Editorial

The Bystander Effect: Vertical Transmission of Affections from Mother to Foetus

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A bystander is usually a cell, or an organism affected by a process that was not meant for it. For example, tumor cells expressing the herpes simplex virus thymidine kinase (*HSV-TK*) gene are sensitive to the drug ganciclovir (GCV); such GMC exposed *HSV-TK*-positive cells are also lethal to *HSV-TK*-negative cells as a result of a "bystander effect", mediated in part by an antitumor response through the release of cytokines (Ramesh et al., 1996). Further, important biological effects of ionizing radiation usually arise in cells that in themselves received no radiation exposure. These include radiation-induced genomic instability in which biological effects occur in the progeny of the irradiated cell after many generations of cell division; and radiation-induced bystander effects in which cells that receive no radiation exposure receive damage signals transmitted from neighbouring irradiated cells (Little, 2003). These responses are mediated by regulatory factors and signals that mediate intercellular communication.

Furthermore, autoreactive T cells exist in healthy individuals and represent a potential reservoir of pathogenic effectors which, when stimulated by microbial adjuvant, could trigger an autoimmune disease. Experimental studies have indicated that xenobiotics, well defined from a chemical point of view, could promote the differentiation of autoreactive T cells towards a pathogenic pathway. It is therefore theoretically possible that compounds present in vaccines such as thiomersal or aluminium hydroxyde can trigger autoimmune reactions through bystander effects; the bystander being the autoreactive T cells (Fournie et al., 2001).

The foetus that is usually affected by processes that occur in the mother can be considered a bystander that suffers from a sort of bystander effect. Thus, the transfer of infections like HIV (http://www.who.int/hiv/topics/mtct/en/), *Toxoplasma gondii* (Jéamieson et al., 2009) and others from mother to child, programmes the child to changes that come to play after birth. Since the immunopathological responses of the mother to these affections usually involve the production of many signal molecules, it is possible that the transfer of such signalling molecules to the foetus also affects their responses to their environment after birth. Severity of the disease may be influenced by the trimester in which the infection is acquired by the mother (Dunn et al., 1999; Remington et al., 2005) or other factors like genetic predisposition (Mack et al., 1999).

Recent experiments in rats seem to point to the possibility that eating a high-fat diet in pregnancy may cause changes in the foetal brain that lead to over-eating and obesity after birth. It is suggested that exposure to triglycerides from the mother's diet stimulates production of orexigenic peptides, which stimulate appetite. (www.pronutrition.org/archive/200811/msg00015.php).

In the final analysis, the foetus is a bystander whose growth and development is affected by what infects the mother, and what the mother eats.

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