The 2013 Cell Biology Symposium, which was the fifth in this series, was focused on the role of the immune system during pregnancy. The symposium was held at the Joint Annual Meeting in Indianapolis, IN, July 8 to 12, 2013.

The symposium was intended to provide an up-to-date look at research related to pregnancy and the immune system. The placenta involves direct contact between fetal and maternal tissues, often termed the fetomaternal interface. Because these tissues are genetically dissimilar, the placenta has often been likened to an allograft. In other words, the placenta represents a graft between 2 allogenic individuals and, therefore, should invoke an immune response leading to tissue rejection. Exactly how and why the fetus is not rejected, but rather is tolerated by the maternal system, has long been a topic of intense study (Medawar, 1953; Ober, 1998; Petroff, 2011; Spencer et al., 2012). This topic has special relevance to production of farmed animals because fertility and reproductive efficiency are major constraints to efficient animal production (Trenkle and Willham, 1977).

The symposium began with a talk by M. G. Petroff from the University of Kansas Medical Center (Kansas City, KS) titled, “Tolerance of the maternal immune system to the fetal semi-allograft” (Petroff et al., 2013). Petroff provided a summary of research since Sir Peter Medawar first proposed (Medawar, 1953) that the immunological interaction between the maternal and fetal systems is suppressed (Veenstra van Nieuwenhoven et al., 2003); this was part of his seminal work on immunological tolerance, for which he was co-recipient of the Nobel Prize in Physiology or Medicine in 1960 (Medawar, 1960). She then described recent work from her laboratory showing that although expression of major histocompatibility complex molecules by the placenta is restricted to primarily immunosuppressive (class 1b) antigens, paternally inherited minor histocompatibility antigens are expressed abundantly by the placenta. Her laboratory has shown that these paternally inherited and placenta-specific antigens enter the maternal system via microvesicles and exosomes shed from the trophoblast. Moreover, although maternal T lymphocytes detect these antigens, the immune response is muted “by placental factors co-expressed with the antigens.”

The second talk, by A. Miyamoto from Obihiro University of Agriculture and Veterinary Medicine (Obihiro, Japan), was titled, “The immune system in corpus luteum formation/angiogenesis/lymphangiogenesis and its role in establishment of pregnancy.” Miyamoto discussed the novel observation that luteal angiogenic factors, such as vascular endothelial growth factors and fibroblast growth factors, may serve as chemoattractants for polymorphonuclear leukocytes (PMN), which are present in greatest number early in luteal development. The PMN, on the other hand, secrete IL-8, which stimulates angiogenesis and lymphangiogenesis in the corpus luteum (CL) as well as migration of neutrophils into the CL. In addition, factors secreted from the pregnant uterus, including interferon tau and PGE2 may stimulate luteal angiogenesis and lymphangiogenesis and also modulate the luteal immune system. This work is summarized in the article by Miyamoto et al. (2014) from this symposium.

The last talk was by M. J. Zhu from Washington State University (Pullman). Zhu discussed the role of maternal obesity in inflammation of the placenta and...