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Urospermia in cattle: effects on seminal quality and fertility

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Abstract. Urospermia, characterized by the presence of urine in the ejaculate, is an important reproductive disorder in cattle that can compromise semen quality and significantly reduce fertility. This condition results from failures in the neurophysiological mechanisms responsible for coordinating seminal emission, ejaculation, and bladder neck closure, and may be associated with neurogenic, anatomical, pharmacological, management-related, or idiopathic factors. Urinary contamination alters the physicochemical parameters of semen, such as pH and osmolarity, and introduces toxic compounds (urea, ammonia, and creatinine), leading to reduced motility, sperm viability, and membrane and acrosome integrity. These effects make ejaculates more susceptible to damage during dilution, cooling, and cryopreservation, negatively impacting artificial insemination programs and reproductive efficiency. Although frequently described in stallions, urospermia also has relevance in modern cattle breeding, particularly in animals used for semen collection in reproductive biotechnologies. This study reviews the main pathophysiological aspects, causes, impacts on semen quality, and practical implications of urospermia in bulls, highlighting the need for accurate diagnosis and appropriate management strategies to minimize its deleterious effects on fertility.

Keywords: urospermia, cattle, semen, fertility, bull.

Introduction

Bovine reproduction plays a fundamental role in the productive efficiency of modern livestock farming, especially in systems that use reproductive biotechnologies such as artificial insemination and semen cryopreservation (Bernardy et al., 2022). In this context, the evaluation of semen quality is essential to ensure satisfactory fertility rates, since physical, chemical, or biological alterations in the ejaculate can directly compromise the fertilizing capacity of sperm cells (Kastelic & Thundathil, 2017). Among the alterations that affect seminal quality, urospermia stands out, a condition characterized by the presence of urine in the ejaculate during emission or ejaculation (Ribeiro et al., 2025). Urospermia occurs due to failures in the neurophysiological mechanisms responsible for coordinating seminal emission, ejaculation, and closure of the bladder neck. Under normal physiological conditions, the sympathetic nervous

system promotes contraction of the internal urethral sphincter during semen emission, preventing urine present in the bladder from refluxing into the urethra at the moment of ejaculation (Canisso & Segabinazzi, 2021). When this control is impaired, semen becomes contaminated with urine, significantly altering the seminal environment and impairing sperm functionality. This condition may be associated with neurogenic factors, anatomical alterations of the urinary tract, pharmacological interference, management failures, and even idiopathic functional disorders (Henry & Echeverri, 2013).

Although frequently described in equines, urospermia is also important in bovine reproduction, especially in animals intended for semen collection for reproductive programs (Canisso & Segabinazzi, 2021). The presence of urine in the ejaculate promotes important alterations in seminal plasma pH and osmolarity, in addition to introducing potentially

toxic substances such as urea, ammonia, and creatinine (Ohashi et al., 2019). These alterations directly affect the structural and functional integrity of spermatozoa, causing reduced motility, decreased cell viability, membrane damage, and impairment of fertilizing capacity (Rabelo, 2009). In addition to the immediate effects on sperm cells, urospermia also represents a technical challenge for semen processing laboratories and artificial insemination centers. Contaminated ejaculates show lower resistance to dilution, refrigeration, and cryopreservation processes, becoming more susceptible to damage caused by freezing and thawing (Ribeiro et al., 2025).

As a consequence, reduced pregnancy rates, lower reproductive efficiency, and economic losses in animal production systems are observed (Kastelic & Thundathil, 2017). The pathophysiology of urospermia involves a complex interaction between neurological mechanisms, autonomic reflexes, and alterations of the lower urinary tract. Control of the bladder neck depends on adequate integration between the sympathetic, parasympathetic, and somatic systems, which are responsible for coordinating urinary retention, seminal emission, and ejaculation (Canisso & Segabinazzi, 2021). Any alteration in this balance may favor urinary reflux during the ejaculatory process. Furthermore, factors related to collection management, such as stress, inadequate sexual stimulation, incorrect use of electroejaculators, and lack of prior bladder emptying, can significantly increase the occurrence of this condition in cattle (Sant'Anna, 2024).

Therefore, understanding the pathophysiological mechanisms involved in urospermia is essential for the diagnosis, management, and prevention of this reproductive disorder (Henry & Echeverri, 2013). The study of this condition contributes not only to the improvement of seminal quality but also to the enhancement of reproductive techniques used in modern cattle production. Thus, the present article aims to review the main aspects related to the pathophysiology of urospermia in cattle, addressing its causes, involved mechanisms, impacts on seminal quality, and consequences for animal fertility.

Contextualization and analysis

Urospermia in cattle should be understood as an integrated failure of the neurophysiological mechanisms coordinating emission, ejaculation, and bladder neck closure (Canisso & Segabinazzi, 2021). Under physiological conditions, emission—a predominantly sympathetic event mediated by α -adrenergic fibers originating from the thoracolumbar segments—simultaneously promotes the propulsion of seminal contents into the pelvic urethra and the functional closure of the bladder neck (vesical trigone). This closure prevents urinary reflux into the urethra during ejaculation. When this system fails, contamination of the ejaculate with urine occurs (Ribeiro et al., 2025).

Mechanisms Allowing the Presence of Urine in Semen

The main pathway leading to urospermia is incompetence of the internal urethral sphincter (bladder neck), which should remain contracted during emission and ejaculation (Canisso & Segabinazzi, 2021). This failure may occur due to:

- ✓ Reduced sympathetic α -adrenergic tone, impairing smooth muscle contraction of the vesical trigone.
- ✓ Lack of coordination between thoracolumbar reflex centers (emission) and sacral reflex centers (ejaculation), resulting in partial or complete opening of the bladder neck during semen passage (Henry & Echeverri, 2013).
- ✓ Presence of residual urine in the pelvic urethra prior to ejaculation, which becomes incorporated into the ejaculate during emission.

Additionally, alterations in the pressure gradient between the bladder and urethra may favor urinary reflux during the rhythmic contractions associated with ejaculation (Rabelo, 2009).

Failure of Bladder Sphincter Control

Control of the bladder neck depends on a delicate balance among: sympathetic innervation (contraction → urinary retention); parasympathetic innervation (bladder relaxation and micturition); and indirect somatic control through coordination of the ejaculatory reflex (Canisso & Segabinazzi, 2021). Urospermia is frequently associated with reduced sympathetic activity or pharmacological interference in this system (e.g., sedatives or α -adrenergic blockers), leading to the inability to maintain bladder neck closure during emission (Ribeiro et al., 2025).

Possible Causes

The etiology of urospermia is multifactorial and may include (Henry & Echeverri, 2013; Ohashi et al., 2019):

- ✓ Pharmacological factors: sedatives, anesthetics, or muscle relaxants that depress the autonomic nervous system.
- ✓ Stress and inadequate sexual stimulation: interfering with integration between central stimuli and peripheral reflexes (Sant'Anna, 2024).
- ✓ Age or body condition: debilitated animals may exhibit reduced neuromuscular control efficiency (Bernardy et al., 2022).
- ✓ Collection frequency: excessively frequent collections may impair reflex coordination (Rabelo, 2009).

Neurological Disorders

Lesions or dysfunctions affecting the neuroanatomical pathways involved (thoracolumbar and sacral) may directly compromise emission and ejaculation reflexes (Canisso & Segabinazzi, 2021). The main mechanisms include:

- ✓ Peripheral neuropathies affecting the hypogastric or pudendal nerves.
- ✓ Spinal cord lesions interrupting communication between reflex centers and cortical control.
- ✓ Degenerative or inflammatory conditions altering nerve conduction (Henry & Echeverri, 2013).
- ✓ These alterations result in loss of synchronization between contraction of the ductus deferens, accessory sex glands, and bladder neck closure (Ribeiro et al., 2025).

Improper Collection Management

Management-related factors are common and often underestimated causes (Sant'Anna, 2024):

- ✓ Failure to empty the bladder prior to semen collection.
- ✓ Improper use of electroejaculators, with excessive or poorly synchronized stimulation.
- ✓ Stressful environments or lack of adequate sexual stimulation.
- ✓ Inappropriate intervals between collections (Bernardy et al., 2022).
- ✓ These factors may induce partial reflex urination or relaxation of the bladder neck during semen collection (Rabelo, 2009).

Alterations in the Urinary Tract

Anatomical or pathological changes in the lower urinary tract also contribute to urospermia (Ohashi et al., 2019):

- ✓ Urinary tract infections (cystitis), altering sensitivity and bladder neck control.
- ✓ Inflammation or lesions of the vesical trigone.
- ✓ Urolithiasis, which may mechanically interfere with urinary flow and proper sphincter closure.
- ✓ Congenital or acquired structural abnormalities (Henry & Echeverri, 2013).

Furthermore, the presence of urine in semen promotes significant physicochemical alterations, such as pH reduction, increased osmolarity, and the presence of toxic compounds (urea and creatinine), which directly impair sperm motility and viability—a critical aspect in semen quality evaluation (Ribeiro et al., 2025).

pH Alterations

Bovine semen presents a slightly neutral to alkaline pH, an essential condition for maintaining sperm motility and structural stability (Rabelo, 2009). Urine, in contrast, may exhibit variable pH values (often more acidic), leading to acidification of the seminal environment. This alteration causes (Henry & Echeverri, 2013):

- ✓ Destabilization of intracellular enzymatic activity in spermatozoa.
- ✓ Reduced efficiency of flagellar beating.
- ✓ Impairment of sperm capacitation.

- ✓ The decrease in pH is directly associated with reduced progressive motility and increased numbers of immotile or erratically moving sperm cells (Ribeiro et al., 2025).

Osmolarity Alterations

Seminal plasma osmolarity is tightly regulated and essential for cellular homeostasis (Ohashi et al., 2019). Urine presents highly variable osmolarity and may be either hypotonic or hypertonic relative to semen. The introduction of urine into the ejaculate results in (Canisso & Segabinazzi, 2021):

- ✓ Osmotic imbalance, leading to excessive water influx or efflux.
- ✓ Cellular swelling or shrinkage of spermatozoa.
- ✓ Alterations in plasma membrane conformation.
- ✓ These events directly compromise membrane integrity, a structure essential for sperm survival and functionality (Rabelo, 2009).

Direct Damage to Spermatozoa

In addition to physicochemical alterations, urine contains potentially deleterious compounds such as urea, ammonia, and nitrogenous waste products, which exert cytotoxic effects on spermatozoa (Ribeiro et al., 2025). The main impacts include (Henry & Echeverri, 2013):

- ✓ Marked reduction in sperm motility, especially progressive motility.
- ✓ Increased plasma membrane permeability.
- ✓ Induction of acrosomal damage.
- ✓ Mitochondrial alterations, reducing ATP production.
- ✓ Prolonged exposure or higher concentrations may lead to irreversible loss of membrane integrity, completely compromising fertilizing potential (Ohashi et al., 2019).

Disorganization of the Seminal Environment

Seminal plasma acts as a highly specialized support system containing proteins, ions, and antioxidant factors that preserve sperm functionality (Rabelo, 2009). The presence of urine disrupts this balance by (Canisso & Segabinazzi, 2021):

- ✓ Diluting essential seminal plasma components.
- ✓ Altering ionic concentrations (Na^+ , K^+ , Cl^-).
- ✓ Reducing the buffering capacity of the medium.
- ✓ Favoring adverse oxidative conditions (Ribeiro et al., 2025).

Consequently, the seminal environment becomes hostile, accelerating sperm degeneration and drastically reducing ejaculate quality (Henry & Echeverri, 2013).

Decreased Sperm Motility

Sperm motility, especially progressive motility, is one of the first parameters affected (Rabelo, 2009). Acidification of the medium, associated with osmotic imbalance and the action of urinary metabolites, interferes with mitochondrial function and ATP production (Ohashi et al., 2019). Consequences include (Ribeiro et al., 2025):

- ✓ Reduced movement speed and linearity.
- ✓ Increased abnormal motility patterns (circular or vibratory movement).
- ✓ Increased percentage of immobile sperm cells.
- ✓ These alterations limit the ability of spermatozoa to traverse the female reproductive tract (Kastelic & Thundathil, 2017).

Structural Damage

The effects of urospermia are not limited to function but also affect sperm morphology (Henry & Echeverri, 2013):

- ✓ Lesions of the plasma membrane and acrosome.
- ✓ Alterations in the midpiece affecting mitochondrial function.
- ✓ Flagellar damage impairing motility.
- ✓ Possible fragmentation of internal structures (Ribeiro et al., 2025).
- ✓ These structural damages are frequently associated with irreversible loss of fertilizing capacity (Ohashi et al., 2019).

Impact on Artificial Insemination

In artificial insemination programs, semen quality is essential for reproductive success (Kastelic & Thundathil, 2017). Ejaculates affected by urospermia present (Rabelo, 2009):

- ✓ Lower pregnancy rates.
- ✓ Reduced efficiency of insemination doses.
- ✓ Greater variability in reproductive outcomes (Bernardy et al., 2022).

Even moderate contamination levels may significantly compromise performance, rendering the semen unsuitable for commercial or technical use (Ribeiro et al., 2025).

Impact on Semen Cryopreservation

Cryopreservation amplifies the negative effects of urospermia (Canisso & Segabinazzi, 2021). Previously compromised spermatozoa exhibit lower resistance to the thermal and osmotic stress associated with freezing and thawing processes (Ohashi et al., 2019). The main effects include (Ribeiro et al., 2025):

- ✓ Marked reduction in post-thaw motility.
- ✓ Increased membrane rupture.
- ✓ Reduced acrosomal integrity.
- ✓ Lower cellular survival rates (Rabelo, 2009).

Furthermore, alterations in the seminal environment impair cryoprotectant action, reducing the efficiency

of semen freezing protocols (Henry & Echeverri, 2013).

Treatment and Management Strategies

Treatment is challenging, and the prognosis for complete resolution is often unfavorable (Canisso & Segabinazzi, 2021). Strategies are divided into pre- and post-ejaculatory actions (Ribeiro et al., 2025):

Pre-ejaculatory Strategies (Prevention):

Bladder Emptying: The simplest method is encouraging the animal to urinate immediately before collection or breeding. Other options include urethral catheterization to drain urine or administration of diuretics (Sant'Anna, 2024).

Pharmacological Induction: The use of imipramine hydrochloride (a tricyclic antidepressant) is indicated to increase bladder sphincter tone, assisting bladder neck closure during ejaculation (Canisso & Segabinazzi, 2021).

Post-ejaculatory Strategies (Semen Processing):

Immediate Dilution: Adding extenders (especially milk-based extenders) directly into the collection cup at the time of ejaculation helps minimize the biochemical shock caused by urine (Rabelo, 2009).

Fractionated Collection: Attempting to collect only the sperm-rich fraction while avoiding contaminated fractions (Henry & Echeverri, 2013).

Washing and Selection Techniques: Density gradient centrifugation (e.g., Percoll) is used to separate viable and morphologically normal spermatozoa from urine and debris (Ohashi et al., 2019). Cushion centrifugation and filtration using specific filters (such as Sperm Filter) have proven to be viable alternatives (Canisso & Segabinazzi, 2021).

Although management allows the use of semen with low contamination levels for cryopreservation or immediate insemination, the impact of urine is time-dependent; the deleterious effect becomes much more intense after 30 minutes of exposure (Ribeiro et al., 2025).

Management Strategies

Urination Stimulation: Encouraging the animal to urinate immediately before semen collection in order to empty the bladder (Sant'Anna, 2024).

Drug Use: Treatment with imipramine hydrochloride has been used to increase bladder sphincter tone and reduce the amount of urine in semen samples (Canisso & Segabinazzi, 2021).

Immediate Dilution: Adding extender directly into the collection cup at the moment of ejaculation to minimize the biochemical shock caused by urine (Rabelo, 2009).

Table 1 – Comparison between normal bovine semen and semen contaminated with urine (urospemia).

Parameter	Normal Bovine Semen	Semen with Urospermia	Observed Impact
Seminal pH	Slightly neutral to alkaline	Acidification or marked pH alteration	Reduction in motility and enzymatic activity
Osmolarity	Stable and controlled	Hypotonic or hypertonic	Cellular swelling or shrinkage
Sperm motility	High progressive motility	Reduced motility and erratic movements	Lower fertilizing capacity
Membrane integrity	Preserved	Increased permeability	Greater cellular degeneration
Acrosomal integrity	Maintained	Acrosomal lesions	Impaired fertilization
Mitochondrial function	Adequate ATP production	Mitochondrial alterations	Reduced cellular energy
Sperm viability	High	Progressive reduction	Lower sperm longevity
Cryopreservation	Good resistance to freezing	High sensitivity to thermal stress	Lower post-thaw motility
Fertility	Normal pregnancy rates	Decrease in reproductive rates	Reduced AI efficiency

Source: Adapted from Henry and Echeverri (2013), Ohashi et al. (2019), and Rabelo (2009), with data related to bovine andrology, seminal quality, reproductive management, and clinical reproductive alterations in bulls.

Table 2. Comparison of the main causes of urospermia in cattle

Cause	Pathophysiological Mechanism	Main Consequence
Neurogenic alterations	Failure in sympathetic innervation of the bladder neck	Urinary reflux during ejaculation
Use of sedatives / α -adrenergic blockers	Reduced tone of the internal urethral sphincter	Inadequate relaxation of the bladder neck
Improper collection management	Excessive stimuli or absence of bladder emptying	Ejaculate contamination
Stress and low sexual arousal	Reflex discoordination between emission and ejaculation	Alteration in ejaculatory dynamics
Cystitis and inflammations	Functional alteration of the vesical trigone	Difficulty in bladder closure
Urolithiasis	Mechanical interference in urinary flow	Sphincter dysfunction
Advanced age / debility	Reduced neuromuscular efficiency	Increased predisposition to urospermia

Source: Adapted from Henry and Echeverri (2013), Ohashi et al. (2019), and Rabelo (2009), with data related to bovine andrology, seminal quality, reproductive management, and clinical reproductive alterations in bulls.

Comparison Between Urospermia and Other Seminal Alterations

Urospermia differs from other seminal alterations because it simultaneously promotes chemical, osmotic, and toxic changes in the ejaculate (Ribeiro et al., 2025). While reproductive inflammatory processes mainly compromise cellularity and leukocyte presence in semen, urospermia directly alters the seminal environment through the presence of urea, creatinine, ammonia, and abrupt changes in pH and osmolarity (Henry & Echeverri, 2013). These modifications produce faster effects on motility and membrane integrity of spermatozoa (Ohashi et al., 2019).

Furthermore, compared to primary sperm defects of testicular origin, urospermia has a predominantly post-ejaculatory impact. In other words, sperm cells may be produced normally by

the testes but become nonviable after contact with urine during ejaculation (Rabelo, 2009). This aspect makes the condition particularly important in artificial insemination and semen cryopreservation programs (Canisso & Segabinazzi, 2021).

Comparison Between Physiological Collection and Electroejaculation

Collection by artificial vagina more closely reproduces physiological ejaculation and presents a lower risk of urinary contamination compared with electroejaculation (Sant'Anna, 2024). According to reports in animal andrology studies, excessive stimulation during electroejaculation may induce inadequate relaxation of the bladder neck and simultaneous reflex urination, favoring episodes of urospermia (Henry & Echeverri, 2013).

In physiological collection, adequate sexual stimulation favors greater synchronization between seminal emission and closure of the internal urethral sphincter (Bernardy et al., 2022). However, factors such as stress, inadequate environment, and low libido may also compromise reflex coordination even during physiological collection (Rabelo, 2009). Studies in bovine andrology demonstrate that behavioral, environmental, and management factors directly influence reproductive efficiency and seminal parameters in breeding bulls (Sant'Anna, 2024).

Relationship Between Seminal Quality and Fertility in Cattle

Studies in bovine andrology demonstrate that alterations in sperm parameters are directly related to conception rates in reproductive programs (Kastelic & Thundathil, 2017). Among the parameters most sensitive to environmental alterations are progressive motility, membrane integrity, and mitochondrial function, which are precisely the main components affected by urospermia (Ohashi et al., 2019).

In addition, research involving semen cryopreservation shows that spermatozoa previously subjected to osmotic or oxidative stress exhibit lower resistance to freezing and thawing processes (Ribeiro et al., 2025). Thus, ejaculates contaminated with urine present lower post-cryopreservation survival potential and lower efficiency in fixed-time artificial insemination (FTAI) protocols (Canisso & Segabinazzi, 2021).

Conclusion

Urospermia in cattle represents a significant challenge for modern animal reproduction, particularly in systems that rely on biotechnologies such as artificial insemination and semen cryopreservation (Ribeiro et al., 2025). The presence of urine in the ejaculate directly and multifactorially compromises semen quality, altering essential physicochemical parameters such as pH and osmolarity, while introducing toxic compounds that reduce motility, viability, and structural integrity of spermatozoa (Henry & Echeverri, 2013; Ohashi et al., 2019). These effects negatively impact fertility, leading to lower pregnancy rates and reduced reproductive efficiency (Kastelic & Thundathil, 2017). The etiology of urospermia is complex, involving neurogenic, pharmacological, anatomical, and management-related factors, which reinforces the need for accurate diagnosis and appropriate prevention and control strategies (Canisso & Segabinazzi, 2021). Inadequate collection practices and unfavorable environmental conditions may further increase the occurrence of this disorder, making technical training and careful management of breeding bulls indispensable (Sant'Anna, 2024). Therefore, understanding the pathophysiological mechanisms of urospermia and its impacts on semen quality is fundamental to minimizing economic losses and ensuring greater efficiency in

reproductive programs (Rabelo, 2009). Investments in diagnostic methods, proper management, and safer collection protocols are essential measures to reduce the incidence of this condition and preserve the reproductive potential of bulls used in reproductive biotechnologies (Bernardy et al., 2022).

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