Response to Comment on "Endothelial ICAM-1 Protein Induction Is Regulated by Cytosolic Phospholipase A2 via Both NF-κB and CREB Transcription Factors"

Rachel Levy

*J Immunol* 2011; 187:2041; doi: 10.4049/jimmunol.1190046

http://www.jimmunol.org/content/187/5/2041.2
Comment on “Endothelial ICAM-1 Protein Induction Is Regulated by Cytosolic Phospholipase A2α via Both NF-κB and CREB Transcription Factors”

I read with great interest the article by Hadad et al. (1) in the February 1, 2011 issue of The Journal of Immunology. The experimental design is systematic but I, however, would like to comment on one essential point and add a word of caution.

The authors used in different experimental approaches the cell line ECV-304 and HUVEC as the “parent line.” ECV-304 is declared by the authors as a spontaneously transformed cell line derived from the Japanese HUVEC culture.

In light of current knowledge, ECV-304 is no longer considered to be of endothelial origin, because it has been confirmed as a derivative of the urinary bladder carcinoma cell line T-24 (2, 3). This information also appears in the Web sites of different cell-line repositories, such as the American Type Culture Collection, the German Collection of Microorganisms, the Health Protection Agency Culture Collections and Cell Cultures, and the Japanese Collection of Research Biosources.

Cross-contamination of cell lines has emerged as a real problem in biomedical research (4); therefore, caution needs to be exercised by all researchers when selecting cell lines for their experimental design, to avoid misleading data.

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References

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The focus of the criticism by Dr. Rojas is the use of the ECV-304 cell line as a model for human endothelium because of its derivation from the human bladder carcinoma T24 cell line. Our study done in ECV-304 cells was repeated in Primary HUVAC (both commercial and isolated from human umbilical cord) and gave identical results, as presented in our manuscript, including the upregulation of cPLA2α by TNF-α, the inhibition of ICAM-1 upregulation by antisense against cPLA2α and the inhibitor of NADPH oxidase activity (DPI), the inhibition of adhesion to differentiated PLB cells by the presence of antisense against cPLA2α, as well as the time course upregulation of cPLA2α, COX-2, and ICAM-1.

Moreover, the in vitro experiments in the ECV-304 cell line, as well as in Primary HUVAC, matched the results of the in vivo experiments showing that ICAM-1 upregulation is dependent on cPLA2. In the Results section, before the description of the experiments with ECV-304 cells, we gave a detailed explanation for the decision to use these cells: ICAM-1 is the only adhesion molecule expressed in these cells, the cells are unregulated by cytokines, and the cells function to adhere to phagocytic cells.

Thus, even if this cell line consists of human endothelial cells contaminated with T24 human bladder carcinoma cells (as appears in the Web sites of different cell-line repositories), it was a useful tool for our studies.

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www.jimmunol.org/cgi/doi/10.4049/jimmunol.1190045

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