
It is an oft-stated maxim that pregnancy represents a natural allograft because the conceptus inherits paternal histocompatibility genes that encode proteins recognized as foreign by the dam. Despite this fact, there is little evidence that immunological rejection or dysregulation is an important cause of pregnancy loss or retarded fetal development in livestock. The one exception may be for conceptuses produced by somatic cell nuclear cloning since increased trophoblast expression of major histocompatibility complex class I proteins and accumulation of CD3+ T lymphocytes in endometrium have been reported for cloned pregnancies. Experiments with rodents and livestock indicate that there are a multitude of mechanisms to prevent immune rejection of the conceptus. Some of these mechanisms are played out at the level of the conceptus (for example, reduced antigenicity of the trophoblast) whereas others involve regulation of maternal immune responses, largely in the endometrium, under the control of maternal or conceptus derived regulatory molecules. There is some evidence in rodents for a temporary maternal anergy toward conceptus antigens. There is also non-specific immunosuppression mediated by regulatory T cells, gamma-delta T cells, M2 macrophages, and the corpus luteum and placenta (via secretion of progesterone). It may be that the prolonged period of local immunosuppression in the endometrium during pregnancy renders the female less able to prevent establishment of microbial infections after parturition. Little is known about the restoration of immunocompetence in the uterus after parturition although some populations of immunoregulatory cells, such as gamma-delta T cells in sheep, are largely cleared in the first week postpartum. Understanding regulation of endometrial immune function during pregnancy and the consequences for postpartum immune function may lead to novel strategies to reduce microbial infections of the uterus during the postpartum period.

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