Current Urology Reports (2023) 24:51–58
https://doi.org/10.1007/s11934-022-01133-6

LOWER URINARY TRACT SYMPTOMS AND VOIDING DYSFUNCTION (J SANDHU, SECTION EDITOR)

# Urinary Incontinence in Elite Female Athletes

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Accepted: 7 November 2022 / Published online: 24 November 2022


## Abstract

**Purpose of Review** To summarize the current understanding on the epidemiology, pathophysiology, and management strategies of urinary incontinence (UI) in female athletes, highlighting findings specific to nulliparous elite athletes.

**Recent Findings** UI occurs in about 20–50% of female athletes of all ages and parity status, around 40% for younger nulliparous athletes, and is more prevalent in high-impact sports. Possible contributing factors to UI in female elite athletes include pelvic floor laxity and bladder neck descent, pelvic floor muscle fatigue, low energy availability, and hypermobility syndrome. In female elite athletes, urinary symptoms negatively affect quality of life, although the effects of symptoms on exercise participation are not well understood. Current management strategies are primarily conservative and centered on behavioral modifications and pelvic floor muscle physiotherapy.

**Summary** UI in female elite athletes appears to be multifactorial. Clarifying how individual factors influence UI in this population will inform athlete counseling, prevention, and treatment strategies.

**Keywords** Pelvic floor dysfunction · Urinary incontinence · Elite athletes · Pelvic floor muscles

## Introduction

Urinary incontinence (UI) has been observed in female athletes of varying ages and parity status. It is thought to result from increased intra-abdominal pressure, particularly during high-impact exercises involving running and jumping, that overcomes urethral pressure and leads to involuntary urine loss. Given a high prevalence even in nulliparous female athletes who have not had the pelvic floor weakened by childbirth, there has been increased interest in understanding the effects of high-impact exercise on the pelvic floor, particularly in athletes training and competing at the elite level who are felt to be at higher risk. While some effects of exercise on the pelvic floor have been explored, additional medical and environmental factors may also contribute to UI. This review aims to summarize our collective understanding of the epidemiology, pathophysiology, and management strategies of UI in female athletes while highlighting findings specific to nulliparous and elite athletes in particular.

## Epidemiology

While prevalence studies are variable in their criteria for age, parity, and definition of "athlete," the overall trend is towards a high prevalence of UI in athletes that increases with the degree of sport impact. A review of UI prevalence studies on women age 12–69 who performed any sport and were not currently pregnant or recently postpartum yielded a UI prevalence ranging from 5.6 to 80%, highest among gymnasts, and the risk of UI increased with the degree of impact within sports [1]. Similarly, a review of studies on female athletes age 18–60 who were not currently pregnant, yielded a broad prevalence range of 19.4 to 76%, with an overall prevalence of 36.1%, although the prevalence of UI did not change with the degree of sport impact [2]. Another meta-analysis on female athletes age 18–45, who were not parous in the past year, yielded an overall UI prevalence of 26%, although

This article is part of the Topical Collection on *Lower Urinary Tract Symptoms and Voiding Dysfunction*

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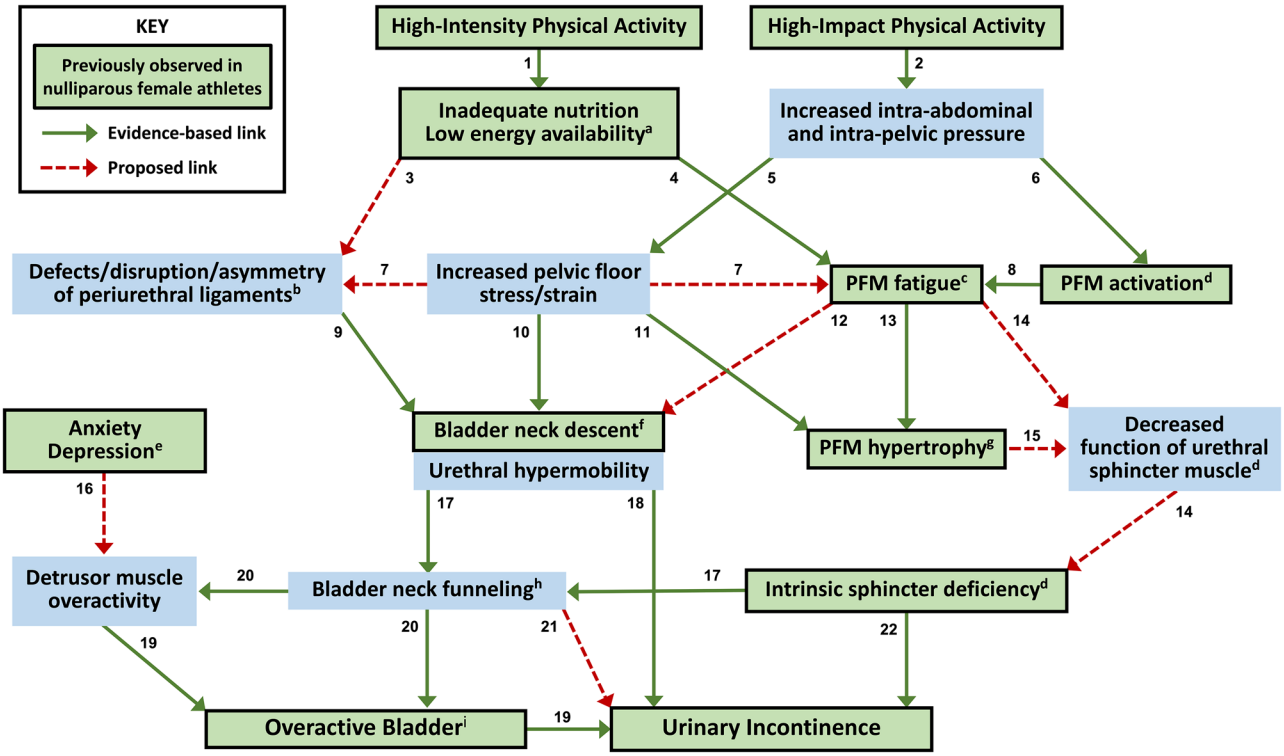


**Fig. 1** Conceptual model of proposed risk factors and mechanisms leading to bladder dysfunction in nulliparous female elite athletes (PFM, pelvic floor muscle). Proposed risk factors for bladder dysfunction (**a**–**h**). **a** Low energy availability as defined by eating disorder criteria is an independent risk factor for UI in nulliparous female athletes [29•]. **b** Defects, disruption, and asymmetry of periurethral ligaments were observed to be more prevalent in women with SUI than in continent women [46], although periurethral ligaments have not been evaluated in nulliparous female athletes specifically. **c** Nulliparous female athletes with UI have been noted to have leakage mostly at the end of training sessions [24] and less PFM endurance compared to non-athletes [16]. **d** A majority of nulliparous females with SUI during exercise had zero or negative urethral closure pressure when coughing in the supine position, despite having synergistic contraction of the urethral sphincter and pelvic floor muscles, leading to the hypothesis that the urethral sphincter muscle may be weak [13]. **e** Anxiety and depression have a higher prevalence in both current and former elite athletes (professional or Olympic, likely nulliparous) compared to the general population [32]. **f** Bladder neck descent was noted on VCUG in adolescent female athletes with SUI [22] and was more prominent on translabial ultrasound with Valsalva in nulliparous female athletes compared to non-athlete controls [15]. **g** Thicker PFMs have been noted in nulliparous female athletes compared to non-athlete controls [14, 15] and in symptomatic compared to asymptomatic athletes [19]. **h** A meta-analysis found that bladder neck funneling is 5×more prevalent in women with SUI than in asymptomatic controls [46]. **i** A systematic review in nulliparous female athletes found a mean UUI prevalence of 22.4% (range 6.1–49.5%) [4••]. Proposed mechanisms of association between risk factors and bladder dysfunction [1–3, 4••, 5–22]. **1** Athletes are subject to inadequate dietary energy intake to offset energy expenditures [28]. **2** High-impact physical activity involving repeated, abrupt increases in intra-abdominal pressure has been associated with higher rates of UI in women [47]. **3** Eating disorders have been proposed to weaken the pelvic floor through inadequate nutrition and growth factors needed to build and strengthen ligaments, fascia, and muscles [47]. Insulin-like growth factor 1, which has been proposed to affect continence by increasing pelvic floor connective tissue collagen production, is decreased in energy-deficient athletes and women with stress UI [48, 49]. **4** Sequela of relative energy deficiency in sport include decreased muscle strength, decreased training response, and decreased endurance performance [28]. **5** Simulated increases in intra-abdominal pressure during jump landings and Valsalva exert stress on the levator ani muscle [50]. **6** PFMs contract during increases in intra-abdominal pressure, including during exercise [51]. **7** It has been proposed that increased intra-abdominal pressure stretches the pelvic floor muscles and ligaments, leading to muscle fatigue and tissue damage [5]. **8** Nulliparous women who reported SUI during activity had decreased maximum voluntary contraction pressure of the PFMs after a strenuous training session [25]. **9** Simulated weakening of periurethral ligaments increases bladder neck descent and urethral hypermobility [52]. **10** Bladder neck mobility has been observed with both coughing and Valsalva in nulliparous women [53]. **11** Mechanical tension produced by stretch leads to increased muscle mass [54]. **12** PFM strengthening reduces bladder neck displacement with Valsalva [55], suggesting that weaker PFMs in the setting of fatigue may have the opposite effect (in one study, attempts to fatigue the pelvic floor muscles with repeated contractions and coughing did not increase bladder neck mobility [53]). **13** Training to volitional fatigue induces skeletal muscle protein synthesis and muscle hypertrophy [56]. Of note, this effect is not expected for fatigue related to inadequate nutrition. **14** Urethral pressure and striated urethral sphincter EMG activity decreased after repeated cough efforts in women with SUI, leading to the hypothesis that SUI is related to fatigue of the both the urethral sphincter and pelvic floor muscles [57]. It is unclear whether decreased function of the urethral sphincter results from fatigue of the striated sphincter muscle itself or decreased support from fatigued PFMs. **15** It may be hypothesized that surrounding hypertrophy of PFMs may lead to compensatory weakening of the urethral sphincter. **16** Anxiety and depression have both been observed to be risk factors and exacerbators of OAB in the general population [33]. **17** Bladder neck funneling has been associated with urethral hypermobility and ISD in middle-aged, parous women with SUI [58]. **18** Rotational descent of the urethra along with bladder neck descent has been observed in patients with SUI [59]. **19** Detrusor overactivity has a good correlation with OAB in the setting of incontinence, and OAB can lead to UI in some patients [60]. **20** Bladder neck funneling and decreased urethral pressure leads to

◂**Caption** (continued)
reflexive detrusor contractility, resulting in SUI-induced detrusor overactivity [34, 35]. **21** Pubourethral ligament laxity has been hypothesized to lead to active opening of the urethra (funneling) by surrounding muscle forces, resulting in urine loss [61]. **22** An extremely low urethral closing pressure has been observed in some patients with SUI [62] [UI, urinary incontinence; SUI, stress urinary incontinence; PFM, pelvic floor muscle; VCUG, voiding cystourethrogram; UUI, urgency urinary incontinence]

this was as high as 75% in volleyball players [3]. A meta-analysis of female CrossFit athletes age 18–71 yielded an overall prevalence of 44.5%, although most of these women were parous. In exclusively nulliparous athletes, a review on athletes age 12–44 reported a UI prevalence of 40.6% [4••], and another review on female athletes age 18–44 found that UI was more common in volleyball players and long-distance runners [5]. A review of studies on nulliparous female athletes less than 19 years of age found an average UI prevalence of 48.6% (range 18–80%) [6].

The type of UI reported in female athletes is predominantly SUI [2], although urge UI has a reported prevalence of 22.4% in nulliparous female athletes [4••]. In addition to during exercise, UI has been reported outside of exercise in female athletes with a prevalence of 34–85% [1], although this review included studies with older, parous women. In a study on nulliparous volleyball players with a mean age of 19, 37.5% of athletes had UI during activities of daily living [7]. In the same study, the majority of symptomatic athletes reported a weak stream of leakage as opposed to drops. In another study of female athletes playing a variety of sports, the majority of symptomatic athletes reported experiencing loss of a few drops at a time [8].

The natural history of UI in female athletes, especially young, nulliparous athletes, is not well defined due to the cross-sectional nature of existing prevalence studies. The risk of SUI in middle-aged adult women has been shown slightly increased with overall lifetime activity and teen strenuous activity (defined as greater than 7.5 h/week of activity), but not lifetime strenuous activity [9]. In a study of former national team elite female athletes, UI prevalence in this population was no different compared to non-athletes, and parity seemed to be a more influential factor [10]. However, among athletes, those who had UI earlier in life were also more likely to have UI later in life. These studies suggest a potential period of vulnerability earlier in life that may be affected by high-impact and/or high-intensity sports. Conversely, low-to-moderate intensity physical activity has been associated with a reduced risk of UI in large population-based studies [11, 12], suggesting that physical activity may be protective to a degree.

## Pathophysiology

The effect of strenuous or high-impact exercise on the pelvic floor remains controversial. It has been proposed that repeated exercise-induced intra-abdominal pressure increases can overload, stretch, and weaken pelvic floor structures, including muscles, fascia, and ligaments [13]. Conversely, intra-abdominal pressure increases are thought to train and strengthen PFMs by stimulating contraction of the PFMs before or during such pressure increases. Given the predominance of SUI in female elite athletes and the trend towards an increasing incidence with the degree of sport impact, it may be inferred that activities such as running and jumping lead to intra-abdominal pressure increases and downward stress to pelvic floor structures that overcome the continence mechanism. Characterization of the pelvic floor and other contributing anatomic and physiologic factors in this setting has been ongoing. A conceptual model of bladder dysfunction in nulliparous female elite athletes is shown in Fig. 1, with more detail on each of the included factors described below.

### Athletes vs. Non-Athletes

Differences in the pelvic floor musculature have been noted between athletes and non-athletes. Compared to non-athletes, athletes were observed to have larger pelvic floor muscles (PFMs) on pelvic MRI [14] and translabial ultrasound, as well as more bladder neck descent and greater levator hiatal area on Valsalva [15]. However, these differences are of uncertain clinical significance, as one study did not assess urinary symptoms [14], and the other only found a 12% prevalence of SUI in the athlete group without commenting on the relationship between symptoms and imaging findings [15]. Two additional studies have evaluated both pelvic floor strength and urinary symptoms in athletes compared to non-athletes. One study found that nulliparous female athletes with a mean age of 24 years competing at the district level or higher in Brazil had stronger pelvic floor and abdominal muscles but less PFM endurance compared to non-athletes [16]. The prevalence of UI in athletes was greater than in non-athletes (53.8% vs. 35.3%), although this difference was not statistically significant, possibly due to small sample size. Of note, 60% of the non-athletes reported practicing high-intensity physical activity, making these results difficult to interpret. Another study found that nulliparous female CrossFit athletes with a mean age of 26 years had a higher prevalence of UI compared to non-athletes (60% vs. 9.5%, $P < 0.001$), but there were no differences in PFM strength as determined by physical examination nor PFM electromyography parameters [17]. These studies raise the questions of how exercise affects PFMs, at what point exercise-induced changes to the pelvic floor become clinically relevant, and whether factors beyond PFM strength and function could be contributing to UI in athletes.

## Symptomatic vs. Asymptomatic Athletes

A few studies have compared pelvic floor anatomy and function between symptomatic (i.e., with UI) and asymptomatic female athletes (i.e., without UI). In a study comparing symptomatic and asymptomatic university physical education students, 6 of 7 symptomatic students undergoing urodynamic testing were found to have evidence of urethral sphincteric incompetence (defined as zero or negative urethral closure pressure while coughing in the supine position), although only 2 of these students demonstrated leakage on a pad test [18]. EMG showed that all subjects had synergistic contraction of the urethral sphincter and PFMs when coughing. In another study, symptomatic, nulliparous, federated football players in Portugal with a mean age of 22 years were noted to have thicker pubovisceral muscles on pelvic MRI compared to asymptomatic athletes, but there were no differences in the digital assessment of PFM strength between groups, leading the authors to posit that SUI may be related to a decreased response or delayed reaction of the PFMs rather than a function of muscle morphology and strength [19]. A study of mostly parous runners with a mean age of 40 also showed that continent and incontinent women had no significant differences in PFM strength or endurance [20]. Interestingly, in another study on nulliparous professional female athletes with a mean age of 24, incontinent athletes were noted to have greater PFM strength than continent athletes [21]. These findings suggest that PFM weakness alone may not be the primary issue and that there is perhaps some compensatory adaptation of the PFMs in response to an underlying incontinence mechanism. While existing studies have evaluated PFM morphology and strength, additional information on the context of UI, bladder positioning, and bladder function may provide important contextual data to help interpret these findings.

## Other Contributing Factors

Bladder neck descent is a potential contributor to UI in female athletes. A retrospective review of nulliparous adolescent females (mean age 15) with SUI showed that 20 out of 33 subjects demonstrated bladder neck descent on voiding cystourethrogram, 17 of which reported being involved in strenuous activity (hip/hop dance, competitive gymnastics, and varsity sports including tennis and running) [22]. Of the group with bladder neck descent, 8 subjects were ultimately managed with surgery after failing conservative measures including pelvic floor physical therapy (Burch procedure in 5, fascial sling in 2, coaptate injection in 1, and artificial urinary sphincter in 1). Out of the 13 subjects that did not demonstrate bladder neck descent, only 1 was an athlete, and the 8 patients who were not lost to follow-up were successfully managed non-surgically. The authors concluded that in physically active, nulliparous girls with SUI, conservative management was more effective in patients without bladder neck descent from pelvic floor laxity. They report that 2 of their subjects were found to have excessively tight pelvic floor musculature during physical therapy, which could contribute to SUI through an impaired involuntary contraction reflex. They also note the importance of characterizing the PFM architecture in patients with SUI and pelvic floor laxity.

Fatigue of the PFMs is another consideration in UI pathophysiology. In an international survey of 452 female CrossFit competitors with a mean age of 36 years, UI was noted to occur most with jumping, running, and at the end of high-repetition and high-intensity sets [23]. While a large proportion of this cohort was parous, nulliparous female elite trampolinists have previously reported leakage mostly at the end of a training session [24]. PFM fatigue has also been established in young, nulliparous women age 20–30 who reported SUI symptoms during physical activity [25]. Women who underwent a 90-min interval training session involving running, jumping, squats, and lunges had a 20% decrease in the mean maximal voluntary contraction pressure of their PFM (as measured by a vaginal catheter) by the end of the training session. The authors of the study acknowledge that they cannot predict whether short-term fatigue of the PFMs after exercise predicts long-term effects on the muscles. Furthermore, data is conflicting on whether nulliparous female athletes have equal or better PFM endurance compared to non-athletes [16, 17], and PFM endurance did not differ with continence status in older, parous women [20]. It may be that PFM fatigue exacerbates or unmasks existing mechanisms of UI as exercise progresses.

Hypermobility syndromes have also been observed in female athletes with UI. Hypermobility, characterized by inherent connective tissue laxity of joints and ligaments due to increased type III collagen compared to type 1 collagen, is more common in females than males and decreases with age [26]. In the absence of a heritable connective tissue disorder such as Ehlers-Danlos syndrome, the term "benign hypermobile joint syndrome" has historically been used. Diagnosis of hypermobility has historically been based on the Beighton scale, which is based on specific characteristics such as hyperextension of the elbow past 190 degrees and ability to place the palms flat on the floor through trunk flexion [27]. Hypermobility may predispose some athletes to perform well in sports in which flexibility is beneficial. In the previously mentioned study that identified urethral sphincteric incompetence in symptomatic university physical education students, 4 out of 11 symptomatic athletes demonstrated benign hypermobility syndrome on clinical exam, while none of the 11 asymptomatic athletes did [18].

The ultrasound study noting thicker pubovisceral muscles and greater bladder neck descent in athletes compared to non-athletes also noted that 3 out of 24 athletes had joint hypermobility syndrome, 2 of whom had a high stretch ratio of the levator hiatus on Valsalva, while none of the 22 controls did [15]. While hypermobility is certainly not universally observed in young female athletes, it seems that it may be an inherent risk factor for UI (hypermobility may predispose to increased pelvic floor laxity and bladder neck descent) and possibly a reflection of a more elite athlete population (hypermobile athletes may be more likely to compete and train at higher levels that subsequently predispose to UI).

Low energy availability (EA) in female athletes has also been linked with urinary incontinence. Low EA occurs when there is inadequate caloric intake to counter exercise-related expenditures and support basic physiologic functions, which can lead to the clinical syndrome known as relative energy deficiency in sport (RED-S) [28]. This concept expands beyond the traditional "Female Athlete Triad" that is limited to EA, menstrual function, and bone health. In RED-S, multiple physiologic systems may be impaired, including but not limited to menstrual function, bone health, mental health, immune health, metabolism, gastrointestinal function, and cardiovascular health. In a study of nulliparous female athletes age 15–30, females with low EA (defined as meeting eating disorder criteria) had twice the likelihood of UI compared to females without low EA, controlling for sport type and menstrual dysfunction [29•]. This study highlights the involvement of low EA and UI even in non-elite populations, as inclusion criteria for athletes in this study was at least 4 h of physical activity a week for at least 6 months. UI in female athletes with eating disorders and low EA has been postulated to result from weakened PFMs, ligaments, and fascia from malnutrition, increased intra-abdominal pressure from repeated self-induced vomiting, low estrogen, and psychotropic medications. In addition to these factors, functional incontinence is thought to develop from an underlying need for control inherent to eating disorders; this control leads to denial of normal bodily functions and decreased awareness of body stimuli [30]. Of note, the relationship between estrogen and UI is not completely clear. Estrogen has been linked to urethral closure and collagen formation for pelvic floor support, but data on the effect of estrogen on the PFMs is conflicting, and data on the effect of estrogen on UI overall has historically been confounded by other comorbidities and age-related changes [31]. Nonetheless, the contribution of low EA to UI in female athletes appears to be multifactorial.

Other factors may contribute to UI in elite female athletes. Anxiety and depression, which have a higher prevalence in elite athletes compared to the general population [32], have both been observed to be risk factors and exacerbators of overactive bladder (OAB) in the general population [33], although the relationship between anxiety, depression, and UI has not been well characterized in nulliparous female elite athletes. There exists the concept of SUI-induced detrusor overactivity (DO), in which the detrusor contracts reflexively in response to funneling of the bladder neck and decreased urethral pressure seen in patients with SUI [34, 35]. Surgical correction of bladder neck funneling can improve DO in some patients [34], although bladder neck funneling alone is not specific for SUI and can be seen in asymptomatic, nulliparous women [36]. A study on mostly nulliparous female elite athletes across multiple sports found that 95% of symptomatic athletes leaked during training, vs. only 51% during competition, leading the authors to postulate that alpha receptors in the urethra may aid continence during competition in the setting of increased catecholamine levels [37]. Constipation and a history of urinary tract infection increased the odds of UI in a large cross-sectional study of young, mostly nulliparous, female elite athletes [38].

## Management Strategies

Management of UI is dependent on the degree to which it impacts quality of life, and the impact of UI appears to be consistent across different levels of athletic competition. In a meta-analysis of the effect of pelvic floor symptoms on exercise participation in women, 47% of women with UI reported that it negatively affected their exercise participation, although the authors point out that none of the eligible studies focused specifically on elite athletes [39••]. A large cross-sectional study of women exercising predominantly at the non-elite level showed that even in nulliparous women, 31% stopped exercise due to pelvic floor symptoms [40]. The majority of women (58%) reported changing the way they participated in exercise (e.g., running more slowly or not jumping), while others reported changing to a lower impact sport (45%) or decreasing frequency of participation (34%). In young, mostly nulliparous female elite athletes who competed at the international level, 39% of athletes felt that leakage affected performance [41]. Despite consistent findings that UI affects performance, UI is likely under-reported in the clinical setting. In a study of predominantly nulliparous female athletes on sports teams in Portugal (although not designated elite athletes) with a mean age of 23, 61% of athletes had never talked to anyone about their leakage, and only 4.5% had discussed it with a health professional [8]. A focus group discussion revealed that athletes who experienced urine loss felt concern, annoyance, frustration, fear that leakage may be triggered by another activity, and concern that their athletic performance were affected, yet they were not aware of the link between sports and UI, nor did they know about any methods to prevent or treat the

condition. This lack of awareness of exercise-related UI highlights opportunities for both education and development of preventative and management strategies.

A variety of management strategies for UI in female athletes have been reported, and additional strategies have been proposed. Previously reported management strategies in non-elite and/or parous athlete populations include preventive urination, restriction of liquid intake, restricting physical activities, exercising at home or close to toilets, using or avoiding tampons, and wearing dark colored clothing to hide leaks [7, 8, 39••, 42]. In a study of mostly nulliparous female elite athletes with UI competing at the international level, of the athletes who reported applying strategies to reduce visible leakage, 75% used pads [41]. Consideration of pessaries and tampons for urethral support has been proposed based on a small study of middle-aged, parous women using a vaginal sponge during exercise [43, 44]. Pelvic floor muscle training (PFMT) has been recommended as first-line therapy in female athletes based on SUI management in the general population and the lack of serious adverse events with PFMT [43]. A meta-analysis found that physiotherapy in elite female athletes overall had a favorable effect on UI, albeit with substantial heterogeneity among studies [45]. It has also been proposed that athletes fail to fully relax their PFMs, leading to increased urinary frequency and ultimately UI [5], highlighting the importance of identifying the underlying pathophysiology in order to design the most effective interventions. Surgery for UI has not been specifically studied in female athletes. However, in a retrospective study of nulliparous adolescent females with SUI, surgery was observed to be more commonly utilized in patients with bladder neck descent, and many of these patients were athletes [22].

## Future Directions

There exists significant overlap in the UI literature between various populations of female athletes across the spectrum of age, parity, and degree of sport impact and intensity, with varying definitions of what one may consider "athlete" or "elite athlete" status. It is important to establish the population of athletes in any given study to inform the applicability of findings. While the effect of UI on exercise participation has been explored in various female athlete populations, there is limited data on this effect in women who are both young and nulliparous, particularly elite athletes who may be at higher risk due to sport impact and intensity. In addition, there is a role for physiologic studies to better characterize the effect of high-intensity and/or high-impact sports on pelvic floor anatomy and function. These studies should ideally integrate relevant demographic, medical, and sport characteristics, comprehensive urinary symptoms, and quality of life data to help better contextualize any anatomic and/or functional findings, providing insight into how specific factors may affect continence in female athletes. Finally, future studies on both baseline and post-intervention outcomes should include quality of life measures to complement physiologic measures such as the pad test or PFM strength.

## Conclusions

UI in female athletes participating in high-impact sports involving running and jumping is thought to be related to impact-induced changes to the pelvic floor, and prevalence is high in nulliparous elite athletes. PFMs have been observed to be larger in athletes compared to non-athletes as well as in symptomatic compared to asymptomatic athletes. Possible contributing factors to UI include pelvic floor laxity and bladder neck descent, PFM fatigue, eating disorders, and generalized hypermobility. Urinary symptoms negatively affect quality of life in female athletes. Current management strategies are primarily conservative and centered on behavioral modifications and PFMT. In the future, with better understanding of how multiple factors influence UI in elite female athletes, progress can be made in counseling athletes, improving prevention strategies, and optimizing treatment approaches.

**Acknowledgements** Special thanks to Robin Noel for assistance with figure graphics.

### Compliance with Ethical Standards

**Conflict of Interest** The authors declare that they have no conflict of interest.

**Human and Animal Rights and Informed Consent** This article does not contain any studies with human or animal subjects performed by any of the authors.

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