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Changes in leucocytes phenotype and functions in case of preeclampsia

Mikhailova Valentina Anatolevna, Selkov S A and **Sokolov D I**
FSBSI "The Research Institute of Obstetrics, Gynecology and Reproductology named after D.O.Ott", Russia

Preeclampsia is a complication of pregnancy that affects health of woman and fetus. Pathogenesis of preeclampsia is closely connected with inflammation in fetal-maternal interface. The aim of the study was to assess leucocyte phenotype and functions in case of healthy pregnancy and preeclampsia. Ethical Committee of FSBSI (The Research Institute of Obstetrics, Gynecology and Reproductology named after D.O.Ott), Saint-Petersburg, Russia, approved the study. Peripheral blood was obtained from 422 women (healthy non pregnant women, healthy pregnant women (38-39 w.g.) and pregnant women with preeclampsia (38-39 w.g.). Such methods as flow cytometry (BD FacsCanto II, USA), atomic force microscopy (NT-MTD, Russia) and cell cultures (THP-1, Ea.Hy926) were used. We showed that expression of CD11c by NK-cells, CD18 by CD8+T-cells and CD119 by monocytes increased in case of preeclampsia comparing with healthy pregnancy. So does *in vitro* adhesion of lymphocytes and monocytes to intact and TNFα activated endothelium. CD107a expression by NK cells was lower and TRAIL expression was higher in case of preeclampsia comparing with healthy pregnancy. Plasma of peripheral blood of all studied patients contained cellular microparticles (average size: 330 nm). In case of preeclampsia the amount of microparticles with receptors CD45 and CD16 increased comparing with healthy pregnancy. Microparticles from plasma of women with preeclampsia *in vitro* influenced expression of CD181, CD18 and CD54 by monocytic cells (THP-1). Observed changes in adhesion receptors expression, adhesion function of T-cells, NK cells and monocytes, TRAIL expression of NK cells are a result of inflammation in decidua, which can be triggered by cellular microparticles present in plasma.

TLR2 located in macrophage sub cellular compartments negatively regulates TLR4 mediated inflammatory response to internalized bacteria

Ludovica F Butto[1], Silvia Melgar[2], John O'Callaghan[2], Ian B Jeffery[2], Hugh M B Harris[2], John MacSharry[2], Fergus Shanahan[2], Paul W O'Toole[2] and **Ken Nally[2]**
[1]Technische Universität Munchen Weihenstephan, Germany
[2]National University of Ireland, Ireland

Recognition of microbes by toll-like receptors (TLRs) is critical for initiation of appropriate innate and adaptive immune responses. How crosstalk between TLRs situated in various cellular locations contributes to host-microbe dialogue and immunoregulation is presently unclear. Here we report a dual role for TLR2 in regulating the response of macrophages to internalized bacteria. Cell surface TLR2 initiated an inflammatory response while sub cellular TLR2 negatively regulated a TLR4-mediated hyper-inflammatory response that was phagocytosis dependent and driven by a type-I interferon autocrine loop. TLR2 deficient macrophages were hyper responsive to commensal and pathogenic bacteria and developed an M1 like phenotype, which was absent in TLR2/TLR4 deficient macrophages. Our findings identify sub cellular TLR2 as being important for the negative regulation of inappropriate TLR4 mediated inflammatory responses to bacteria.