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F. CARSWELL

*Department of Child Health,
Royal Hospital for Sick Children,
St Michael's Hill,
Bristol BS2 8BJ*

Risks of infantile atopic dermatitis from parental atopy

Sirs, I have read with the usual interest the new paper by Ruiz *et al.* [1]. However, there are several points that deserve some remarks.

The authors declare that the studies on the effectiveness of prolonged breastfeeding in the prevention of allergic diseases of children have yielded conflicting results. The allergy-preventive importance of breastfeeding has been clearly assessed since 1936 [2] by both prospective and retrospective studies. My study shows that 81% and 43% of studies, respectively, have confirmed the indisputable advantages of breastfeeding, while the conflicting studies are 21% and 28% respectively (two studies found no differences). Therefore the prospective studies have demonstrated that preventive measures are effective in the very great majority of cases, for example in our studies in 94% of cases [2-10].

As regards the importance of environmental controls, which we proposed and applied together with dietary manipulations since the 2nd International Symposium on Immunological and Clinical Problems of Food Allergy in 1982 [3], the recent study by Arshad *et al.* [11] confirms these measures, demonstrating that they are highly effective in reducing the prevalence of allergic disease: 14% vs 40% according to the employment or not of environmental controls in babies, who were all fed either with breast milk or a soy hydrolysate formula. In our studies the figures are quite similar according to the dietary manipulations (breast or soy milk in the study group, cows' milk in the controls) associated with the environmental controls. The results shows that 51/244 children developed atopic symptoms during the follow-up: 18/100 breast-fed, 8/79 breast- and soy-fed, and 25/65 cows' milk-fed infants. When we consider together the first two groups, only 14.5% (26/179) of the study group compared to the 38.5 (25/65) of the control group developed atopic manifestations during the follow-up [4] ($P \leq 0.0000$), thus confirming that dietary and environmental measures are able to significantly prevent atopy onset in high-risk babies, at least until the age of 3 years and 8 months. Similar results were obtained in a multi-

centre study comprising 2270 infants (77/1201 of the study group vs 48/280 of the controls. $P \leq 0.0000$) [11]. We have already stressed elsewhere [12] that postponing the atopy development leads to a lessening of the severity of the clinical manifestations, and even to atopy avoidance forever. In conclusion the prospective preventive studies have largely confirmed the high value of breastfeeding alone or associated with dietary manipulations, together with environmental controls in the prevention of allergic disease in at risk babies.

Why have Ruiz *et al.* not advised and/or applied to their cohort of babies strict dietary manipulations and environmental measures? In particular in 18/39 children solids were introduced <3 months and in 21/39 >3 months (which month?). Then we read that in 12 babies the number of solids was ≤ 2 and $6 > 2$. It is well known that early weaning significantly affects the prevalence of atopic manifestations. Since 1983 it has been demonstrated that total solid food elimination by six months was necessary for the prophylaxis of atopic disease [13]. Recently a 10-year longitudinal study has confirmed that the introduction of four different solids in the first 4 months of life exposes the child to a risk of recurrent or chronic eczema 2.9 times higher than that of babies not exposed to such a risk. The risk was 5.45 in children not weaned until the 4th month, 7.5% when one to three, and 12.7% when four or more solids were introduced in the first 4 months [14]. Therefore it is quite a consequence that 28% of the 39 infants developed atopic dermatitis [1]. In our study [4] only 8/179 (4.5%) children of the study group, all with an atopic parent, and 10/65 (15.4%) of the controls developed atopic dermatitis ($P = 0.0008$). It seems to me that the difference of preventive measures in at risk babies is striking, especially when a disease such as atopic dermatitis confronts doctors with one of the most demanding challenges [15].

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A. CANTANI
*University of Roma 'La Sapienza',
 Medical School,
 Viale Regina Elena 324,
 00161 Roma,
 Italy*

Paracetamol anaphylaxis

Sirs, In their recent article, Leung *et al.* [1] presented four patients with adverse reactions to paracetamol with

clinical features of immediate hypersensitivity responses. The use of the term 'anaphylaxis' to describe these cases, though, may be misleading since an IgE-mediated immunological mechanism was not proven. The distinction is not just a semantical one, but whether the mechanism of these reactions is a non-immunological release of mediators or a truly allergic response.

We have recently reported [2] a case of an acute reaction (generalized urticaria and lipoedema) to paracetamol, proven by rechallenge with 75 mg of the drug. An oral challenge with 500 mg of aspirin was well tolerated. Both skin-prick test results to paracetamol and specific-IgE determination by RAST to a protein conjugate of the drug were positive in our patient. We validated our results by skin testing 30 control subjects and testing sera from 10 non-allergic donors.

Our results suggest that at least some of those cases of paracetamol sensitivity without co-existing aspirin intolerance might be allergic in nature. Skin tests are generally of little value for the diagnosis of drug allergy, but they may be nonetheless useful tools, if properly performed and controlled, in detecting cases of allergic reactions to chemicals.

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J. A. MARTIN, M. LAZARO, M. CUEVAS,
 E. ALVAREZ-CUESTA
*Servicio de Alergia,
 Hospital Ramon y Cajal,
 28034 Madrid,
 Spain*