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The Placenta's Innate Defense Against Zika

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Introduction

Zika virus is an arthropod-borne virus that belongs to the Flaviviridae family, genus Flavivirus, and was first isolated in 1947 from the serum of a sentinel Rhesus monkey in Uganda, Africa. Since its discovery, the virus has caused major outbreaks in several countries, and it has been linked to severe complications in pregnant women, neonatal birth defects, and the congenital zika syndrome. Maternal-fetal transmission of ZIKV can occur in all trimesters of pregnancy, and the placenta and its cells play an important role in these cases. The decidua basalis and chorionic villi, maternal-fetal components of the placenta, have a dense immunological infiltrate composed of Hofbauer cells, mastocytes, dendritic cells, and macrophages, which are primary cells of the innate immune response.

Zika fever is caused by an arbovirus that is primarily transmitted by mosquitos of the Aedes genus. The zika virus was discovered in Africa in 1947 in the blood of Rhesus monkeys living in the zika forest, and it was first detected in humans in Asia in 1966, but its potential impact on public health was not recognised until the virus caused outbreaks in the Pacific from 2007 to 2015, when it began to spread across America. ZIKV transmission has been confirmed in 87 countries or territories in the Americas. Due to the virus's introduction into immunologically virgin populations and the widespread presence of vectors, large outbreaks have clearly occurred in many countries and territories [1].

Description

Following the 2015 epidemic and state of emergency, there was an intense search for and better understanding of the role of the placenta in ZIKV infection. Vertical transmission of ZIKV suggests tropism by placental cells. The placenta is defined as a temporary and chimeric organ formed during pregnancy from maternal and foetal tissue, the functions of which are critical for a healthy pregnancy. This organ is in charge of nutrition, gas exchange, and toxic waste removal, as well as providing endocrine and immunological support to the foetus and regulating the physiology of the mother and foetus throughout gestation and delivery. Preeclampsia and foetal growth restriction are examples of gestational complications caused by problems with placental formation that can result in foetal or maternal morbidity and even mortality [2].

A distinct foetal invasion of trophoblasts into the decidua characterises human hemo-monochorionic placentation. The foetal placenta is made up of trophoblasts and extraembryonic mesoderm, which proliferate faster than the embryo after implantation. The chorionic villi form the functional structure of the placenta, which has three layers: the surface layer of syncytiotrophoblasts, the layer of cytotrophoblastic cells, and the mesoderm with the endothelium of the foetal vessels. The placenta's maternal component, the basal decidua, is formed from the endometrium. The vessels in this section of the endometrium supply blood to the intervillar spaces. By the fourth month, the junctional zone has formed, containing trophoblast and decidual cells as well as amorphous material [3].

After this time, most trophoblastic cells degenerate, leaving only the decidual and chorionic plate, as well as the intervillous spaces. The decidua forms several decidual septa that project into the intervillous spaces during the fourth and fifth months. Thus, maternal and foetal blood do not mix, with the exception of capillary wall rupture, which occurs only rarely outside of the delivery situation. The placental membrane or barrier is what separates foetal and maternal blood. The foetal endothelium, the connective tissue in the villous axis with mesenchymal cells and fibroblasts, the cytotrophoblast, and the syncytiotrophoblast ensure it. However, as the placenta matures, this layer becomes thinner in order to facilitate product exchange across the placental membrane while remaining compact enough to prevent many types of infections.

The immune status of a pregnant woman has long been thought to be suppressed, but new research shows that immune responses at the maternal-fetal interface are not simply suppressed, but are highly dynamic. Given that the foetus and placenta are semi-allogeneic grafts that should be rejected by an immunologically competent host, pregnancy is a major immunological paradox. The foetus, on the other hand, is protected from immunological aggression, implying that there are complex adaptations on both sides so that the immune system acts on tolerance rather than rejection. The human placenta is distinguished by the formation of immunoprivileged fetal-maternal interfaces, in which foetal tissues come into direct contact with the maternal immune system.

The decidua basalis contains a rich immunological infiltrate during pregnancy, which affects and can be affected by the dynamics of the maternal-fetal interface. The decidua, which serves as an anchor point for the placenta, and the intervillous space are the two main interfaces. It has already been stated that a pro-inflammatory environment is required prior to implantation and not as a result of successful implantation. Deciduous leukocytes, particularly dendritic and natural killer cells, aid in blastocyst apposition, adhesion, and invasion [4,5].

Conclusion

Understanding host-pathogen relationships in ZIKV infections can be difficult. To effectively control the infection, an early and correct innate immune response is required. Maternal-fetal transmission is well documented in the literature, and several innate immune cells found in the placenta are conducive to ZIKV infection. As a result, Hofbauer cells, decidual macrophages, mastocytes, and dendritic cells may act as viral replication sites, contributing to higher viral loads and transplacental infection. Nonetheless, ZikV can induce a pro-inflammatory environment in placental tissue, activate innate immune cells, and thus overlap the antiviral response. More research is needed to understand the mechanisms of Zika virus immunopathogenesis in pregnant women's placentas and their links to poor neonatal outcomes.

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Conflict of Interest

There are no conflicts of interest by author.

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