

## LETTER TO THE EDITORS

## Cell adhesion molecules on large granular lymphocytes and endothelial cells in decidua of early human pregnancy

Sirs: We read with great interest the article by Tortosa et al. (1993) concerning the expression of cell adhesion molecules (CAM) by endothelial cells of early human decidua. Using immunohistochemical techniques, these authors found strong expression of ICAM-1, P-selectin and HLA-DR on decidual endothelial cells, whereas staining of these cells for E-selectin, INCAM-110 and VCAM-1 was weak or absent. Tortosa et al. suggest that this differential expression of CAM by decidual endothelial cells may be responsible for the recruitment of the large and unusual population of lymphoid cells found in the decidua, which is composed mainly of CD56++ large granular lymphocytes (LGL) with an NK cell phenotype (representing more than 70% of the intradecidual lymphoid cells) and T cells. However, decidual lymphocytes were not investigated for the expression of counter-receptors for the endothelial CAM in this study. In recent investigations of the distribution of CAM on decidual LGL and endothelial cells we found a similar pattern of immunoreactivity of decidual endothelial cells for ICAM-1, VCAM-1 and E-selectin (Marzusch et al. 1993; Ruck et al., in press). In addition to the immunohistochemical investigations, we also studied the expression of CAM on decidual and peripheral CD56+ lymphocytes by flow cytometry, which revealed that the  $\alpha$ -chains of the integrin counter-receptors for both ICAM-1 and VCAM-1 –  $\alpha_L$  and  $\alpha_4$ , respectively (Springer 1990) – are found on the majority of decidual and peripheral LGL. As the expression of

ICAM-1 on decidual endothelial cells is much stronger than that of VCAM-1, we propose that ICAM-1- $\alpha_L\beta_2$  interactions represent the more important mechanism of binding to endothelium in the migration of CD56+ LGL from the peripheral blood into the decidua. This hypothesis is supported by the results of in vitro experiments in which the ICAM-1- $\alpha_L\beta_2$  interaction was identified as the main mechanism in the binding of NK cells to endothelium in the absence of stimulation (Allavena et al. 1992). However, ICAM-1 is also expressed by endothelial cells in other tissues (Dustin et al. 1986), for example the human liver, where it is upregulated in various inflammatory disorders (Volpes et al. 1991) without large numbers of LGL being present in the inflammatory infiltrate. It is therefore probable that there are additional local factors in the decidua that operate to modulate the migration of LGL from the blood and/or stimulate in situ proliferation of these cells.

### References

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