

### **Uterine defense mechanism:**

Uterine defense mechanisms against contaminant micro-organisms were maintained in several ways:

**Anatomically** : by the simple or pseudostratified columnar epithelium covering the endometrium, uterine muscles, natural barrier (cervix, vestibule and valve)

**Chemically** :by mucus secretions from the endometrial glands and hormones (estrogen).

**Immunologically**: through the action of polymorphonuclear inflammatory cells and humoral antibodies, but the degree of interaction is not clear.

Disruptions of these mechanisms allow opportunist pathogens, mostly microorganisms found in the posterior gastro-intestinal tract and around the perineal area, to colonise the endometrium and cause an endometritis.

Under normal circumstances, there are several mechanisms, which prevent pathogens from colonizing the genital tract.

The major anatomical barriers between the contaminated world and the relatively sterile environment of the uterus, include the vulva, the vestibule (guarded by a muscular sphincter), and the cervix.

It should be noted that, although the vulva may appear of little consequences as a barrier, it is, in fact, remarkably efficient at preventing faecal contamination of the tubular genitalia. In cattle, the cervix is formidable barrier composed of series of mucosal lined collagenous rings.

In addition, the cervical-vaginal mucus (especially the scant, tenacious mucus of the luteal phase) can function as a physical barrier for organisms that would otherwise ascend the reproductive tract.

The circular and longitudinal layers of the uterine musculature provide physical propulsion of particular material, including microbes.

Epithelial cells are the first to make contact with potential pathogens that enter the uterus. Epithelial and stromal cells interactions are critically important for endometrial function, with stromal cells affecting epithelial cells through both the release of soluble factors and turns over of extra cellular matrix. Conversely, epithelial cells affect

stromal cells function through the release of soluble factors and cell to cell contact PGE<sub>2</sub> regulate epithelial cells proliferation and is mediated indirectly by uterine stroma.

Estradiol and progesterone have both opposing and complementary effects on the female genital tract with estradiol stimulating epithelization (especially of the vaginal lining and endometrial gland), and vascularization of the endometrium, and increased production of cervical mucus and oviductal secretions, enhancement of uterine contractility, initiation of sexual receptivity.

Cattle are resistant to uterine infections when progesterone concentrations are basal and they are susceptible when progesterone concentrations are increased.

The high estradiol concentrations that occur at estrus and parturition cause changes in number and proportions of circulating white blood cells, with a relative neutrophilia and a "shift to the left".

Moreover, at estrus, the blood supply to the uterus is increased under the influence of estradiol, whilst at parturition there is a massive blood supply to the gravid uterus. This increased blood supply, coupled with the migration of white cells from the circulation to the uterine lumen, enables vigorous and active Phagocytosis of bacteria to occur. Estradiol also causes an increase in the quantity and nature of vaginal mucus, which also plays an important role in defense of the uterus against bacteria by providing a protective physical barrier and by flushing and diluting the bacterial contaminants.