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Fertility challenges in dogs: A review

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Abstract
The domestic male dogs are non-seasonal breeder. The female's reproductive cycle is characterized by prolonged proestrus and estrus phases, and they are monoestrus. Prior to ovulation, the estrogen peak that occurs during the estrous cycle is accompanied by an increase in the amount of progesterone in the blood. Diestrus follows estrus, followed by anestrus. The hypothalamic-pituitary-gonadal axis controls the ovarian cycle. Testosterone is an essential component of the hypothalamic-pituitary-gonadal axis, which regulates male spermatogenesis. Domestic dogs and the majority of wild canids have comparable reproductive cycles. After diestrus, there is a prolonged period of ovarian inactivity. Numerous factors contribute to dog breeding failure, and a precise diagnosis necessitates extensive research. Breeding failures can be two types, infectious and non-infectious. The majority of reported cases of bacterial endometritis were discovered to be among the infectious causes in the bitch. Endometrial degenerative disorders, uterine cysts, and primary and secondary Anoestrus are examples of non-infectious causes. Infectious and non-infectious reasons can also affect males. Other non-infectious causes of male infertility include acquired anatomical defects and bilateral cryptorchidism. Infertility is caused by prostatitis, spermatocele or sperm granulomas, genital duct obstruction, or inguinal or scrotal hernia. Orchitis and epididymitis are caused by infections that change the quality of semen. There are significant effects of nutrition on reproductive function as well.

Keywords: Infertility, nutrition, dog, failures, infection

1. Introduction
Due to the growing popularity of purebred dogs as pets and the rising demand for these expensive pets, owners now often discuss fertility-related issues with their veterinarian. Domestic dogs are non-seasonal breeders, in contrast to their wild counterparts such as the grey wolf, coyote, and dingo, which reproduce seasonally (Anke *et al*., 1989) [1]. The reproductive cycle of the female dog is monoestrous, featuring extended proestrus and estrus phases, each lasting around nine days. Estrus is characterized by a surge in estrogen levels that coincides with a progressive increase in progesterone levels, occurring just before ovulation (Ariu *et al*., 2016) [2]. Following estrus, the diestrus phase begins, lasting approximately two months, irrespective of whether fertilization has occurred. This is succeeded by the anestrus phase, which can span from two to ten months and is marked by a period of ovarian dormancy (Arlt *et al*., 2012) [3].
In female dogs (bitches), the ovarian cycle is controlled by the hypothalamic-pituitary-gonadal (HPG) axis (Bindari *et al*., 2013) [7]. Prior to the onset of proestrus, there is an increased frequency of gonadotropin-releasing hormone (GnRH) pulses from the hypothalamus, which in turn stimulates the anterior pituitary to secrete follicle-stimulating hormone (FSH) and luteinizing hormone (LH) (Arlt *et al*., 2012) [3]. This elevation in gonadotropin release promotes follicular development and activates gonadal steroidogenesis. The resulting rise in estrogen levels during proestrus induces a surge in LH, which leads to ovulation approximately 60 hours later (Boland *et al*., 2001) [8]. Canine oocytes possess distinct features, notably a high concentration of cytoplasmic lipids compared to those of other mammalian species (Canfield *et al*., 1990) [11]. Lipid yolk bodies initially emerge within the cytoplasm of

the primary oocyte and continue to accumulate throughout oogenesis, resulting in a characteristically dark appearance that sets them apart from oocytes of other mammals (Chapman *et al*., 1997) [13].

The spatial distribution of lipid bodies within canine oocytes varies depending on the reproductive stage. Oocytes collected during the follicular phase exhibit a diffuse dispersion of lipid bodies, whereas those obtained during anestrus or the luteal phase display a more localized distribution, either at the periphery or around the nucleus (Chastant-Maillard *et al*., 2011) [14].

In dogs, oocytes are ovulated at an immature stage and typically require 48 to 72 hours within the oviduct to complete nuclear maturation (Chlopik *et al*., 2020) [15]. Following maturation, the oocyte retains its fertilization capacity for approximately 4 to 5 days and remains viable for up to 6 to 7 days after ovulation (Comizzoli *et al*., 2009) [19].

In bitches, fertilization typically takes place in the mid to distal region of the oviduct. Two-pronuclei zygotes are generally observed around 92 hours after ovulation in dogs, and between 29 to 73 hours post-mating in raccoon dogs (Concannon *et al*., 2009) [20].

Bitch infertility is frequently caused by structural, physiological, neoplastic, and viral factors. Bitch infertility can be caused by structural factors that interfere with conception or are typified by bitch ambiguous genitalia. Physiological abnormalities rank as the second most significant cause of infertility among bitches. These anomalies appear as silent heat, split heat, extended anoestrus, and irregular ovulation. The bitch is unable to carry a typical pregnancy due to infectious causes of infertility that are spread during coitus or during estrous.

Male dogs are non-seasonal breeders, with spermatogenesis occurring continuously throughout the year. In contrast, spermatogenesis in strictly seasonal canids is restricted to the breeding season (Concannon *et al*., 2012) [21]. This process is regulated by the hypothalamic-pituitary-gonadal (HPG) axis, with testosterone serving as a key regulatory hormone (Concannon *et al.*, 2011) [22].

In dogs, spermatozoa can be collected once sexual maturity is attained, typically between 6 to 8 months of age (De Bosscher *et al*., 2001) [26]. Maturation of sperm occurs within the epididymis, and the gametes gain the capacity to fertilize upon reaching the cauda epididymal region (De los Reyes *et al*., 2009) [28]

Research in domestic dogs has demonstrated that the complete spermatogenic cycle spans approximately 62 days. Following ejaculation, spermatozoa are capable of surviving within the female reproductive tract for up to 7 days (Comizzoli *et al.*, 2009) [19].

In vitro studies have shown that canine spermatozoa are capable of penetrating immature oocytes (Dodgson *et al*., 2012); [29] however, *in vivo* fertilization does not occur until approximately 83 hours after ovulation, even when sperm are already present in the reproductive tract. Additional *in vitro* findings suggest that metaphase II oocytes in dogs may need an extra 12 to 24 hours to attain full developmental competence, typically corresponding to 5-6 days following the LH surge (Dooley *et al*., 1990) [30].

Infertility can be categorized as acquired if it develops after the animal has become fertile or congenital if it manifests early in sexual life. Normal libido, whether present or absent, aids the clinician in classifying infertility and narrowing down the range of potential reasons. The inability to mate, lack of desire, and inability to produce litters following typical mating are the three most prevalent concerns regarding male canine infertility.

Breeding failure in dogs

Breeding failure is a common issue in dogs and may arise from a variety of causes, affecting both males and females (bitches). Accurate diagnosis necessitates a comprehensive evaluation, including detailed history taking (Durrant *et al*., 1998) [31], physical examination, and laboratory testing (Elrod *et al*., 1993) [32]. Laboratory investigations typically involve microbial culture of vaginal swabs, cytological assessments, evaluation of semen quality, and analysis of dietary factors.

Breeding failure in dogs may result from either infectious or non-infectious factors. Among the infectious causes, bacterial endometritis is identified as the leading contributor in most reported cases involving bitches. Non-infectious causes encompass conditions such as primary and secondary anoestrus, uterine cysts, and degenerative changes of the endometrium (England and Verstegen, 2001; England *et al*., 2012). [33, 34] In male dogs, breeding failure may also arise from both infectious and non-infectious causes. Non-infectious factors contributing to infertility include bilateral cryptorchidism, acquired anatomical defects, spermatocele or sperm granulomas, inguinal or scrotal hernias, and prostatitis (Farstad *et al*., 1989) [35]. Infectious conditions such as orchitis or epididymitis can compromise semen quality, thereby affecting fertility (Farstad *et al*., 1989) [35]. Additionally, nutrition plays a critical role in reproductive function; inadequate nutrition can lead to poor body condition, delayed puberty, and eventual infertility (Feldman *et al*., 1996) [36].

Infertility in the Bitch

Infertility in bitches refers to the inability to conceive or produce offspring, which may result from a range of factors including systemic illnesses, organ dysfunctions, infectious agents, and hormonal imbalances. Accurate diagnosis necessitates the collection of a detailed case history encompassing signalment, general health status, prior medical treatments, and the reproductive history of the male used for mating (Duuant *et al*., 1998) [31]. A structured and systematic diagnostic approach is essential for identifying the underlying cause (Elrod *et al*., 1993) [32].

Infectious causes

Various pathogenic organisms can contribute to infertility in bitches. Among them, bacterial endometritis is recognized as a primary cause in a significant number of cases. Bacterial species such as *Pasteurella multocida*, Group G *Streptococcus*, *Staphylococcus intermedius*, *Escherichia coli*, and *Proteus mirabilis* have been isolated from the uteri of infertile bitches, with the origin of these pathogens likely being the cranial vagina (Fontaine *et al*., 2009) [37]. Additionally, *Brucella canis* a species-specific pathogen is known to induce infertility through embryonic resorption, late-term abortion, or the birth of weak or clinically normal pups that may serve as sources of infection (Fontbonne *et al*., 1999) [38].

Brucella Canis

A Gram-positive bacterium called B. canis is capable of causing infertility and abortion.

It is the only known bacterium that specifically causes bitches to become infertile. Initially discovered in the United States (Moore and Bennett, 1967; Carmichael and Kenney, 1968) [71,

[12], Brucella infertility has now spread to a number of other nations. B. canis can spread by a number of routes, including as congenital infection, sexual transmission, contact with the vaginal secretions of infected bitches, and contact with aborted fetal or placental tissue. According to Moore and Gupta (1970) [72], venereal disease is the most prevalent type of infection. The majority of abortions take place between days 45 and 55 of pregnancy, though early fetal resorption, stillbirth, or, less frequently, poor pups may occur.

Although the condition can be diagnosed by isolating the bacterium from blood or aborted tissue, a negative blood culture does not necessarily mean that an infection is not present because the bitch may not be bacteraemic for extended periods of time.

It is fortunately easy to diagnose infections using the plate agglutination test for screening and tube agglutination for confirmation; titres of 1:200 or above are indicative of infection. In clinical situations, treating the illness with a combination of streptomycin and tetracycline is frequently successful; yet, antimicrobial therapy does not eradicate the organism from tissues (Johnston *et al.*, 1982) [55].

Toxoplasma gondii

T. gondii infection results in fetal mortality, stillbirth, early birth, and abortion (Cole *et al*., 1954; Siim *et al*., 1963) [18, 88]. The virus can be passed on to surviving infected puppies. A toxoplasma infection's effects on public health should be taken into account at the time of diagnosis.

Canine herpesvirus

In most cases, adult dogs with canine herpesvirus only exhibit a few minor symptoms that are restricted to the genital or respiratory system. But the virus can also induce vaginal lesions in the bitch, which can lead to stillbirths, abortions, and infertility (Hashimoto and Hirai, 1986) [47].

Placental lesions and fetal infection seem to be the outcomes of the pregnant bitch's infection (Hashimoto *et al*., 1979) [49]. Small, greyish white foci with localized degeneration, necrosis, and eosinophilic intranuclear inclusion bodies are present in the macroscopically undeveloped infected placentae. In the vestibule, variable-sized vesicles are regularly seen (Hashimoto *et al*., 1983) [48]. These lesions are often visible at the start of pro-oestrus, indicating that venereal transmission is likely significant in adult dogs.

Canine adenovirus

Canine adenovirus infection during pregnancy is known to cause the birth of frail or dead pups that pass away a few days after parturition (Spalding *et al*., 1964) [94]. However, the virus is typically consumed and results in newborn death (Cornwell, 1984) [23].

Cystic endometrial hyperplasia and pyometra

Pyometra, also known as cystic endometrial hyperplasia, is the most severe uterine condition that affects bitches (Kida *et al*., 2006) [60]. Pyometra typically develops 20-70 days following the termination of heat (Bigliardi *et al*., 2004) [6]. The uncontrolled proliferation of endometrium under progesterone with inflammatory cells in the uterine layers is a defining feature of this diestus illness (Zdunczyk *et al*., 2006) [107]. A higher progesterone concentration has the effect of lowering uterine immunity (Sugiura *et al*., 2004) [96]. The three main causes of pyometra are neoplastic ovarian disorders, protracted estrus, and cystic ovaries (Kida *et al*., 2006) [60]. E. coli adheres to the uterus thanks to changes in the hormones produced by the ovaries and their receptors in the blood (de Bosschere *et al*., 2002) [25]. When progesterone is used to interrupt estrus, the uterus's inflammatory response is triggered (Noakes *et al*., 2001) [75]. Insulin-like Growth Factor 1 has been shown to have greater amounts in endometrial epithelial cells and contribute to the development of cystic endometrial hyperplasia (DeCock *et al*., 2002) [27]. According to Arora *et al*. (2006) [4], bacterial infections and hormonal imbalances are two possible causes of cystic endometrial hyperplasia. According to recent research, the window of opportunity for pyometra caused by E. coli is 11-21 days following the peak of LH (Tsumagari *et al*., 2005) [101]. The symptoms of pyometra include mucoid vaginal discharge from endometrial deterioration and a tomato soup-like taste (Switonski *et al*., 2000) [98]. Off-feeding, elevated body temperature and polyuria are further indicators of pyometra (Bedrica and Sacar, 2004; Fransson *et al*., 1997) [5, 39]. The uterus seems fluid-filled on ultrasonography (Bigliardi *et al*., 2004) [6]. There are two forms of pyometra that are usually found: close and open. When intoxication causes the body temperature to rise, this is known as closed pyometra. The closed pyometra also has an increase in leukocyte content, going from 15,000 to 60,000/mm^3 (Bigliardi *et al*., 2004) [6]. The histological abnormalities include degenerative changes in the nucleus's shape, bacterial colonies, and massive cystic endometrial glands (Groppetti *et al*., 2010) [45]. For older bitches, ovariohysterectomy is the preferred course of treatment (MacIntire *et al*., 2004) [63]. Prostaglandin (PGF2α) is administered to young bitches with open pyometra upon the owner's request to maintain reproductive activity (Gilbert *et al*., 1989) [40]. Until the uterus returns to its normal structure, PGF2 α is subcutaneously injected at a dose of 250 ug/kg (extremely high dose needs confirmation) every 12 hours for three to five days (Meyers-Wallen *et al*., 1986) [66].

Non-infectious causes: Congenital abnormalities

Structural abnormalities of the vulva, vestibule, and vagina such as circumferential vaginal strictures can contribute to infertility in bitches by preventing normal copulation (Hemler *et al*., 1980) [52]. Although the occurrence of infertility due to these anatomical defects is relatively rare, a comprehensive reproductive examination before the first mating is essential. In cases where vaginal septa are identified, surgical removal is a viable corrective option (Hollett *et al*., 2006) [53].

Ovarian agenesis is uncommon and does not result in infertility unless it affects both ovaries. There have also been reports of ovarian dysplasia in bitches with an unusually high number of chromosomes (Johnston *et al*., 1985) [56]. Bitches often have caudal reproductive tract strictures. These might result in symptoms of chronic vaginitis or vulval pruritis (Holt and Sayle, 1981; Soderberg, 1986) [54, 91].

It is uncommon for external genitalia to have congenital abnormalities. It is known that vulval hypoplasia is linked to perivulval dermatitis (Christiansen, 1984) [16]; however, there is insufficient evidence to link this condition to early neutering.

Neoplasia

Bitches rarely get ovarian tumors, which make up around 1% of all neoplasms (Cotchin, 1961; Hayes and Harvey, 1979) [24, 51]. Ovarian neoplasia is more common in older dogs [; it often manifests at 8 years of age (Withrow and Susaneck, 1986) [105]. Germ cell, epithelial, or sex cord stromal tumors can all arise from the ovaries. The most significant are

granulosa cell tumors, which have the potential to grow to enormous sizes and manifest ascites or other mass effect-related symptoms. Radiography, ultrasound, abdominal palpation, and clinical symptoms are typically used to diagnose ovarian tumors (Goodwin *et al*., 1990) [42].

The incidence of uterine tumors is low (Brodey and Roszel, 1967) [9]. Fibroleiomyomata have been the most commonly documented instances of these lesions. Although they are discreet and benign, they can bleed and produce a bloody vulval discharge. Cervical tumors are uncommon, although benign vaginal and vestibule tumors, such as fibromata, fibroleiomata, and lipomata, are more prevalent (Withrow and Susaneck, 1986) [105].

The bitch's vagina, external genitalia, and canine penis are all impacted by the transmissible venereal tumor (TVT). When the recipient's vaginal mucosa is "seeded" by infected cells during coitus, the tumor is transmitted (Cohen, 1974) [17]. Licking the tumor may cause auto-transmission to the nasal and oral mucosa. Usually reaching their maximum size after 5-7 weeks, the lesions—which can be single or many and are frequently friable and multilobulated regress spontaneously within 6 months (Moulton, 1961) [73]. Several chemotherapy regimens, such as vincristine and cyclophosphamide, as well as radiation therapy and surgical debulking have been reported (Calvert *et al*., 1982; Thrall, 1982) [10, 100].

Cystic conditions of the uterus

Cyclic bitches are susceptible to proliferative and degenerative disorders of the endometrium, with cystic endometrial hyperplasia (CEH) being the most commonly observed condition (England and Verstegen, 2001; England *et al*., 2012) [75, 34]. CEH primarily results from the repeated hormonal stimulation of the endometrium by estrogen and progesterone, a process exacerbated by the delayed downregulation of estrogen receptors. This condition is associated with reduced uterine perfusion, fluid accumulation, inflammation, and impaired uterine clearance following mating (England *et al*., 2012) [34]. Similar histopathological changes may also be induced by local bacterial irritation (Goto and Noda, 1992; Hharrison *et al*., 1984) [44, 46].

Infertility can occur even in bitches exhibiting regular estrous cycles, often due to progressive cystic degeneration of the endometrium (England and Moxon, 2012; Feldman *et al*., 1996) [34, 36]. Degenerative endometrial conditions such as glandular fibrosis, pseudoplacentational endometrial hyperplasia, and chronic endometritis are among the most commonly observed pathological findings in infertile bitches (Haslett *et al*., 2002) [50].

Primary and secondary anoestrus

Primary anoestrus, defined as the absence of estrus by 24 months of age, may result from underlying organ dysfunction or previous medical treatments (Elrod *et al*., 1993) [32]. Diagnosis involves a detailed case history and the exclusion of conditions such as silent heat, genetic abnormalities affecting sexual development, hypothyroidism, and other systemic illnesses (Duuant *et al*., 1998) [31]. Notably, a case of primary anoestrus linked to diet-induced hypothyroidism has been documented (Goff *et al*., 1999) [41]. In contrast, secondary anoestrus is characterized by an extended inter-estrus interval (Gorlinger *et al*., 2005) [43].

Hormonal imbalances

Prolonged estrus in bitches may be associated with hypoestrogenism, characterized by a lack of receptive behavior and reduced vaginal mucus secretion (Johnston *et al*., 2001) [57].

Hypoluteoidism refers to inadequate luteal function during pregnancy, wherein serum progesterone levels fall below 5 ng/ml between the 4th and 5th weeks of gestation, often resulting in embryonic resorption or abortion (Johnton *et al*., 1994) [58]. This condition can be classified as either primary or secondary. Primary hypoluteoidism occurs without any identifiable cause, whereas secondary hypoluteoidism arises due to infectious or non-infectious disturbances during pregnancy that lead to fetal stress (Keenan *et al*., 1998) [59].

The diagnosis of primary luteal deficiency is largely based on the exclusion of secondary causes. However, the existence of primary luteal insufficiency due to intrinsic dysfunction of the corpora lutea remains a subject of debate (Durrant *et al*., 1998) [31]. A thorough case history is essential, as recurrent pregnancy losses may suggest underlying luteal insufficiency or concurrent endocrine disorders such as hypothyroidism.

The influence of hypothyroidism on fertility in dogs remains a topic of debate. Some studies have reported adverse effects, including reduced conception rates, increased peri-parturient mortality, and lower birth weights in puppies (Krassas *et al*., 2004) [61]. Conversely, other investigations have found no significant difference in the incidence of reproductive disorders between hypothyroxinemic and euthyroid dogs (Linde-Forsberg *et al*., 2001) [62].

In cases of recurrent fetal resorption or abortion, assessment of serum thyroxine and thyroid-stimulating hormone (TSH) levels is recommended. It is important to note that thyroid dysfunction may also result from systemic or organ-specific diseases. While most instances of hypothyroidism are acquired, congenital forms have also been documented (Makler *et al*., 1981) [64].

Infertility in the Male Dog

Male infertility in dogs remains poorly understood, with the underlying cause remaining unidentified in approximately 70% to 74% of cases (Durrant *et al*., 1998) [31].In human medicine, poor semen quality is often managed using assisted reproductive technologies such as *in vitro* fertilization (IVF) or intracytoplasmic sperm injection (ICSI); however, these techniques are not yet widely accessible or standardized for canine use (Mehta *et al*., 2015) [65]. The overall prognosis for infertility in male dogs is generally poor. Therefore, a thorough clinical evaluation is essential, as infertility may be an early indicator of an underlying systemic health disorder.

Anatomical abnormalities

Bilateral cryptorchidism in male dogs results in azoospermia and consequent infertility, whereas unilateral cryptorchidism typically does not impair reproductive function (Meyers-Wallen *et al*., 1991) [67]. In large breeds, significant sexual dimorphism can lead to mechanical difficulties during copulation, particularly when the female is unable to support the male's weight during mating.

Male infertility may also result from acquired anatomical abnormalities such as spermatocele, sperm granulomas, stenosis or obstruction of the genital ducts, as well as inguinal or scrotal hernias. These conditions can lead to azoospermia or aspermia, thereby impairing fertility (Mialot *et al*., 1985) [68].

Epididymal, testicular and urinary problems

Spermatozoa are produced in the testicles and gain motility and fertilizing capacity during their passage through the

epididymis. Therefore, any pathological condition affecting these organs can result in infertility (Farstad *et al*., 1989) [35]. Additionally, conditions such as cystitis or urethritis may impair sperm motility by altering the pH of the urethral environment. Similarly, diet-induced alkalinization of urine can have a comparable negative effect on sperm function (Mir *et al*., 2013) [69].

Retrograde ejaculation

Retrograde ejaculation refers to the backward flow of semen into the urinary bladder during ejaculation, which can result in aspermia or oligospermia. The hypogastric nerve facilitates bladder neck closure during ejaculation; however, a small volume of sperm typically enters the bladder (Moor *et al*., 1969) [70]. Fertility may be compromised when this retrograde flow becomes excessive. Contributing factors include bladder fullness at the time of ejaculation, as well as conditions such as urethral calculi, cystitis, and urethral strictures following surgical procedures (Nagashima *et al*., 2015) [74].

Prostatic problems

Prostatitis is a significant contributor to infertility in male dogs, as it reduces ejaculate volume and adversely affects sperm motility. This condition often alters the pH of prostatic fluid (Farstad *et al*., 1989) [35], thereby hindering the free movement of spermatozoa. Infectious agents responsible for prostatitis may exert direct cytotoxic effects on sperm cells, leading to their destruction in situ, or may impair their progression through the female reproductive tract due to the presence of pyospermia or hematospermia (Farstad *et al*., 1989) [35].

Hormonal problems

Disruptions in the hypothalamic-pituitary axis can adversely impact spermatogenesis and male fertility, with effects ranging from transient to severe (Nomura *et al*., 1990) [76]. A decline in semen quality typically becomes evident over several weeks to months, during which time matings are unlikely to result in pregnancy (Durrant *et al*., 1998) [31]. If left unaddressed, the deterioration in semen quality may progress to complete azoospermia, rendering infertility irreversible.

Hypopituitarism is a recognized cause of azoospermia and can contribute to infertility in male dogs. Tumors affecting the hypothalamus or pituitary gland may similarly disrupt reproductive function (Panciera *et al*., 2012) [77]. Prolactin-secreting adenomas have also been implicated in negatively affecting fertility. Furthermore, idiopathic insufficiency characterized by deficient secretion of gonadotropins namely follicle-stimulating hormone (FSH) or luteinizing hormone (LH) can impair spermatogenesis (Durrant *et al*., 1998) [31].

Testicular tumors, particularly those arising from Sertoli or Leydig cells, can impair spermatogenesis through excessive hormone secretion, even when small and confined to a single testis. These tumors negatively impact fertility by directly damaging testicular tissue, triggering local inflammation, increasing intra-scrotal temperature, and producing elevated levels of estrogens or androgens that disrupt the hypothalamic-pituitary axis via negative feedback mechanisms (Durrant *et al*., 1998) [31]. Additionally, endocrine disorders such as hypothyroidism and adrenal gland dysfunction are also recognized as potential contributors to infertility.

Infectious diseases

Infectious conditions represent a significant cause of infertility in male dogs, particularly in breeding kennels. Infections such as orchitis or epididymitis can compromise semen quality and thereby reduce fertility. Although there is no conclusive evidence that viral infections directly cause male infertility (Farstad *et al*., 1989) [35], infectious agents may be present in seminal fluid and transmitted to bitches during mating, potentially resulting in infertility in the female. Canine brucellosis is a major infectious cause of infertility, leading to a rapid deterioration in semen quality and resulting in both acute and chronic orchiepididymitis (Randal *et al*., 1990) [78]. Additionally, *Mycoplasma* and *Ureaplasma* species have been isolated from the preputial and urethral regions of infertile male dogs (Farstad *et al*., 1989) [35].

Fungal infections have been implicated as potential contributors to reproductive disorders in male dogs. *Blastomyces dermatitidis* has been identified in a documented case of orchitis and in multiple cases of balanoposthitis (Randal *et al*., 1990) [78].

Genetic problems

Chromosomal abnormalities can contribute to infertility even in phenotypically normal male dogs. One such genetic condition is Kartagener's syndrome, which is characterized by a combination of chronic respiratory tract disease, male infertility, and hydrocephalus (Reynaud and Fontbonne, 2005; Rhoades *et al*., 1977) [79, 80].

Drugs and Infertility

Steroid hormones including corticosteroids, androgenic or anti-androgenic agents, and estrogens as well as certain pharmaceutical drugs such as antineoplastic agents, cimetidine, and the tricyclic antidepressant amitriptyline, may disrupt the central regulation of spermatogenesis or interfere with sperm maturation in the epididymis. These effects can contribute to a progressive decline in male fertility (Durrant *et al*., 1998) [31].

Abnormal sexual behavior

In male dogs exhibiting reduced libido, distinguishing between organic and psychological causes can be challenging. Interestingly, the same underlying condition such as poor semen quality leading to infertility despite normal sexual behavior may, in some instances, also impair Leydig cell function, resulting in decreased libido (Reynaud *et al*., 2005) [79].

Miscellaneous causes

Excessive mating frequency in male dogs can lead to a reduction in libido, while extended periods of sexual inactivity particularly in giant breeds may result in diminished semen quality (Robinson *et al*., 1996) [81]. The initial ejaculate following prolonged sexual rest often contains a high proportion of aged and non-viable spermatozoa that have accumulated in the epididymis (Reynaud *et al*., 2005) [79]. Similarly, obesity, especially due to excessive peri-scrotal fat, can negatively affect semen quality in a comparable manner.

Physical trauma such as dog bites, lacerations, kicks, or blunt force to the testes can compromise the blood-testis barrier, potentially triggering autoimmune spermatogenic arrest through the formation of anti-sperm antibodies (Rhoades *et al*., 1977) [80]. Similar immunological disruption occurs in cases of brucellosis, often resulting in sperm agglutination (Farstad *et al*., 1989) [35]. Additionally, fucosidosis, a congenital lysosomal storage disorder, affects the function of epididymal epithelial cells and leads to the retention of cytoplasmic droplets, a condition that has been

reported in dogs (Durrant *et al*., 1998) [31]. Idiopathic testicular degeneration is another prevalent cause of infertility in dogs, typically associated with azoospermia (Reynaud *et al*., 2005) [79].

Effects of nutrition on reproduction

The relationship between nutrition and reproductive function plays a critical role in determining reproductive efficiency (Root Kustritz *et al*., 2005) [82]. Undernutrition can lead to reduced body weight and poor body condition, delayed onset of puberty, prolonged postpartum intervals before conception, and disruption of normal ovarian cyclicity due to suppressed gonadotropin secretion—all of which contribute to increased rates of infertility (Root *et al*., 1995) [83].

Deficiencies in energy, protein, fats, vitamins, as well as micro- and macro-minerals are closely linked to impaired reproductive performance. Among these, energy balance is considered the most critical nutritional factor influencing reproductive dysfunction in animals (Schweigert *et al*., 1988) [84].

The influence of dietary protein on reproductive function is multifaceted (Seagerson *et al*., 1982) [85]. Prolonged protein deficiency has been shown to negatively impact reproductive efficiency. Conversely, excessive protein intake—beyond the animal's physiological requirements—can also impair reproductive performance, as observed in cattle (Segalini and Hericher, 2009; Sengupta *et al*., 2019) [86, 87].

Fatty acids and cholesterol serve as essential precursors for the synthesis of reproductive hormones. Increasing dietary fat intake has been associated with elevated levels of hormones such as progesterone and prostaglandins, and fats may exert direct effects on the reproductive axis. These effects can be independent of, or additive to, the benefits of enhanced energy intake. Elevated progesterone concentrations during the luteal phase are generally linked to improved fertility outcomes, while higher dietary fat levels have been shown to promote follicular development (Smith *et al*., 2019) [89]. Such changes in hormone production and follicular dynamics may contribute to enhanced reproductive performance (Smith *et al*., 2006) [90].

Vitamins play vital roles in numerous physiological processes, including reproductive function. Among them, vitamin E is particularly important due to its role as an intracellular antioxidant. It neutralizes reactive oxygen species and lipid hydroperoxides by converting them into non-reactive forms, thereby preserving the integrity of membrane phospholipids and protecting cells from oxidative damage and lipid peroxidation (Sontas *et al*., 2014) [92].

In conditions of vitamin E and selenium deficiency, reactive oxygen species accumulate, leading to damage of cellular membranes and disruption of several critical processes involved in reproductive function. These include the synthesis of steroids (Sonta *et al*., 2009) [93], prostaglandins (Stoecker *et al*., 1990) [95], sperm motility, and embryonic development (Surai *et al*., 1999) [97]. Consequently, deficiencies in vitamin E and selenium have been shown to negatively affect various reproductive parameters such as ovulation rate (Talavera *et al*., 1985) [99], uterine motility, sperm motility and transport (Tsutsui *et al*., 1989) [102], conception rate, postpartum recovery, fetal membrane expulsion (Wichtell *et al*., 1996) [103], embryo viability, milk production, and postnatal growth (Wilborn *et al*., 2012) [104].

Minerals are essential for numerous physiological functions in animals, including reproductive processes (Sengupta *et al*., 2019) [87]. Deficiencies or imbalances in mineral intake are frequently associated with reduced reproductive performance. While it is well-established that adequate mineral levels are necessary, the effects of marginal deficiencies or subtle imbalances remain poorly understood. Similarly, excessive mineral intake may have detrimental consequences on reproductive health (Xu and Feng, 2017) [106].

Conclusion

Breeding failure is frequently observed in dogs and can result from a wide range of infectious or non-infectious causes. Accurate diagnosis necessitates comprehensive evaluation, including physical examination and appropriate laboratory investigations. The relationship between nutrition and reproductive performance is well-established, with significant implications for fertility outcomes. Inadequate intake of energy, protein, fats, vitamins, and both micro- and macro-minerals is consistently linked to suboptimal reproductive efficiency.

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