ABSTRACT

Inflammation of the reproductive tract of a cow occurs when the physical and functional barriers to contamination are breached or specific infection occurs. Commonly, contamination occurs at parturition and to a lesser extent at estrus. Uterine contamination following calving is common, but most healthy cows are able to clear the uterus of bacteria in the first 2 to 3 wk after calving. Persistent infections are more likely to be caused by Actinomyces pyogenes. Specific venereal infections tend to be more host-adapted and produce a lower grade inflammation. Nonspecific bacterial contamination of the endometrium generally induces a neutrophilic influx into the stratum compactum and uterine lumen. Neutrophils phagocytize bacteria with the aid of opsonins in the uterine fluid. Mast cells and eosinophils may also contribute to the inflammatory reaction, which may damage the surface epithelium and release vasoactive substances that allow leakage of serum antibodies into the uterine secretions. Specific antibodies of immunoglobulin (Ig) isotype A, M, G1, and G2 in uterine secretions have been described. In model species, the immune capability of the uterus is influenced by steroid hormones, especially estradiol, which increases secretory component and both IgA and IgG content in uterine secretions and increases the activity of antigen-presenting cells in the uterus. Similar cyclic fluctuations in immune components have been described for cows, including changes in the population of subsurface cytotoxic and helper T cells and changes in the expression of major histocompatibility II antigen on surface cells.

INTRODUCTION

Inflammation is the specific or nonspecific immune response of higher organisms to tissue injury or to the invasion of that tissue by foreign or perceived-as-foreign organisms (59). When it occurs in the uterus of the dairy cow, the clinical consequences include a reduction in fertility as measured by calving-to-conception intervals, first service conception rates, and other performance indices (7, 9, 28, 40, 54, 70). A generally conventional series of cellular and chemical responses constitute the inflammatory reaction. And, for the enormous surface area that is exposed to environmental flora at the mucosal surfaces of the respiratory tract, gastrointestinal tract, and to a lesser extent the reproductive tract, an effective system of mucosal defense, including mucosal inflammation, has developed at this interface. Immune protection of the reproductive tract requires most of the same cellular and chemical strategies as do other mucosal surfaces. However, some unique influences of the endocrine environment that mediate reproductive events also significantly affect this mucosal immune system and will be discussed in the context of their influence on protection from microbial attack.

Anatomical and Physical Protective Mechanisms

Effective defense against reproductive tract invasion by environmental organisms is mediated by anatomical and functional barriers as well as nonspecific and specific immune responses. 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. In the cow the cervix is a formidable barrier composed of a series of mucosal-lined collagenous rings. In addition, the cervico-