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Mucosal Immunology: A Short Notes

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Introduction

Neonatal mucosal immunology

The lymphoid tissues of the mucosal surfaces of gastrointestinal (GI), respiratory tracts, etc. on the whole called MALT, in addition to the primary function of preserving sterile the delicate epithelial surfaces in contact with the external environment, should also able to recognize the external antigens and to respond by sensitizing CD4 T cells. If there is a malfunction in normal physiology, the host is at risk of enduring a cell mediated tissue damage [1-4]. Cell-mediated immunity (CMI) resembles a double-edged sword, since many aspects of this cascade imply the immanent risk of being detrimental to the structures to be protected; thus, it is evident the necessity of reducing the immune response impact on mucosal surfaces, by limiting at the least needed for the appointed goal, to eliminate pathogenic antigens [2,4-6]. Although manifold defense barriers both natural and immune (such as sIgA) provide reliable defense lines for the epithelial external surfaces, the high solubility and low MW of many non-pathogenic antigens, either inhalant or dietary, make a frequent event the antigen penetration within epithelial tissues [7].

Foetal mucosal immunology

An array of factors acting during the intrauterine life have been shown as promoting fetal mucosal immunology in children. The *in utero* sensitization, recently amplified by substantial data, has gained a salient place among the prenatal factors. CB T lymphocytes proliferate in the presence of allergens, while testing CB cells with a-lactalbumin, ß-lactoglobulin and bovine seroalbumin, there was a meaningful proliferative response [8]. Both an intrauterine sensitization to foods, and aeroallergens were demonstrated, hence supporting the concept that fetal programming by the mother during the second and early third trimester of pregnancy results in FIS T cell priming. The recent observation of HDM in amniotic fluid collected during amniocentesis at 16 to 17 weeks of gestation and in CB provides evidence that both trans placental and trans amniotic routes of exposure can occur *in utero* [9,10]. Consequently, the hypothesis that FIS T cells are exposed during gestation to maternally derived allergens is supported by a line of evidence. and maternal exposure to allergens may prompt the development of fetal T-specific responses. Therefore, we may propose a timing in the FIS, that is immunologically active before 16 weeks of gestation, as I demonstrated in a study of staminal cells, that were resolutive in a variety of disease [11].

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