

Abstract

Pregnancy represents a major challenge to the maternal immune system. From an immunological point of view, a fetus is a semi-allograft. The mechanisms providing immunological paradox of fetal tolerance are still not well known and require further research. A complex network of immuno-endocrine interactions ensures fetal growth and development within the maternal uterus. The hormone playing an indispensable role in pregnancy is progesterone.

The aim of this thesis is to summarize current knowledge of the effects of progesterone on the immune system in pregnancy and its mechanisms. Progesterone can affect target cells via the classical nuclear progesterone receptors, which act as transcription factors, or it can act using a variety of other ways, including non-genomic rapid signaling.

Progesterone optimizes conditions for successful establishment and maintenance of pregnancy, changes the amount, localization and characteristics of immune cells and production of cytokines. It reduces the antigen-presenting capacity of dendritic cells, monocytes, and macrophages, suppresses NK cell cytotoxicity, supports the proliferation of uterine NK and dendritic cells, affects B cells and induces the formation of T regulatory cells and their recruitment into the fetal-maternal interface.

The wide range of immunomodulatory properties of progesterone deserves further investigation. A better understanding of these properties may pave the path to development of appropriate diagnostics and treatment of infertility, miscarriage, preterm delivery, but also some autoimmune diseases and cancers.

Keywords

progesterone, pregnancy, semi-allogeneic fetus, miscarriage, immuno-endocrine interactions, progesterone receptors, regulatory T lymphocytes, macrophages, NK cells, dendritic cells