

Letter to the Editor

Are the intracellular cytokine profiles of cord blood T lymphocytes useful for detecting a high risk of asthma in the newborn?

Despite the increasing prevalence of allergic diseases in children and young adults in western countries during recent years, all genetic and epidemiological studies, while suggesting an important role of inheritance from the mother, have failed to identify children's risk factors for the future development of respiratory allergy (1).

Hagendorens and co-workers conducted a prospective study on immunological markers in cord blood for the prediction of allergic diseases in children in a group of 33 healthy, full-term newborn infants, 23 of whom were considered at risk for atopy (2). In the newborns from atopic ($n=23$) and non-atopic ($n=10$) parents, they were unable to demonstrate differences in T-lymphocyte sub-populations and/or in the numbers of interleukin (IL)-2, IL-4, and interferon- γ (IFN- γ)-producing T cells. The authors conclude that at present, no parameter other than atopic status of the child itself can be used to evaluate the predictive power of the potentially new markers for atopy. Unfortunately, they limited their evaluation of cytokine-producing T lymphocytes to IL-2, IL-4, and IFN- γ , and could not provide any follow-up data.

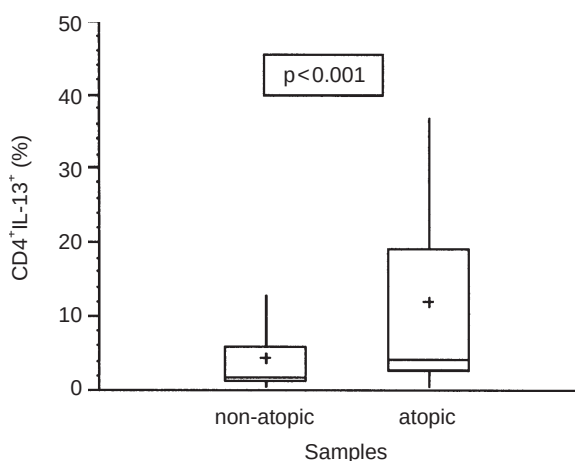


Fig. 1. Double-color cytofluorimetric detection of interleukin-13 (IL-13) on CD4⁺ cord blood T cells. Samples derived from atopic mothers ($n=68$) were compared with those from non-atopic mothers ($n=140$). Box-plots of different values are depicted (Mann-Whitney rank-sum test).

In a recent study (3) on a larger cohort of newborns from atopic ($n=68$) and non-atopic ($n=140$) mothers, we also found no differences in the number of IL-4-producing T cells at birth. However, the number of CD4⁺ IL-13⁺ cord blood T lymphocytes was significantly higher in the group of newborns from atopic mothers (Fig. 1). A greater incidence of wheezing and/or asthma-equivalent symptoms were found in these children at the 1-year follow-up. Furthermore, the development of symptoms positively correlated with the highest percentages of CD4⁺ IL-13⁺ cells at birth (logistic regression chi-square=4.792, $p=0.028$).

Recently, an IL-4-independent pathway of T helper 2 (Th2)-mediated inflammation has been demonstrated in asthma (4). Naive T cells from IL-13-deficient mice show impaired Th2 cytokine secretion when cultured *in vitro* under Th2-biasing conditions (5), and experiments on transgenic mice expressing IL-13 within the lung confirm the role of this cytokine in experimental asthma (6). Our data closely correlate the presence of CD4⁺ IL-13⁺ T lymphocytes at birth and future development of asthma with the atopic status of the mothers. The two human IL-13 receptors (R α 1 and R α 2) are encoded by single genes on chromosome Xq13 (7), and the recently described association between genetic variants of IL-13 R α 1 and atopy (8) provides the first evidence of an X-linked inheritance of atopic disorders.

In our opinion, the intracellular IL-13 profile of cord blood CD4⁺ cells might be a useful tool for a more accurate identification of the newborn at high risk of developing asthma.

References

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- Fabrizio Spinozzi MD
Elisabetta Agea MD
Laboratory of Experimental Immunology and Allergy
Department of Clinical and Experimental Medicine
University of Perugia
Policlinico Monteluce
I-06122 Perugia, Italy
- Fernando M. de Benedictis MD
Pulmonary Unit
Department of Pediatrics
University of Perugia
Policlinico Monteluce
I-06122 Perugia, Italy