



**Review Article**

**Cryptorchidism in Mammals-A Review**

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**ABSTRACT**

Cryptorchidism is seen more often in horses, cats, dogs, and pigs and rare in ruminant. It is believed to result from heredity, an autosomal recessive mode of inheritance or a dominant gene with the incomplete penetrance. Ruling out castration as well as conducting physical examinations can easily diagnose cryptorchidism. It can be controlled by complete castration or culling of the affected animal and where appropriate, the animal be used as food animal other than for breeding purposes.

Key words: cryptorchidism, mammals, ruminant.

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**INTRODUCTION**

The term cryptorchidism is a combination of three Greek words, “kriptós” (hidden, occult), “orchis” (testicle) and “idion” (small, diminutive) (Ferreira, 1986). Cryptorchidism is failure of one or both testes to be positioned in the scrotum at the normal time for a species. Typically, it is detected at birth or shortly thereafter (Amann and Veeramachaneni, 2006). De Graaf described cryptorchidism as a problem to humans, dogs and rams as far back as 1668, of which he also quoted previous publications of 1582 and 1618 (Jocelyn and Setchell, 1972). Until recently, the general perception was that cryptorchidism was a single disease with moderate heritability, incomplete penetrance, expressed only in males (sex-specific expression), and concentrated by inbreeding or minimized by culling affected males and all siblings (Amann and Veeramachaneni, 2006).

Based on recent evidence however, cryptorchidism is viewed as a multifactorial disease rather than a single disease (Hutson *et al.*, 1997, Klonisch *et al.*, 2004, Hutson and Hasthorpe, 2005; Amann and Veeramachaneni, 2007).

### **Prevalence of Cryptorchidism in Mammals**

Genera within mammalian families show variations in testes position. Mamoulakis *et al.*, (2012) classified testes position in mammals as transabdominal and inguino-scrotal. He further said that each category can be further subdivided into three groups: position 1: embryonic (no descent from the ancestral/embryonic position), position 2: intermediate (partially descended, intra-abdominal position), position 3: internal inguinal and position 4: emergent (some part protrudes through the inguinal ring), position 5: beyond the inguinal ring but not within a true scrotum), and position 6: scrotal (into a true scrotum). Nonmammalian species such as fish, birds, turtles, crocodiles have their testes located at position 1.

Cryptorchidism in mammals, results when the testis is retained in any of these locations apart from the scrotum (Dunbar *et al.*, 1996). Cryptorchidism can be either unilateral or bilateral (Colville and Joanna, 2002), and is usually seen unilaterally (the left testis is retained more often than the right one) (Ladds, 1993; Hafez, 1995; Nascimento and Santos 1997; Barth, 2006; Ayodeji and Suwaiba, 2013). Among unilateral cryptorchid animals, the right testicle is retained in the abdomen in approximately 80% to 90% of affected animals (Ott and Memon, 1980; Mialot, 1988; Smith *et al.*, 2007). However, some studies have shown that left and right testicular retention are almost equal in occurrence (Nelson and Couto, 1994; Acland, 2001). Cryptorchidism is seen more often in horses, cats, dogs, and pigs compared to other species (Ladds, 1993). It is rare in ruminants (Arthur *et al.*, 1983; Foster, 2007; Edmondson *et al.*, 2012), therefore has low prevalence among the genital abnormalities of goats (Tarigan *et al.*, 1990).

The prevalence of cryptorchidism in animals was reported as 0.1-12% (Amann and Veeramachaneni, 2007), while the prevalence of the condition in feral goats in Australia and native goats in Iran were 1.1% and 2.9%, respectively (Tarigan *et al.*, 1990; Kafi *et al.*, 2007). In South-eastern Nigeria, unilateral cryptorchidism was reported in West African Dwarf (WAD) goats (Ezeasor, 1985; Emehelu *et al.*, 2005; Wekhe *et al.*, 2006) with a high prevalence of 70% in an area where persistent breeding with cryptorchid bucks occurred (Emehelu *et al.* 2005). This led Ezeasor (1985) to report that unilateral cryptorchidism is a common condition among West African Dwarf (WAD) goats of south-eastern Nigeria with the right testes being consistently retained and abdominal in position. In semi-arid zone of North-western, Nigeria, Ayodeji and Suwaiba (2013) recorded a prevalence of 1.74% in bulls similar to the report of Kumi-Diaka *et al.* (1989) but contrary to the reports of Amann and Veeramachaneni (2007), who recently reviewed cryptorchidism in animals and reported prevalence rates of less than 0.5% in bulls and 0.1-0.7% in sheep.

### **Causes of Cryptorchidism in Mammals**

The exact aetiology of cryptorchidism remains obscured due to a relative lack of knowledge on the precise molecular mechanism(s) governing testicular descent (Mamoulakis *et al.*, 2012). Generally in animals, the causes of cryptorchidism have been attributed to gene, anatomy or endocrine system (Mattos *et al.*, 2000). Little is known about the pathogenesis of cryptorchidism in bulls, although it is believed to be hereditary, while in rams, it is most likely to be as a result of an autosomal recessive mode of inheritance, but it may also be due to a dominant gene with the incomplete penetrance (Foster and Ladds, 2006). For endocrine cause, it is believed to be as a result of abnormal production of testosterone or absence of Müllerian inhibiting hormone since normal testicular descent requires both for it to occur (Özygit, 2007).

In some species of animals, other factors have been incriminated. These factors include prolonged breech labour (Depue, 1984), navel infections during testicular descent

(Romagnoli, 1991), exposure of the fetus to an increased maternal oestrogen concentration (Depue *et al.*, 1983), or antiandrogenic chemicals (Hutson *et al.*, 1994) or maternal vitamin A deficiency during fetal development (Wilson *et al.*, 1953).

### **Consequences of Cryptorchidism**

The difference in temperature between the body cavity and the scrotum can amount to 2-6°C, depending on ambient temperature and the species under consideration (Carrick and Setchell, 1977). The lower temperature of the scrotum relative to that of the body cavity is assumed to be critical for mammals, since surgical transplantation of the testis to the abdomen results in a complete inhibition of spermatogenesis in all scrotal species studied so far (VanDemark and Free, 1970; Harrison, 1975; Setchell, 1978; Kandeel and Swerdloff, 1988; Abney and Keel, 1989; Berg, 1989), except in a tropical rodent (Bronson and Heideman, 1993). This is because, a slight increase in temperature for a short time or experimental cryptorchidism, which is more convenient for longer periods, results in a rapid loss of mature germ cells (Henriksen *et al.*, 1995; Setchell and Mieusset, 1996; Sailer *et al.*, 1997).

Pinart *et al.*, (1997) reported that unilateral abdominal cryptorchidism in male goats can cause disturbances at the end of spermatazoa maturation during spermiogenesis of the normal testis, but not in the epididymal maturation process. Also, spermatogenic function in the contralateral testis in the scrotum of unilaterally cryptorchid humans (Kohdaira, 1976; Schenck and Neumann, 1977) and dogs (Shirai *et al.*, 1966; Mengel and Moritz, 1976; Kawakami *et al.*, 1988) has been reported to be inferior in function to the normal males.

Males and females carry the gene and can transmit the pathology to their offspring (Nelson and Couto, 1994; Mickelsen and Memon, 1995; Nascimento and Santos, 1997). So it is important to realize that normal animals can transmit the pathology due to its recessive character (Mickelsen and Memon, 1995).

Neoplasia, in the ectopic testis are more frequent than in the eutopic, being Sertoli cell tumors and seminomas mainly, and they generally appear in animals of six to ten years of age (Romagnoli, 1991; Niemand and Suter, 1992).

When cryptorchidism is bilateral, the animal is sterile (Burke, 1986; Mialot, 1988; Daels *et al.*, 1991; Romagnoli, 1991; Nelson and Couto, 1994; Allen, 1995; Hafez, 1995; Mickelsen and Memon, 1995; Nascimento and Santos, 1997).

### **Histomorphology of the Cryptorchid Testes**

The gross and microscopic appearance is related to its location and the age of the affected animal (Ozygit, 2007). Grossly, Acland (1998) reported that the cryptorchid testis is normal before puberty; after sexual maturation, the testis becomes progressively smaller and fibrotic. According to Cox (1986) and Igbokwe *et al.* (2009), cryptorchid testicles are smaller and soft to touch with smoother or more pitted surfaces compared to the scrotal testicles. As for the compensatory hypertrophy of the contralateral testes of the unilateral cryptorchid testes, there are conflicting reports with some supporting the compensatory hypertrophy (Ladds, 1993) and some with no increase in the size (Igbokwe *et al.*, 2009).

Microscopically, tubular hypoplasia is seen in the parenchyma of cryptorchid testis, no tunica vaginalis and its capsule (tunica albuginea) may be thicker than the scrotal testicles (Ozygit, 2007). The seminiferous tubules of the cryptorchid testis are hypoplastic with poorly differentiated germinal epithelium, with or without central hollow and in some testes; the tubules are separated by expanded areas of connective tissues (Igbokwe *et al.*, 2009). Cox (1986) described only one single layer of spermatogonia, primary spermatocytes and Sertoli cells, with the Sertoli cells being characterized by increased Golgi's complex and smooth endoplasmic reticulum (site of steroidal synthesis) and decreased number of lysosomes.

### **Is the effect of Cryptorchidism Reversible?**

Cryptorchidism induced in the immature rat before sexual maturation results in a significant impairment in all aspects of testicular function, including spermatogenesis, sertoli cell and Leydig cell function (Jegou *et al.*, 1984). However, orchidopexy prior to sexual maturation has been found to restore spermatogenesis (Karpe *et al.*, 1981; Jegou *et al.*, 1984). This seems likely to result from sparing spermatogonia from damage in the immature cryptorchid testes (Moore, 1924; Jegou *et al.*, 1984). Even in humans, where the testicle is surgically moved and placed in the scrotum at a young age, the spermatogenesis returns to normal, because the spermatogonia are still normal (Junqueira and Carneiro, 1990).

The result of studies from a number of reports have demonstrated that following the induction of cryptorchidism in adult rats, Sertoli cell and Leydig cell function is impaired in addition to interference with spermatogenesis (Gupta *et al.*, 1975; Rager *et al.*, 1975; Hagenas and Ritzen, 1976; Kerr *et al.*, 1979a,b; Keel and Abney, 1980; Risbridger *et al.*, 1981a,b; Jegou *et al.*, 1983a ). In the adult, the spermatogenesis failed to recover following periods as short as 10 days of cryptorchidism (Hayashi and Cedenho, 1980; Jegou *et al.*, 1983b). Surprisingly, in 130-day cryptorchid rat, preservation of spermatogonia was considerably greater than when cryptorchidism was induced in the adult state (Jegou *et al.*, 1984).

### **Diagnosis**

The first step is to inspect the scrotum after obtaining a castration history. The scrotum and inguinal region should be examined for the presence of an incisional scar which indicates that castration is attempted. This is then followed by palpation. In unilateral cryptorchid animals, digital palpation of the descended testis and associated spermatic cord can indicate whether the left or right testis is retained. Since the palpation of the testis, that lies inside inguinal canal is often difficult (Rodgerson and Hanson, 1997), then a pressure exercised on the abdomen may allow the testicle to appear if it is in the inguinal canal (Mialot, 1988).

The use of ultrasonographic examination can also be of help in reaching a diagnosis. Its use through the caudal portion of the abdomen per rectum via a 5-MHz probe has been found useful in identifying an abdominal testis in horses, since abdominal testes are less echogenic than scrotal testes (Jann and Rains, 1990).

Other several methods employed in man for diagnosis of the cryptorchid testicles like hormonal evaluation, pneumoperitoneography, herniography, venography, arteriography, computerized tomography , magnetic resonance imaging, laparotomy and laparoscopy (Castilho, 1990) , may also be advocated for, if the cost implication of a method to the value of a cryptorchid animal in question warrants such an extra step.

### **Control**

The best choice of control is to perform complete castration of the cryptorchid animal (Mattos *et al.*, 2000) and retain as food animal if appropriate and not for breeding purposes. Despite the lack of a strong basis for the claim that cryptorchidism is a heritable condition (Amann and Veeramachaneni, 2007), there is no doubt that in some species like dogs and pigs, brother to sister mating over several generations increases incidence of cryptorchidism (Cox *et al.*, 1978, Mikami and Fredeen 1979, McPhee and Buckley 1984). So, if due to one reason or the other, the surgery could not be performed then, it is recommended to cull at least the cryptorchid male.

## **CONCLUSION**

Cryptorchidism poses an economic problem especially in breeding industries. Thus, when encountered, strict control measures outlined above should be in place as it is believed to be hereditary.

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