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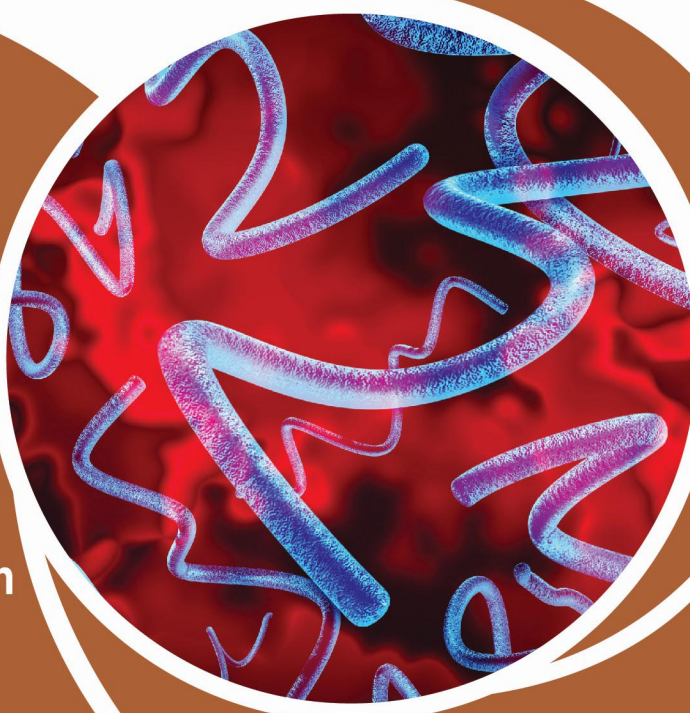
Obs and Gynae PG Focus Series

Parasitic Infections in Pregnancy



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CHAPTER

Immunology of Parasites and Pregnancy

INTRODUCTION

The immune response to parasitic infection leads to a complex interplay with the immune changes of pregnancy. CD4-T cells can differentiate into one of two types of T-helper cells, a T-helper cell type 1 (Th1) or a T-helper cell type 2 (Th2) cell. Th1 cells activate a cytotoxic response through cytokines, including interferon-gamma (IFN-γ), tumor necrosis factor-alpha (TNF-α), and interleukin (IL)-12.[1] The main role of Th2 cells is to activate the humoral immune system as well as to secrete IL-4 and IL-5. IL-4 stimulates IgE production and IL-5 signals eosinophils **(Table 1)**. In pregnancy, the Th1/cytotoxic response is downregulated; whereas, the Th2 response predominates. Classically, immunoglobulin E (IgE) and eosinophils, through the Th2 pathway, are involved in the immune response against helminths (parasitic worms); whereas, cytotoxic T cells and macrophages, through the Th1 pathway, are recruited to a greater extent against protozoan infection. Thus, although helminthic infection can be dangerous in pregnancy, the severity of maternal, placental, and fetal infection is not as severe as with protozoan infections, which are generally managed by the cytotoxic immune system **(Table 1)**.

The maternal immune system may tolerate fetal antigens by suppressing cell-mediated immunity while retaining normal humoral immunity. These changes are known to occur locally at the maternal–fetal interface but may also affect systemic immune responses to infection. Although pregnant women are not immunosuppressed in the classic sense, immunologic changes in pregnancy may induce a state of increased susceptibility to certain intracellular pathogens, including viruses, intracellular bacteria, and parasites.

TABLE 1: General tenets of immune response to parasitic infections.

Helminthic	*Protozoa*
Th2 cells produce IL-4 and IL-5	Th1 cells produce IFN-γ
IL-4 and IL-5 stimulate the production of IgE	IFN-γ activates macrophages
IgE activates mast cells and eosinophils	Macrophage activation allows the destruction of intracellular parasites

(IFN-γ: interferon-gamma; IgE: immunoglobulin E; IL-4: interleukin 4; Th2: T-helper cell 2)

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MATERNAL–FETAL INTERFACE

The fetal allograft is exposed to the maternal immune system at the placenta and fetal membranes (the amnion and chorion), collectively described as the maternal–fetal interface. On the fetal side of the interface, the placenta and membranes enclose the fetus and are derived entirely from fetal tissue. Forming a specialized epithelial surface within the placenta, fetal syncytiotrophoblast cells directly contact maternal blood for nutrient exchange. On the maternal side of the interface, the uterine tissue in contact with the placenta and fetal membranes, the decidua, is rich in specialized maternal immune cells including lymphocytes and macrophages.[2] Despite the prolonged direct exposure of decidual leukocytes and maternal blood to fetal antigens, the immune system does not recognize the fetus as foreign. Several mechanisms underlie this maternal tolerance of fetal tissues.

HUMORAL IMMUNITY

Humoral immunity is also known as antibody-mediated immunity. Humoral immunity results from the recognition of pathogens by specific antibodies. Most effective against extracellular pathogens, humoral immunity is essential for fighting many bacterial infections. The bacteria become coated in antibodies, which then mediate the uptake of the pathogens by phagocytic cells, including neutrophils and macrophages. The presentation of the bacterial antigens on the surface of the macrophage then stimulates B lymphocytes specific to the pathogen, and the B cells produce more antibodies to control the infection. This humoral immune response is augmented by Th2 lymphocytes, which provide costimulation and induce replication of the B-cells. The Th2 response during pregnancy results in vigorous antibody-mediated immunity to pathogens.[2]

CELL-MEDIATED IMMUNITY

Essential for controlling intracellular pathogens, cell-mediated immunity involves lymphocyte recognition of cell-associated foreign antigens, followed by destruction of the infected host cells. In contrast to humoral immunity, this arm of the immune response is stimulated by T-helper type I (Th1) lymphocytes and the cytokines they release. The most important effectors of the cell-mediated immune response, cytotoxic T lymphocytes, are the main immune cells that recognize foreign antigens on the surface of infected "self" cells. Cells infected with viruses or other intracellular pathogens are cytotoxic T lymphocytes' most common targets. The cell-mediated immune response is critical for controlling such pathogens because their intracellular location shelters them from antibody binding.

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T-HELPER CELLS AND THE TH1-TH2 SHIFT

Emphasis on cell-mediated immunity versus humoral immunity changes according to the type of T-helper lymphocytes responding to an infectious threat. Multiple factors, including the cytokine environment and costimulatory molecules present during activation of the T-helper cell, determine the development of either Th1- or Th2-helper phenotype. One hypothesis is that, in addition to hormonal factors that affect the Th1-Th2 balance, macrophages present at the maternal–fetal interface release predominantly Th2-stimulating cytokines and contribute to the overall dominance of humoral immunity during pregnancy.[3] In addition to stimulating B lymphocytes, Th2 cells suppress the cytotoxic T lymphocyte response, decreasing the robustness of cell-mediated immunity. Recent studies show that progesterone binds to a blocking factor induced by the same hormone (PBF) in T lymphocytes. This binding induces the grouping of JAK homodimers, which triggers the activation of the STAT6 pathway. Once STAT6 is activated, it is translocated to the nucleus, thus promoting transcription of IL-4, IL-10, and TGF-β. These cytokines, in turn, inhibit the Th1 response and promote the Th2 response, a mechanism that prompts the generation of immunoglobulins **(Fig. 1)**. This is the mechanism by which progesterone can inhibit the Th1 type response and exacerbate the Th2 type response, characterized by increased IL-4, IL-5, and IL-10 levels. In the uterine decidua, the Th2 cytokine environment favors the activation of B lymphocytes, resulting in the stimulation of antibody secretion and suppression of cell-mediated immunity.[3] This phenomenon is often referred to as the Th1-Th2 shift of pregnancy and is thought to contribute to maternal tolerance of the fetus by suppressing the antifetal cell-mediated immune response. However, this type of immunity may entail higher susceptibility towards Th1-dependent parasitic infections, like those caused by Leishmania major. These infections not only present an increase in Th2 type immunity but also a decrease in Th1 type immunity. In infections caused by *Neospora caninum* and *Toxoplasma gondii*, the number of parasites correlates with the decrease in IL-2 and INF-γ (Th1 type immunity), while the Th2 type has no effect on infection.

SYSTEMIC IMMUNE CHANGES

An evolving model of pregnancy-associated immune changes suggests that the hormonal environment of pregnancy contributes to local suppression of cell-mediated immunity at the maternal–fetal interface while mediating a systemic change toward Th2 dominance. The fact that the local Th1–Th2 shift may also influence the systemic maternal immune response during pregnancy is evidenced in pregnant patients with autoimmune disorders. Women with rheumatoid arthritis, a predominantly cell-mediated autoimmune disorder, tend to experience remissions during pregnancy.[4]

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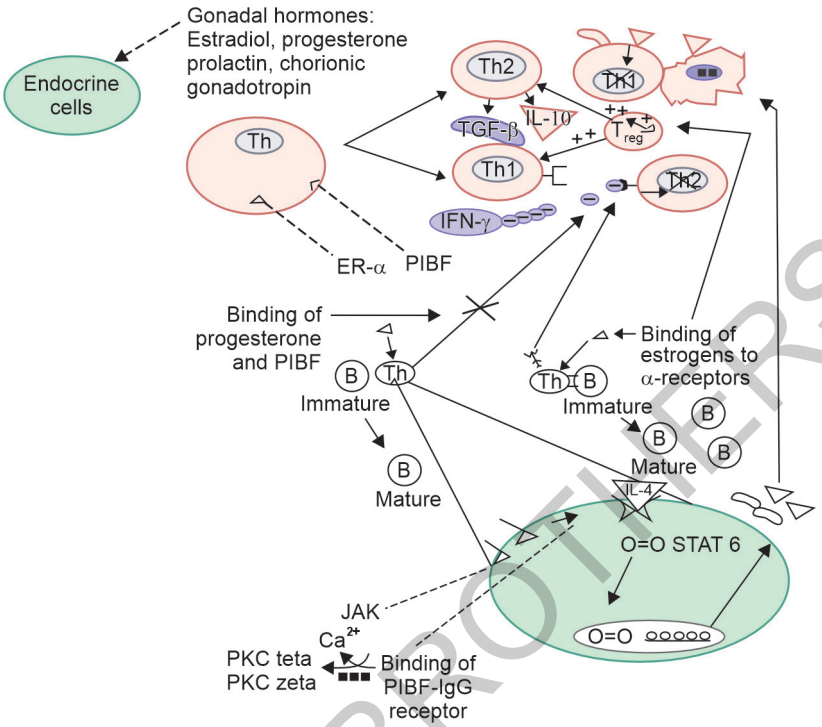

Fig. 1: Cellular communication among the different systems. Mode of interaction of progesterone and estradiol with T lymphocyte through the protein PIBF. Once it binds to the receptor, phosphorylation ensues by which the JAK/STAT6 receptors are activated, inducing transcription of IL-4, IL-10, and TGF-β. These cytokines and T_{reg} production inhibit Th1 and promote the Th2 type response.[7] (IL: interleukin; PIBF: progesterone-induced blocking factor; STAT6: signal transducer and activator of transcription 6; TGF-β: Transforming growth factor beta; Th2: T-helper cell type 2)
Source: Vargas-Villavicencio JA, De León-Nava MA, Morales-Montor J. Immunoendocrine Mechanisms Associated with Resistance or Susceptibility to Parasitic Diseases during Pregnancy. Neuroimmunomodulation. 2009;16(2):114-21.

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Similarly, patients with multiple sclerosis have fewer exacerbations while pregnant but worsening symptoms during the postpartum period.[5] Systemic lupus erythematosus, however, a predominantly antibody-mediated autoimmune disorder, often worsens during pregnancy, perhaps due to increased immunoglobulin synthesis and decreased clearance of immune complexes resulting from robust Th2 activity.[3,6] These well-studied changes in the severity of autoimmune disorders during pregnancy illustrate systemic immune alterations that occur in conjunction with the Th1–Th2 shift. Systemic suppression of cell-mediated immunity may contribute to increased susceptibility to some intracellular pathogens including viruses, bacteria, and parasites during pregnancy.

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