

## Letter to the Editor

# CD30 expression in relation to atopic heredity and allergic disease during childhood

Dear Editor,

In the December issue of *Pediatric Allergy and Immunology*, Stencel-Gabriel et al. describe how surface levels of CD30 on cord blood-derived CD4<sup>+</sup> T cells correlate with atopic heredity and IgE levels in plasma of mothers and newborns (1). CD30 and its ligand (CD30L) were suggested to influence Th1/Th2 responses several years ago (2) and their role in allergic disorders has been implicated [reviewed in Bengtsson (3)]. However, for allergic disorders the role of CD30 is still not clear and the immunological consequences of CD30-CD30L interactions are not yet fully understood. I and/or my former collaborators were studying CD30 and CD30L and their roles in allergic disorders some years ago, and several of our published articles are used as references in this article by Stencel-Gabriel et al. (their reference nos 5, 6, 14, 15, 20).

In their article (1), Stencel-Gabriel et al. repeatedly claim that their findings are similar to the results in one of our previously published articles (4), where we investigated surface expression of CD30 and CD30L in placenta in relation to maternal atopy. When reading the article by Stencel-Gabriel et al., the reader can erroneously get the impression that we studied CD30 expression on CD4<sup>+</sup> T cells and measured mean fluorescence intensity on isolated cells. To avoid confusion in the future, I would like to point out the following regarding some of our previous work:

We investigated the presence of CD30<sup>+</sup> and CD30L<sup>+</sup> cells in human term placenta and also measured soluble CD30 (sCD30) in serum from mothers and from cord blood and related our findings to maternal atopy (4). The numbers of CD30 and CD30L-positive cells in placenta were investigated with immunohistochemistry. Staining intensity was not evaluated. CD30 was expressed on decidual stromal cells only while CD30L was detected on HLA-DR<sup>+</sup>

macrophage-like cells throughout the mesenchymal chorionic villi. We did not investigate the presence of these two molecules on T cells, as T cells are virtually absent from a term placenta. We were not able to report anything regarding the mean fluorescence intensity of the staining, as we performed no flow cytometry-based analyses. We concluded that rather than mirroring maternal atopy, sCD30 in cord blood and placental CD30 expression may reflect a Th2-deviated intra-uterine environment.

Another of our published articles (5, and ref. 14 in Stencel-Gabriel et al.) is further used to back-up their statement 'Thus, the binding of CD30 to the CD30ligand (CD30/CD153) is believed to be important in several immunoregulatory processes (14).' It should be stressed that in this work (5), we did not at all investigate CD30-CD30L interactions or their possible immunoregulatory role. Instead we evaluated the use of sCD30 in cord blood and in peripheral blood during childhood as a predictive marker for atopic disease and/or eczema in pediatric patients. We concluded that sCD30 was an unreliable marker for atopic disease and/or eczema in children (5).

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## References

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