

Correspondence

Can natural killer cells represent an early life immune response in development of allergic asthma? More precise data analysis should be considered*To the Editor:*

We read with great interest the study by Altman et al¹ reporting important mechanisms in an early life immune pathway involved in T_H2 polarization leading to the development of allergic asthma in high-risk children.¹ It is now well accepted that natural killer (NK) cells are activated in response to allergens. NK cells may promote allergic sensitization, T_H2 immune response, development of eosinophilic inflammation, and airway hyperresponsiveness.² This study was performed in 3 cohorts by step and shows PBMCs' higher levels of expression of NK cell-related genes in the specific allergic asthma group.¹ We agree with the authors that their results do provide new insight into the early life immune events that lead to T_H2 skewing and disease development allergic asthma. However, we are concerned about some small issues in the data analysis that may have effects on the validity of the results.

First, a general linear model was used with a gamma distribution and log link function to calculate a parameter estimate and *P* value.¹ As is known, the general linear model is a linear model with identity link and responses only normally distributed. The errors are usually assumed to be uncorrelated across measurements, and follow a multivariate normal distribution. If the errors follow a gamma distribution, generalized linear models may be chosen to relax assumptions about *Y* and *U*. Or if it is just misspelled for these 2?

Second, the enrolled participants from the URECA (Urban Environment and Childhood Asthma) study all have a parental history of allergic diseases or asthma and children with maternal asthma were significantly more likely to have asthma at age 7 years (odds ratio, 1.79; 95% CI, 1.18-2.74; *P* = .01).³ And it is shown in the full cohort that there are significant differences between groups in the ratio of participants whose mother has had asthma (*P* < .01).¹ Considering that gene expression may be affected by maternal asthma, we suggest taking this factor as a random effect factor into the simulation linear model analysis.

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