REVIEW ARTICLE

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Potential role of the urinary microbiome in female urinary incontinence – a scoping review

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Abstract

Introduction and Objective. Urinary incontinence (UI) is defined as any involuntary leakage of urine and may affect up to half of women, depending on the population and methodology used. Consequently, there is an ongoing need for objective markers that could improve phenotyping of women with lower urinary tract symptoms (LUTS) and support more targeted management. A major conceptual shift occurred after the demonstration that urine is not sterile. Using culture-independent methods (16S rRNA sequencing) and enhanced quantitative urine culture (EQUC), resident microbial communities have been identified in the female bladder.

Review Methods. The aim of the scoping review is to map and summarize evidence from 2012–2024 on the relationship between the urinary microbiome and female UI, with particular emphasis on differences reported between UI subtypes (SUI, UUI/OAB, and MUI), and to identify key gaps in knowledge, and methodological challenges relevant to future diagnostic and therapeutic research.

Brief description of the state of knowledge. Pearce et al. compared catheterized urine from women with and without UUI symptoms using both 16S rRNA sequencing and EQUC. They reported lower overall abundance of Lactobacillus in the UUI group, with enrichment of genera including Gardnerella, and differences in Lactobacillus species distribution (e.g., L. gasseri more frequent in UUI vs. L. crispatus more frequent in controls).

Summary. Evidence accumulated over the last 12 years confirms that urine in healthy women is not sterile and that urgency-predominant phenotypes (OAB/UUI) most consistently differ from asymptomatic controls in the structure of the urinary microbiome community. Reported patterns most often include reduced Lactobacillus dominance and/or increased prevalence of genera such as Gardnerella, Aerococcus, Proteus, or Enterobacteriaceae.

Key words

Lactobacillus, microbiota, metagenomics, urinary bladder, urinary incontinence, overactive urinary bladder, 16S Ribosomal RNA

INTRODUCTION AND OBJECTIVE

Urinary incontinence (UI) is defined as any involuntary leakage of urine, and represents one of the most common manifestations of female lower urinary tract symptoms (LUTS). Clinically, UI is usually classified into stress urinary incontinence (SUI), urgency urinary incontinence (UUI), and mixed urinary incontinence (MUI), although symptoms frequently overlap and may coexist with urgency, frequency, nocturia, dysuria, or recurrent urinary tract infection-like complaints [1, 2]. Contemporary guidelines emphasize that women with LUTS require phenotype-oriented assessment because similar symptoms may arise from different mechanisms, including urethral sphincter dysfunction, detrusor over-activity, altered bladder sensation, pelvic floor dysfunction, infection, inflammation, or mixed pathophysiology [2, 3].

The terminology of LUTS and UI has evolved over time, but the distinction between storage, voiding, and post-micturition symptoms remains central to clinical interpretation [4]. Symptom-based differential diagnosis is particularly challenging in urgency-predominant LUTS, where urgency, frequency, nocturia, and bladder discomfort may resemble urinary tract infection (UTI), despite negative standard urine culture. As a result, there is growing interest in objective markers that could support more precise phenotyping of women with LUTS and improve diagnostic and therapeutic stratification.

A major conceptual shift occurred after the demonstration that urine is not sterile. Using culture-independent methods, including 16S rRNA gene sequencing, and enhanced quantitative urine culture (EQUC), resident bacterial communities were identified in the adult female bladder [5, 6]. These findings challenged the traditional view of the urinary tract as a sterile environment and laid the foundation for research into the urinary microbiome as a potential contributor to LUTS, UI, recurrent UTIs, and other urogynaecological disorders.

Early studies comparing women with and without urgency urinary incontinence showed that urinary microbial communities may differ according to symptom status, with reduced Lactobacillus dominance and enrichment of genera, such as Gardnerella, Aerococcus, Staphylococcus, and Streptococcus, reported in some UUI cohorts [7].

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Subsequent reviews have summarized the emerging evidence linking urinary microbiome characteristics with LUTS, OAB, UUI, recurrent UTI, treatment response, and postoperative complications in urogynaecological populations [8–14].

Current evidence suggests that alterations in the urinary microbiome are most consistently reported in urgency-predominant phenotypes, including OAB and UUI. These associations include differences in microbial diversity, predominance of *Lactobacillus*, the presence of potentially uropathogenic or pro-inflammatory taxa, and, in some studies, associations with symptom severity or treatment response [13, 14]. In contrast, SUI is primarily associated with urethral sphincter deficiency, pelvic floor dysfunction, connective tissue changes, and neuromuscular factors; therefore, evidence supporting a direct urinary microbiome-driven mechanism in uncomplicated SUI remains limited. MUI represents a more complex phenotype because stress- and urgency-related mechanisms coexist in variable proportions, and microbiome-related findings may be influenced by the urgency component, menopausal status, hormonal exposure, age, previous UTI history, and the overlap between urinary and vaginal microbial communities.

OBJECTIVE

The aim of this scoping review is to map and summarize recent evidence on the relationship between the urinary microbiome and female urinary incontinence, with particular emphasis on studies published since 2018. The review focused on differences reported across UI subtypes, including SUI, UUI/OAB, and MUI, and aims to identify methodological limitations relevant to future diagnostic, biomarker, and therapeutic research. Earlier landmark studies were included only when essential for understanding the development of urinary microbiome research.

REVIEW METHODS

Design and reporting framework. A scoping review was conducted to map available evidence on urinary microbiome characteristics in adult women with UI and related LUTS. The review was reported in accordance with the PRISMA extension for Scoping Reviews (PRISMA-ScR), with additional attention to transparent reporting of search strategy, eligibility criteria, study selection, and narrative synthesis [15]. PRISMA 2020 principles were also considered where relevant to the presentation of the study selection process and flow diagram [16]. Given the heterogeneity of phenotypes, sampling techniques, laboratory methods, bioinformatic workflows, and reported outcomes, a scoping rather than meta-analytic approach was considered appropriate.

Information sources and search strategy. A structured literature search was performed for studies published primarily between January 2018 – December 2024, with additional verification of relevant records available up to January 2025. Earlier studies were included only when they represented seminal conceptual, methodological, or phenotype-defining contributions, such as the initial demonstration of the female urinary microbiome, the introduction of EQUC, or foundational studies directly linking urinary microbiome profiles with urgency urinary incontinence. This strategy was adopted to ensure that at least 75% of the reference list reflected recent evidence while preserving essential historical context.

The following databases and registries were searched: MEDLINE/PubMed, EMBASE, Scopus, Web of Science, Cochrane Library, Google Scholar, and ClinicalTrials.gov. The search was completed in January 2025. Search terms were combined using Boolean operators and included:

- 'urinary incontinence' AND 'microbiome';
- 'urinary microbiome' AND 'female lower urinary tract symptoms';
- 'urinary microbiota' AND 'overactive bladder';
- 'stress urinary incontinence' OR 'SUI' AND 'microbiome';
- 'urgency urinary incontinence' OR 'UUI' AND 'microbiome';
- 'mixed urinary incontinence' OR 'MUI' AND 'microbiome';
- 'overactive bladder' OR 'OAB' AND 'urinary microbiome';
- 'urinary tract infection' OR 'UTI' AND 'urinary microbiome';
- 'catheterized urine' AND 'microbiome';
- 'enhanced quantitative urine culture' OR 'EQUC'.

To improve reproducibility, an example of the complete PubMed search strategy is provided below: ('urinary microbiome' OR 'urinary microbiota' OR 'urobiome' OR 'enhanced quantitative urine culture' OR 'EQUC') AND ('urinary incontinence' OR 'stress urinary incontinence' OR 'SUI' OR 'urgency urinary incontinence' OR 'UUI' OR 'mixed urinary incontinence' OR 'MUI' OR 'overactive bladder' OR 'OAB' OR 'female lower urinary tract symptoms' OR 'LUTS') AND ('female' OR 'women') AND ('2018/01/01' [Date – Publication]: '2025/01/31' [Date – Publication]).

Additional earlier studies were identified through reference checking when they were considered seminal for the conceptual or methodological development of urinary microbiome research.

Eligibility criteria. Studies were eligible if they met the following criteria:

- population: women aged ≥18 years;
- phenotype: UI, including SUI, UUI, MUI, OAB, or related LUTS in which urinary microbiome characteristics were assessed;
- study type: randomized trials, cohort studies, case-control studies, cross-sectional studies, longitudinal observational studies, pilot studies, and relevant systematic or narrative reviews used for contextual interpretation;
- language: English;
- methods: urinary microbiome assessed using 16S rRNA gene sequencing, EQUC, expanded culture-based methods, metagenomic sequencing, or related microbial profiling methods;
- urine collection: catheterized urine, midstream voided urine, or clearly defined urogenital sampling when relevant to the interpretation of urinary/vaginal microbial overlap.

Exclusion criteria included: non-human studies, paediatric populations, non-English publications, studies not focused on female participants, papers lacking primary or contextual relevance to the urinary microbiome and UI/LUTS phenotypes, and publications addressing only general UTI management without microbiome-related analyses.

Earlier studies published before 2018 were retained only if they met at least one of the following criteria: they first
- demonstrated the presence of bacterial communities in the adult female bladder;
- introduced or validated enhanced culture-based approaches such as EQUC;
- established foundational associations between urinary microbiome profiles and UUI/OAB phenotypes;
- repeatedly cited in subsequent reviews and primary studies as methodological or conceptual landmarks.

These studies were not intended to broaden the historical scope of the review, but to provide essential context for interpreting more recent evidence.

Study selection. After duplicate removal, titles and abstracts were screened, followed by full-text eligibility assessment. Screening was performed independently by two reviewers, and disagreements were resolved through discussion. Selection decisions were documented in a structured screening workflow. Studies were grouped by dominant clinical phenotype: OAB/UUI, SUI, MUI, perioperative urogynecological cohorts, and methodological/contextual studies.

Data charting and synthesis. For included studies, data were charted on: study design, sample size, patient phenotype, urine sampling method, microbiome assessment method, dominant taxa, alpha and beta diversity findings, symptom associations, treatment-response associations, postoperative outcomes, and key methodological limitations. Given substantial heterogeneity in sampling techniques, DNA extraction protocols, sequencing targets, culture methods, contamination control, bioinformatic pipelines, and reported clinical outcomes, results were synthesized narratively and organized by UI subtype. Comparisons with asymptomatic controls were summarized when available. Data charting was performed using a predefined extraction form developed for this review. Two reviewers independently extracted key information from the included studies, including study design, population characteristics, clinical phenotype, urine collection method, microbiome assessment technique, main microbial findings, symptom-related associations, treatment-response data, and methodological limitations. Any discrepancies in extracted data were discussed and resolved by consensus. Because the purpose of this scoping review was to map the available evidence rather than to estimate pooled effects, no meta-analysis was performed.

Methodological considerations. The urine sampling method was treated as a major source of heterogeneity. Catheterized urine was considered more reflective of bladder microbiota, whereas midstream voided urine may include periurethral, vulvar, or vaginal microbial contributions. This distinction is particularly important in low-biomass microbiome research, where contamination introduced during sample collection, DNA extraction, amplification, sequencing, or reagent processing can substantially influence results [42–44]. Differences in sequencing regions, taxonomic assignments, rare-taxa filtering, and contamination control strategies were therefore considered when assessing cross-study comparability.

DESCRIPTION OF THE STATE OF KNOWLEDGE

Study selection and general characteristics. Records identified n = 1,945; records screened n = 750; reports sought n = 501; reports assessed n = 155; studies included n = 20. Additional reviews, guidelines, and methodological publications were used to contextualize the findings and support interpretation. The included primary evidence was heterogeneous with respect to phenotype definitions, urine collection method, microbial profiling technique, and reporting standards. Most studies focused on urgency-predominant LUTS, especially OAB and UUI, whereas evidence for SUI and MUI was comparatively limited.

The strongest and most consistent evidence relate to OAB/UUI, where urinary microbial community structure has been associated with symptom presence, symptom severity, treatment response, inflammatory pathways, and possible cross-ecosystem interactions between the bladder and vagina. In contrast, SUI studies do not support a robust SUI-specific urinary microbiome signature. MUI studies suggest that urinary microbiome findings may be more closely related to urgency-type leakage than to stress-type leakage.

Overactive bladder (OAB) and the urinary microbiome. Four earlier studies investigating OAB were included in the primary synthesis, with additional recent studies and reviews used for contextual interpretation. Curtiss et al. compared midstream urine samples from women with OAB and asymptomatic controls and reported differences in urinary microbial community structure, with Lactobacillus more frequently detected in controls, and Proteus more frequently observed in OAB samples [17]. Using catheterized urine, Wu et al. also found significant differences between women with OAB and controls, reporting relatively higher abundance of Proteus and Aerococcus in OAB and reduced Lactobacillus and Prevotella compared with controls [18].

Gill et al. evaluated OAB symptoms in relation to pyuria, bacterial growth, and urinary microbial ecology. Their findings suggested that bacterial growth and inflammatory features may contribute to OAB symptom expression in some women, supporting the hypothesis that microbial and host inflammatory factors may interact in urgency-predominant LUTS [19]. Javan Balegh Marand et al. compared the urinary microbiota of women with OAB, with and without detrusor overactivity, and of controls. They reported significant differences in microbiome composition across groups and suggested that distinct urinary microbial profiles may be linked to specific OAB phenotypes, including urodynamically characterized detrusor overactivity [20].

More recent studies have expanded this area by investigating culture-enhanced approaches, modified culture protocols, and viral components of the urogenital microbiome. Kaya et al. investigated bladder microbiota in female patients with OAB using catheterized urine and modified culture-based methods, reporting reduced detection of Lactobacillus gasseri and more frequent detection of Staphylococcus epidermidis in the OAB group compared with controls [21]. Sun et al. reported that detection of urinary viruses was associated with more severe symptoms and altered bacteriome features in women with OAB, suggesting that future studies may need to consider microbial communities beyond bacteria [22].

Overall, OAB studies support the concept that women with OAB may differ from controls in urinary microbial

community structure. However, findings are not uniform across studies. Differences in urine sampling, OAB definition, detrusor over-activity assessment, sequencing method, culture method, contamination control, and statistical pipeline likely contribute to inconsistent results.

Urgency urinary incontinence: composition, severity, and treatment response. Urge urinary incontinence (UUI) is the UI subtype most consistently linked with urinary microbiome alterations. Pearce et al. compared catheterized urine from women with and without UUI symptoms using both 16S rRNA gene sequencing and EQUC. They reported lower overall Lactobacillus abundance in the UUI group, enrichment of genera including Gardnerella, Aerococcus, Staphylococcus, and Streptococcus, and differences in Lactobacillus species distribution, with *Lactobacillus gasseri* more frequent in the UUI group and *Lactobacillus crispatus* more frequent in the controls [7].

In a subsequent study, Pearce et al. described clinical associations of urinary microbial patterns in women undergoing treatment for UUI. Sequence-positive status and microbial community patterns were associated with symptom burden, UUI episode frequency, treatment response, and post-treatment UTI risk [23]. Karstens et al. detected polymicrobial communities in both UUI and control groups and reported that lower urinary microbiome diversity was associated with greater symptom severity, suggesting that community structure rather than the presence of a single organism may be clinically relevant [24].

Thomas-White et al. examined the relationship between baseline urinary microbiota and response to solifenacin. Respondents tended to have fewer isolates and/or less complex urinary communities at baseline, supporting the hypothesis that the urinary microbiome may influence pharmacological response in UUI [25]. Chen et al. assessed women with refractory UUI and recurrent UTI using midstream urine and reported heterogeneous urinary microbiota profiles, proposing that persistent bladder colonization or dysbiosis may contribute to refractory symptoms [26]. However, interpretation of midstream samples requires caution because they may include periurethral or vaginal microbial contributions.

Mechanistic data remain limited but biologically plausible. Abbasian et al. provided experimental evidence suggesting that selected uropathogens, including Escherichia coli and *Gardnerella vaginalis*, may induce calcium influx and contractility through extracellular ATP release, whereas *Lactobacillus* species did not trigger this pathway [27]. These findings support the hypothesis that selected microbial taxa may influence bladder sensory or contractile pathways relevant to urgency symptoms.

Nardos et al. investigated bladder–vaginal microbial relationships and reported greater overlap and altered network dynamics in women with UUI compared with controls [28]. This finding is important because it suggests that UUI may not reflect bladder microbial ecology alone, but rather interactions across the urogenital microbial environment.

Treatment-associated changes in the microbiome have also been explored. Halverson et al. reported that pre-treatment urinary microbiome features were associated with response to solifenacin, but not mirabegron, and that post-treatment changes in microbial richness were observed [29]. Mueller et al. evaluated women with UUI undergoing sacral neuromodulation and reported longitudinal urinary microbiome characteristics during treatment, contributing to the emerging evidence on microbiome dynamics during advanced therapies [30]. Gabriel et al. assessed urinary microbiota, local inflammatory markers, and response to anti-cholinergic therapy in women with UUI; their pilot study suggested that factors beyond urinary microbiome composition and local immune response may contribute to treatment outcomes [31].

Collectively, urgency-predominant conditions show the strongest association with differences in the urinary microbiome. Reported patterns most often include reduced *Lactobacillus* dominance, increased *Gardnerella* or other potentially uropathogenic taxa, altered richness or diversity, and possible links with clinical severity or treatment response. However, the evidence remains associative, and causality has not been established.

Stress urinary incontinence (SUI) – limited evidence for a direct urobiome signal. Evidence linking uncomplicated SUI directly with a specific urinary microbiome pattern remains limited. Thomas-White et al. evaluated the urinary microbiota of women with uncomplicated SUI and found no clear association between microbiome diversity and SUI symptoms. Instead, microbial diversity was associated with such factors as BMI, hormonal status, and concomitant urgency symptoms [32]. This is consistent with the established mechanical and neuromuscular pathophysiology of SUI, in which urethral sphincter deficiency, pelvic support defects, connective tissue remodelling, parity, aging, and pelvic floor dysfunction, are considered major drivers.

Nevertheless, urinary microbiome profiling may have relevance in surgical or perioperative contexts. Fok et al. reported that urinary symptoms were associated with selected urinary microbes in urogynaecological surgical patients, suggesting that microbial features may be related to urgency or irritative symptoms in mixed surgical populations [33]. Thomas-White et al. further reported that urinary microbes were associated with postoperative UTI risk after urogynaecological surgery, particularly in contexts characterized by reduced *Lactobacillus* dominance and enrichment of uropathogenic taxa [34].

A postpartum study by Zhang et al. reported vaginal, not urinary, dysbiosis patterns in women with SUI symptoms, including higher diversity and reduced *Lactobacillus* dominance in the SUI group [35]. This study should therefore be interpreted as evidence on the vaginal ecosystem rather than the bladder niche. It may, however, be relevant to the broader concept of urogenital microbial ecology and pelvic floor disorders.

Overall, current data do not support a robust, reproducible, SUI-specific urinary microbiome signature. At present, urinary microbiome profiling in SUI appears more relevant to postoperative UTI risk stratification, concomitant urgency symptoms, and perioperative urogynaecological outcomes than to the primary pathogenesis of uncomplicated SUI.

Mixed urinary incontinence (MUI) – community-level differences and menopausal status. MUI is a heterogeneous phenotype in which stress- and urgency-related mechanisms coexist in variable proportions. Komesu et al. compared catheterized urine from women with MUI and controls

and reported no significant difference in *Lactobacillus* predominance in age-matched groups. However, differences in specific urinary microbial communities were observed when comparing women with MUI to younger controls, whereas findings were less distinct in older women, suggesting a possible modifying effect of menopausal status and age-related factors [36].

In a subsequent analysis, Komesu et al. reported associations between vaginal and urinary microbiomes in women with MUI and controls. They identified overlapping genera, including *Lactobacillus, Gardnerella, Prevotella*, and *Ureaplasma*, as well as taxa more specific to the urinary niche [37]. These findings highlight the importance of distinguishing urinary microbial communities from vaginal microbial communities, while also recognizing that the two ecosystems may be biologically connected.

Carnes et al. investigated urinary and vaginal microbiome community types in women with MUI and reported that lower *Lactobacillus* dominance and higher alpha diversity in urinary communities were associated with more frequent incontinence episodes, particularly urgency-type leakage. Vaginal community composition was not clearly linked to symptom severity [38]. These findings suggest that the urinary microbiome may be more closely related to the urgency component of MUI than to the stress component.

Taken together, MUI findings suggest that urinary community structure, especially *Lactobacillus* dominance and overall diversity, may be associated with symptom burden in some cohorts. However, results remain inconsistent and may be confounded by menopausal status, estrogen exposure, age, BMI, previous UTI, antibiotic exposure, comorbidities, and differences in sampling and analytical methods.

Menopause, estrogen status, and urinary microbial ecology. Menopausal status and estrogen exposure are important modifiers of the urogenital microbiome. Reduced estrogen levels may alter the vaginal and urinary microbial environment, decrease *Lactobacillus* dominance, and increase susceptibility to recurrent UTI or dysbiosis-associated symptoms. Thomas-White et al. reported that vaginal estrogen therapy was associated with increased Lactobacillus in the urine of postmenopausal women with OAB symptoms [39]. This finding suggests that hormonal status may influence urinary microbial ecology and should be considered in future studies of OAB, UUI, and MUI.

Recent reviews further emphasize that age, menopausal transition, prior UTI history, antibiotic exposure, and estrogen status may shape the urinary microbiome and modify susceptibility to UTI and LUTS [40]. Therefore, future urinary microbiome studies in women with UI should stratify participants by menopausal status, systemic or local estrogen use, history of recurrent UTI, and recent antimicrobial exposure.

Methodological heterogeneity and interpretability. Across phenotypes, major limitations include variability in urine collection, laboratory workflows, sequencing targets, bioinformatic processing, contamination control, and outcome definitions. Catheterized urine is generally considered more reflective of bladder microbiota, whereas midstream voided urine may include periurethral, vulvar, and vaginal taxa. This issue is especially important in women because the urethra, vulva, and vagina are anatomically close and may share microbial communities.

Microbiome research in urine is also technically challenging because urine is a low-biomass sample. In such samples, contamination from reagents, collection devices, extraction kits, laboratory environments, or sequencing workflows, can distort microbial profiles. Recent methodological papers emphasize the need for negative controls, standardized DNA extraction, rigorous contamination filtering, and transparent reporting of bioinformatic pipelines [41–44].

Another limitation is that many studies are cross-sectional. Therefore, it remains unclear whether urinary microbiome alterations precede symptom development, result from altered bladder physiology, reflect prior antibiotic exposure or a history of UTI, or act as modifiers of symptom severity. Longitudinal studies are required to clarify temporal relationships and to evaluate whether microbiome patterns change during behavioural, pharmacological, hormonal, neuromodulatory, or surgical therapies.

SUMMARY

Evidence accumulated over the last decade confirms that urine in healthy women is not sterile, and that the female lower urinary tract contains microbial communities detectable by enhanced culture and sequencing-based methods. Urgency-predominant phenotypes, including OAB and UUI, show the most consistent associations with differences in the urinary microbiome compared with asymptomatic controls. Reported patterns most often include reduced *Lactobacillus* dominance, increased detection of *Gardnerella, Aerococcus, Proteus, Staphylococcus, Streptococcus, Enterobacteriaceae*, or other potentially uropathogenic taxa, and associations with symptom severity or treatment response in selected studies.

In contrast, evidence for a distinct SUI-related urinary microbiome signature remains limited. SUI is primarily driven by mechanical, connective-tissue, urethral-sphincter, and pelvic-floor mechanisms. Microbiome profiling in SUI may be more relevant to postoperative UTI risk, concomitant urgency symptoms, or broader urogenital dysbiosis than to the primary pathogenesis of uncomplicated SUI.

MUI findings are heterogeneous. Available evidence suggests that urinary microbiome features may be associated especially with urgency-type leakage and symptom burden, but these relationships may be modified by menopausal status, hormonal exposure, age, prior UTI, antibiotic use, and the overlap between urinary and vaginal microbial communities.

Limitations of the study. This scoping review has several limitations. Although the review focused primarily on recent literature, selected older studies were retained because they represent seminal conceptual, methodological, or phenotype-defining contributions. Only English-language publications were included. Formal risk-of-bias assessment was not performed, which is consistent with the exploratory purpose of a scoping review, but limits conclusions regarding the strength of evidence. Substantial methodological heterogeneity limits comparability and prevents meta-analytic synthesis.

Table 1. Main findings in OAB studies

Study	Participants	Design	Urine sampling/method	Key findings
Curtiss et al., 2017 [17]	95; 60 OAB, 35 controls	Case-control	Midstream urine; 16S rRNA sequencing	OAB and controls differed in community structure; *Lactobacillus* was more frequent in controls, whereas Proteus was more frequent in OAB.
Wu et al., 2017 [18]	55; 30 OAB, 25 controls	Case-control	Catheterized urine; 16S rRNA sequencing	OAB was associated with relatively higher *Proteus* and *Aerococcus*; *Lactobacillus* and *Prevotella* were relatively reduced.
Gill et al., 2018 [19]	46; 24 OAB, 22 controls	Observational/ case-control	Midstream urine, culture, and microscopy	OAB symptoms were associated with bacterial growth and pyuria-related findings, suggesting microbial-inflammatory contribution in some patients.
Javan Balegh Marand et al., 2023 [20]	33; OAB with DO, OAB without DO, controls	Observational	Urine microbiome profiling; 16S rRNA sequencing	OAB subphenotypes differed in urinary microbiome composition; detrusor overactivity was associated with a distinct microbial profile.
Kaya et al., 2025 [21]	40; 20 OAB, 20 controls	Case-control	Catheterized urine; modified expanded culture method	*Lactobacillus gasseri* was detected less frequently and Staphylococcus epidermidis more frequently in OAB patients than in controls.
Sun et al., 2022 [22]	Women with OAB and controls	Observational/ metagenomic	Urinary virome and bacteriome analysis	Detection of urinary viruses was associated with aggravated symptoms and altered bacteriome features in women with OAB.

OAB — overactive bladder; DO — detrusor overactivity; 16S rRNA — 16S ribosomal RNA gene sequencing.

Table 2. Main findings in UUI studies

Study	Participants	Design	Sampling/method	Main findings
Pearce et al., 2014 [7]	118; 60 UUI, 58 controls	Cohort/case-control	Catheterized urine; 16S rRNA + EQUC	UUI was associated with lower overall *Lactobacillus* abundance and enrichment of *Gardnerella, Aerococcus, Staphylococcus*, and *Streptococcus; Lactobacillus gasseri* was more frequent in UUI, whereas *Lactobacillus crispatus* was more frequent in controls.
Pearce et al., 2015 [23]	182	Randomized trial cohort/ context of UUI treatment	Catheterized urine; 16S rRNA	Sequence-positive status and urinary community patterns were associated with symptom burden, treatment response, and post-treatment UTI risk.
Karstens et al., 2016 [24]	20; 10 UUI, 10 controls	Case-control	Catheterized urine; 16S rRNA	Polymicrobial communities were detected in both groups; lower diversity was associated with greater symptom severity.
Thomas-White et al., 2016 [25]	134; 74 UUI, 60 controls	Cohort	Catheterized urine; EQUC/16S rRNA	Baseline microbiome was associated with a response to solifenacin; responders tended to have fewer isolates or less complex communities.
Chen et al., 2018 [26]	39; refractory UUI + recurrent UTI	Observational	Midstream urine; 16S rRNA + strain typing	Heterogeneous microbiota were reported; persistent colonization was proposed as a contributor to refractory symptoms.
Abbasian et al., 2019 [27]	Experimental	Mechanistic/ cell model	Cell models	Uropathogens induced calcium influx and contractile responses via extracellular ATP release; Lactobacillus species did not trigger this pathway.
Nardos et al., 2022 [28]	Women with and without UUI	Network analysis	Bladder and vaginal communities	Greater bladder–vagina overlap and altered microbial network dynamics were reported in UUI.
Halverson et al., 2023 [29]	97 UUI	Observational	Catheterized urine; pre/ post treatment	Pretreatment microbiome was associated with solifenacin response but not mirabegron; post-treatment richness changes were observed.
Mueller et al., 2023 [30]	19 UUI	Longitudinal observational	Catheterized urine; sacral neuromodulation	Longitudinal patterns of the urinary microbiome were described during sacral neuromodulation.
Gabriel et al., 2023 [31]	Women with UUI treated with anticholinergics	Prospective pilot study	Catheterized urine; culture, 16S rRNA, cytokines	Microbiota and local inflammatory markers were evaluated before and after treatment; response appeared to involve factors beyond microbiome composition alone.

UUI — urgency urinary incontinence; UTI — urinary tract infection; EQUC — enhanced quantitative urine culture.

Table 3. Main findings in SUI and perioperative urogynecological studies

Study	Participants/ context	Design	Sampling/method	Main findings
Thomas-White et al., 2017 [32]	Women with uncomplicated SUI	Observational	Urinary microbiota assessment	No robust SUI-specific urinary microbiome signature was found; microbial diversity was associated with BMI, hormonal status, and urgency symptoms.
Fok et al., 2018 [33]	Urogynecological surgical patients	Observational	Urinary microbiome assessment	Urinary symptoms were associated with selected urinary microbes in surgical cohorts.
Thomas-White et al., 2018 [34]	Urogynecological surgical patients	Observational	Urinary microbiome assessment	Urinary microbes were associated with the risk of postoperative UTI.
Zhang et al., 2024 [35]	Postpartum women with SUI symptoms	Observational	Vaginal microbiome assessment	Vaginal, not urinary, dysbiosis was reported in women with SUI; findings should not be interpreted as direct bladder microbiome evidence.

SUI — stress urinary incontinence; UTI — urinary tract infection

Table 4. Main findings in MUI studies

Study	Participants	Design	Sampling/method	Main findings
Komesu et al., 2018 [36]	Women with MUI and controls	Case-control	Catheterized urine; urinary microbiome profiling	No significant difference in *Lactobacillus* predominance in age-matched groups; age and menopausal status appeared to modify findings.
Komesu et al., 2020 [37]	Women with MUI and controls	Observational	Vaginal and urinary microbiome profiling	Overlap between vaginal and urinary microbiomes was reported, but some taxa appeared more specific to the urinary niche.
Carnes et al., 2024 [38]	Women with MUI	Observational	Urinary and vaginal microbiome community typing	Lower *Lactobacillus* dominance and higher urinary alpha diversity were associated with more frequent incontinence episodes, especially urgency-type leakage.

MUI — mixed urinary incontinence.

SUMMARY

Although urinary microbiome profiling is not currently suitable for routine clinical use in women with UI, several potential future applications warrant consideration.

1) Purinary microbiome patterns could help distinguish urgency-predominant phenotypes from primarily mechanical SUI in diagnostically ambiguous cases.
2) Microbiome features may contribute to risk stratification for recurrent UTI, postoperative UTI after urogynecological surgery, or persistent symptoms despite standard therapy.
3) Baseline urinary microbial profiles may eventually support individualized treatment selection, particularly in UUI/OAB, where associations with anticholinergic and beta-3 agonist responses, as well as neuromodulation outcomes, have been explored.
4) Microbiome-informed interventions, including vaginal estrogen therapy, probiotics, targeted antimicrobial approaches, or microbiota-modulating strategies, may become relevant in selected subgroups.

However, several barriers must be addressed before clinical implementation. These include the lack of standardized urine collection protocols, differences between catheterized and midstream samples, low risk of contamination from low biomass, heterogeneous sequencing and culture methods, absence of validated diagnostic thresholds, limited longitudinal evidence, and insufficient data on causality. Cost, turnaround time, availability of specialized laboratory workflows, and the need for clinically interpretable reports also limit translation into routine practice.

Founding source
Grant of Medical University of Lublin number: 321.

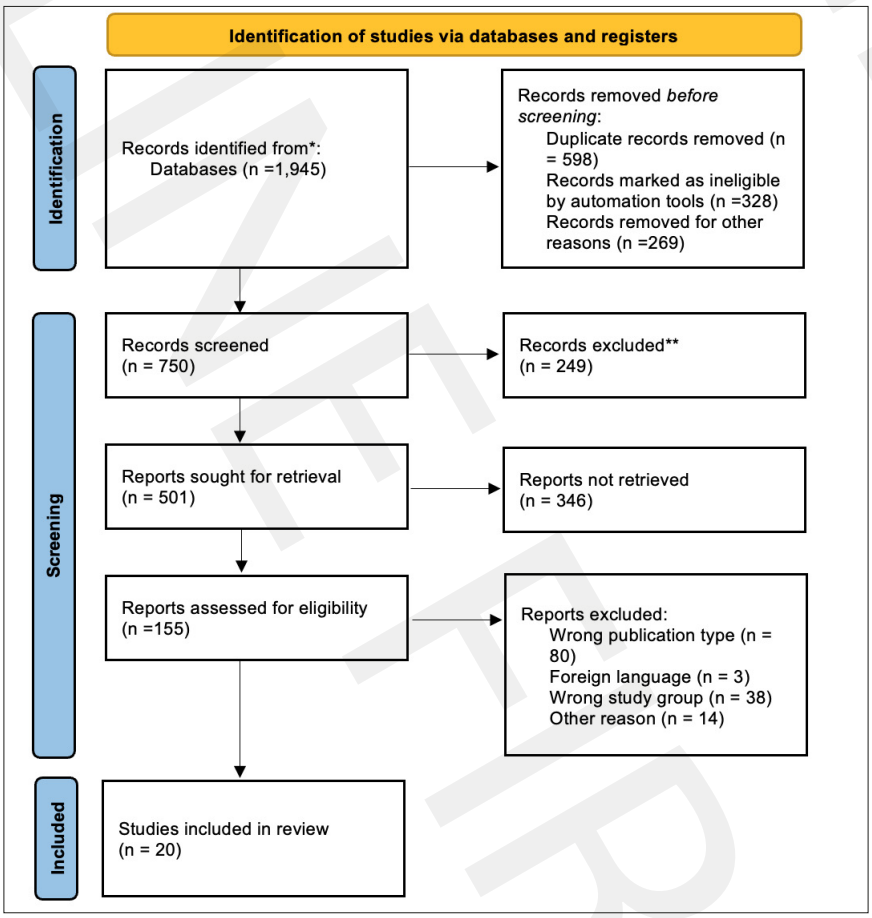

Figure 1. Study selection flow chart according to PRISMA-ScR. Screening of records, full-text eligibility assessment, and final inclusion in the scoping review

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