

Cytopathology-2015: Reduced fertility in aging roosters due to retained spermatozoa in Sertoli cells - Avi Rosenstrauch - Achva Academic College, Israel.

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After artificial insemination, the fertility of the rooster peaks at 96% at 32 weeks, decreases to 75% at 70 weeks and only 20% at 110 weeks. In previous studies, we found that the reduction in fertility was concomitant with a decrease in the concentration of ejaculated sperm and the plasma concentration of testosterone. The present study examined: the development of spermatogenic cells and their relationship to the Sertoli cells that support them in the testicular seminiferous tubules; and the functioning of the Leydig cells that produce and secrete testosterone. Roosters aged 32, 70 and 110 weeks were compared using light and electron microscopy. We found that the decrease in the plasma testosterone level in aging and fertile cocks was characterized by a decrease in Leydig cells: number and volume of mitochondria, where the biosynthesis of testosterone is triggered by the cleavage of cholesterol; rough endoplasmic reticulum involved in protein synthesis and smooth endoplasmic reticulum involved in testosterone secretion. However, the spermatogenesis remained normal and the cells showed a regular ultra-structure. The reduced production of testicular sperm was caused by their retention by Sertoli cells in the seminiferous tubules. These Sertoli cells have lost their actin filaments which participate in the spermiation of the fertile rooster. We concluded that the low fertility of aging roosters was linked to the reduction in testosterone levels, which resulted in impaired spermiation due to a deficiency of actin filaments in Sertoli cells.

A Sertoli cell (a kind of sustentacular cell) is a "nurse" cell of the testes which is fragment of a seminiferous tubule and aids in the process of spermatogenesis, the fabrication of sperm.

Development

Sertoli cells are necessary for male sexual development. During male development, the SRY gene activates SOX9, which then activates and forms a direct-acting

loop with FGF9. The proliferation and differentiation of Sertoli cells are mainly activated by FGF9. The absence of FGF9 tends to develop a female. Once fully differentiated, the Sertoli cell has been considered terminally differentiated and is unable to proliferate. Consequently, once spermatogenesis has started, no more Sertoli cells are created. Recently however, some scientists have found a way to induce Sertoli cells to have a juvenile proliferative phenotype outside the body. This gives rise to the possibility of repairing certain defects that cause male infertility. It has been suggested that Sertoli cells may come from the fetal mesonephros. It is activated by follicle stimulating hormone (FSH) secreted by the adenohypophysis and has an FSH receptor on its membranes. It is precisely positioned in the convoluted seminiferous tubules (since it is the only domicile in the testes where sperm are produced). The development of Sertoli cells is directed by the factor-determining protein of the test

Several possible pathways may be involved in the pathogenesis of this condition. The researchers hypothesized that the congenital absence of germ cells can result from a correct failure of the migration of the gonocytes. The Yq11 County is also acknowledged as the azoospermia factor region (AZF region). Microdeletions in the Yq11 region of the Y chromosome, particularly in the AZFb / b + c region, have been found in some patients with SCO syndrome and are another possible cause of the disease. Deletions between palindromes P5 and P1 and between P4 and P1 have also been reported. Yang et al. also hypothesized that two deletions in the interval region between P4 and P3 could also be involved in the pathophysiology of the condition. Apoptotic elimination and altered maturation of germ cells by mutations of Fas, FasL and active caspase-3 have also been reported recently in the literature. Patients have normal levels of luteinizing hormone (LH) and testosterone. The low level of inhibin-B leads to a higher than normal level of follicle

stimulating hormone (FSH). A study by Stouffs et al. have shown that in white men with SCO syndrome, karyotype abnormalities, such as Klinefelter syndrome, are the most common abnormality observed. The pathophysiology of the disease can be summarized as follows:

- Mostly idiopathic
- Genetics as discussed above
- Exposure to toxins causing a drop in spermatogenesis
- Exposure to radiation from the testicular region
- History of trauma resulting in decreased sperm production
- Viral infection

Structural:

The occlusal junctions of Sertoli cells form the blood-testicular barrier, a structure that separates the interstitial blood compartment of the testicle from the ad luminal compartment of the seminiferous tubules. Due to the apical progression of spermatogonia (sperm stem cells), the occlusal junctions must be dynamically reformed and broken to allow immuno-identical spermatogonia to cross the blood-testicular barrier in order to become immunologically unique. Sertoli cells control the entry and exit of nutrients, hormones and other chemicals into the tubules of the testes and make the ad luminal compartment a preferred immune site.

The cell is also responsible for establishing and maintaining the niche of spermatogonia stem cells, which ensures the renewal of stem cells and the differentiation of spermatogonies into mature germs which progress gradually in the long process of spermatogenesis, ending with the release of sperm according to a process known as spermiation. Sertoli cells bind to spermatogonia cells via N-cadherin and galactosyltransferase (via carbohydrate residues).

Immunomodulatory properties of Sertoli cells:

In addition to the expression of factors crucial for the maturation of spermatozoa, Sertoli cells produce a wide range of molecules (either on their surface or soluble) capable of modifying the immune system (IS). The

ability of Sertoli cells to modify the immune response in the tubule is necessary for successful maturation of sperm. Sperms express neoepitopes on their surface as they progress through different stages of maturation. They can trigger a strong immune response if they are placed elsewhere in the body.

History

Sertoli cells are so termed because of their namesake Enrico Sertoli, an Italian physiologist who discovered them while studying medicine at the University of Pavia in Italy.

He issued a description of this cell in 1865. The cell was exposed by Sertoli with a Bethel microscope purchased in 1862, which he used during his medical studies.

In the 1865 publication, his primary description used the terms "tree-like cell" or "stringy cell" and most importantly he referred to these as "mother cells". It was further scientists who cast-off Enrico's family name, Sertoli, to label these cell in publications, starting in 1888. As of 2006, two textbooks that are enthusiastic specifically to the Sertoli cell have been published.

Differential diagnosis:

It is essential to properly rule out other conditions that may arise with low sperm count. Some of the deviations to consider are:

- Azoospermia
- Leydig cell hyperplasia
- Klinefelter syndrome
- Terminal testis failure
- Hypo spermatogenesis