tetracyclines) should not be prescribed during pregnancy because of possible toxic effects on the fetus. [11] Treatment should be followed by a repeat urine culture to confirm the clearing of the organism in the tract. [78]

Non- pharmacological treatment

Dietary changes

Stapleton [79] in his study demonstrated other methods for UTI prevention. These include consumption of cranberries, blueberries, and other acidic fruits, which are believed to inhibit bacterial adherence to uroepithelial cells in-vitro. Some believe that tannins contained in the fruits aid in preventing fimbriated bacteria such as *E. Coli* from implanting into tissue. While Miller et al [80] in his studies contradicted that the use of cranberry juice for UTI, as studies on cranberry juice, did with nonpregnant females, have failed to show an overall decrease in asymptomatic bacteriuria or a decrease in symptomatic UTI. Griffiths [81] has reviewed the literature on UTI and cranberry juice and reported that there is insufficient research to support treating UTI with cranberry juice. While Foxman et al [82] demonstrated that Cranberry juice is thought to act by reducing bacterial adherence to the bladder wall and regular intake of at least 300 ml a day has been associated with a reduced risk of urinary tract infections. The incidence of bacteriuria in those taking cranberry juice was 42% of those in the control group and they were also found to be four times more likely to clear bacteria spontaneously. However, Beerepoot [83] conducted more recently a randomized trial comparing trimethoprim prophylaxis with cranberry capsules in 221 pre-menopausal women and reported that they had a complaint of recurrent lower urinary tract infection. Bosmans et al [84] in his study demonstrated that the Overall trimethoprim was associated with a lower incidence of symptomatic infection and a subsequent cost-effectiveness analysis has also shown that cranberry tablets are not a cost-effective means of prophylaxis. Dasgupta et al [85] conducted a research study and demonstrated that Carbonated drinks also contain preservatives and anti-oxidants including ascorbic acid and citric acid and these have been shown to augment bladder muscle contraction by enhancing Ca2+influxwhile ascorbic acid has been shown to increase presynaptic neurotransmitter release. Consequently, the consumption of carbonated drinks may be associated with the development, or aggravation, of OAB symptoms and lower UTI symptoms. [86] Srikrishna S et al [87] in his study reported that the effect of carbonated drinks on urinary symptoms has been investigated in 20 asymptomatic volunteers in a four-way crossover study comparing carbonated water, Diet Coke, caffeine-free Coke, and Classic Coke. There was a significant increase in frequency with Diet Coke and caffeine-free Coke compared with carbonated water and Classic Coke. Urinary urgency was also significantly increased with Diet and caffeine-free Coke compared to carbonated water and there was a smaller increase with Classic Coke. Overall those drinks containing artificial sweeteners were associated with an increase in frequency, urgency severity, and urgency episodes.

A prospective cohort study conducted by Jura et al [88] on 65,176 women aged 37–79 years from the Nurse's Health Study and Nurse's Health Study II has demonstrated a weak dose-dependent positive association between caffeine consumption and urgency incontinence although not for mixed and stress incontinence with the attributable risk of urgency incontinence associated with caffeine was reported as 25%. There was no such effect with decaffeinated coffee. Decreasing dietary fat may account for some of the benefits of weight loss in women with urinary symptoms and dietary manipulation may be useful as a form of conservative management in these patients. [86] Decreasing fluid consumption significantly decreased voiding frequency,

urgency and incontinence episodes in patients with detrusor overactivity, and/or urodynamic stress incontinence. [89] The possibility of dietary modification may have an important, and cost-effective, role in primary prevention of lower urinary tract symptoms in women and may also offer an additional form of conservative therapy to be used alongside lifestyle factors such as weight loss and fluid modification before considering pharmacological treatment. [86]

Lifestyle modifications

A healthy lifestyle may be one of the most important factors in avoiding lower urinary tract symptoms. [86] Olds et al [90] in their research suggest two other preventive methods for UTI, both of which are well known to nurses: (1) avoiding bladder irritants such as caffeine and carbonated beverages, and (2) teaching women about wiping from front to back to avoid spreading anal bacteria to the urethra.

Stapleton et al [91] cite several methods to prevent UTIs:

1. Increase fluid intake to at least eight glasses per day to maintain bladder hygiene.
2. Improve voiding habits by always responding to initial urge to void.
3. Void after intercourse to rid the urethra of bacteria acquired during sex.
4. If there is a history of atypical anatomy or recurrent UTI, talk to the healthcare provider about prophylaxis with antibiotics. Agents of choice include cephalexin, TMP/SMX, and nitrofurantoin.

Table 1: Review of studies concerning the effects of hormones and pregnancy on UTI

Author and Publication Year	Type of Study	Sample size	Outcome
Annaldasula 2018 [94]	Case-control study	242	UTI during pregnancy may lead to serious complications including adverse outcomes for both mother and child including pre-term birth and small-for-gestational-age babies. Pregnancy women are more prone to the risk of urinary tract infection. There is a greater need to suspect UTI during pregnancy especially after 20 weeks of the gestational period.
Zacche et al., 2017 [97]	Prospective Cohort Study	840	This study demonstrated a correlation between risk factors and urinary tract infection. It is associated with a significant impairment of health-related quality of life and, subsequently, with a substantial economic burden.
Abdel-Aziz et al., 2017 [52]	Cross-sectional study	171	They assessed the risk factors that predispose expectant mothers to develop ASB including age, gestational stage, parity, sexual activities
Suskind et al., 2016 [53]	Cohort study	48,283	Concluded that Urinary tract infections (UTIs) are the most common type of bacterial infection, accounting for enormous morbidity and mortality on both an individual and societal level.
Giarenis et al., 2016 [99]	Cross-sectional study	1006	Lower urinary tract symptoms (LUTS) are common in women with a reported prevalence up to 66.6% in studies
Ironmonger et al., 2016 [95]	Cross-sectional	6000	Eighty-six percent of respondents reported that they use antibiotic prescribing formularies to treat UTI. The majority of these respondents 73% stated that they used a formulary provided by their PCT; with 45 (12 %) reporting using more than one formulary.
Miron et al., 2015 [101]	Prospective study	250	They assessed the concordance of late pregnancy and post-UTI renal ultrasonogram (RUS) in children with first proven simple UTI. Urinary tract infection (UTI) is relatively prevalent in children, affecting 3–5% of females and 1.5% of males.
Robinson et al., 2013 [4]	RCT	2129	The female genital and lower urinary tracts are sensitive to the effects of the female sex steroid hormones throughout life. Estrogen deficiency occurring following the menopause is known to cause atrophic change and may be associated with lower urinary tract symptoms such as frequency, urgency, nocturia, urgency incontinence and recurrent infection.
Ekinci et al., 2012 [100]	Retrospective study	57	Pregnant women with UTI and ureteral calculi should initially be treated conservatively, with analgesia, hydration, and antibiotics, if necessary.
Rosenberg et al., 2011[98]	Case-Control study	195	Studied the physiological changes in the pregnancy like increased renal plasma flow, and glomerular filtration rate, causing a state of hypercalciuria and hyperuricosuria
Hillier et al., 2006 [102]	RCT	208	They concluded that, for the purpose of surveillance of antibiotic-resistant bacteria, it would be ideal if GPs could request urine specimens for all patients presenting with suspected UTI.
Kesim et al., 2004 [96]	RCT	511	Urinary tract infection is a frequent complication of pregnancy, and it may be symptomatic or asymptomatic. In the research, they had observed one baby with bilateral hydronephrosis, one atrial septal defect, and one congenital hypothyroidism resulting from mothers prescribed antibiotics and urinary antiseptics because of UTI.
Alran et al., 2004 [93]	Case-control study	78	Maternal outcomes were assessed including preeclampsia, blood transfusion, endometritis, and urinary tract infection.

CONCLUSION

Hormones, especially estrogen and progesterone are known to have an important role in the function of the lower urinary tract throughout adult life. Estrogen helps in endometrial cell proliferation. Depletion of this level may cause variations in vaginal flora and collagen content (decreases) which may increase the adhesion rate or colonization of bacteria in the urinary tract increasing the rate of infections. Whereas, progesterone antagonizes the action of estrogen hormone and relaxes the uterine smooth muscles. This hormone is usually increased during pregnancy and causes many anatomical and functional changes. Progesterone is also known to cause a decrease in muscle tone of ureters, which causes dilation of the passage, finally leading to decreased urinary flow and voiding frequency. This results in stagnant of urine in the bladder and ureters causing the bacteria to colonize into it. Hence, the increase in progesterone leads to urinary incontinence and infections. In pregnancy, physiological changes in the urinary tract are usually observed in the seventh week of gestation. Major changes are dilation of renal pelvis and ureters. This also causes mechanical compression of the urinary bladder and ureters causing urine stagnant leading to urinary tract infections. These infections resolve through selected antibiotic regimens and a few dietary changes like consumption of cranberry juice, decreased intake of diet cokes, decreased fatty food savoring etc. And this infection recurrence can be prevented by certain lifestyle modifications and preventive steps.

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How to cite this article: Konapala LA, Vesalapu V, Kolakota RK et.al. Pregnancy and hormonal effects on urinary tract infections in women: A scoping review. International Journal of Research and Review. 2018; 5(10):407-420.

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