HTLV-I Infection of the Placenta -Trophoblast and Macrophage-

Fujino T(1), Ikeda T(2), Takesako S(2), Shiokawa H(2), and Nagata Y(2)
1: School of Allied Medical Sciences, 2: Dep Obst & Gynec Faculty of Medicine, Kagoshima Univ, Kagoshima

We have reported that 22%(2/9) of placentas from Human T-lymphotropic virus type I(HTLV-I)-seropositive pregnant women were infected by HTLV-I(1). However, the incidence of HTLV-I infection to cord blood lymphocytes was reported to be up to 7%(2). Therefore, it is suggested that there are defence mechanisms to prevent transmission of HTLV-I from placenta to fetus. Trophoblast is the cell which is bathed by maternal blood, and macrophage has been thought to play some roles in defense mechanism against infection. In the present study we obtained populations of cells enriched for macrophage and for trophoblast, as well as a population of mixed types of cells from human term placentas, and then compared susceptibility to HTLV-I infection between them.

Term placentas from 3 HTLV-I-seronegative pregnant women were obtained just after delivery. Placentas were digested by collagenase/disperse and DNAse. After Percoll gradient centrifugation the cells at densities between 1.045 and 1.065 g/ml were collected. The collected cells were composed of trophoblast, macrophage, fibroblast, and endothelial cell. They were then subjected to immunomagnetic seperation(3). Trophoblast-enriched cells were obtained by a negative selection method using monoclonal antibodies against HLA class I and II, and macrophage-enriched cells by using monoclonal antibodies against cytokeratin and vimentin. Populations of cells enriched for macrophage, for trophoblast, and of mixed types of cells were incubated with MT-2 cells(HTLV-I-infected cell line)(4) for 4 days. Control cultures were done without MT-2 cells. They were then tested by immunocytochemistry for...
reactivity with GIN-14 (monoclonal antibody against HTLV-I gag proteins) (5).

GIN-14-positive cells were observed in all the three populations of cell cultures, and there was no difference in the incidence of GIN-14-positive cells between the three populations. In the culture of the population of cells enriched for trophoblast, both mononuclear and binuclear cells were reactive with GIN-14. In the culture of macrophage-enriched cells, there was a cell which seemed to have had phagocytosed GIN-14-reactive cells.

In the present study, we confirmed that both trophoblast and macrophage are infected by HTLV-I, and it was suggested that macrophage may play some role in infection and protection of HTLV-I transmission from mother to fetus via placenta. However, it seemed necessary to culture the cells longer after co-culturing with MT-2 cells to detect the difference in the incidence of GIN-14-reactive cells between the cultures.