Mediators of Inflammation, **8**, 181–182 (1999)

Letters to the Editor

BN 52021 decreases alveolar macrophage-mediated lung injury in experimental extrinsic allergic alveolitis

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EDITOR – We read with great interest the publication from Pérez-Arellano and colleagues.[1] The aim of this study was to evaluate the participation of PAF in extrinsic allergic alveolitis. We would like to bring to your attention the fact that we published a similar study a few years ago.[2] Both studies show striking methodological similarities, namely the use of the same antigen to induce EAA and the same PAF antagonist. However, we reported a decreased total BAL cell counts in BN 52021 treated animals in contrast to the results as described by Pérez-Arellano *et al*. We acknowledge that they looked at more parameters than we did, but it would have been fair just to mention our previous publication in the introduction and discuss discrepancies between their and our results. Considering the easy access to published materials with databases such as PubMed, we question if such an omission was intentional, especially when both studies reach a similar conclusion.

References

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2. Tremblay GM, Israel-Assayag E, Sirois P, Cormier Y. Murine hypersensitivity pneumonitis: evidences for the role of eicosanoids and platelet activating factor. *Immunol Invest* 1993; **22:** 341–352.

Reply to G. Tremblay

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EDITOR – We regret that Dr Tremblay's work was not mentioned in our paper. A more thorough searching of the database might have found his paper, but at the time we were unaware of its existence. For this we would ask you to send our apologies to Dr Tremblay.

However , we disagree with a number of points made by Dr Tremblay, namely:

(a) We do not think that there are 'striking methodological similarities' between both papers. Briefly, we used an experimental model previously described in hamsters (*Mediators Inflamm* 1995; **4:** 43–48), because in a previous review (*J Invest Allergol Clin Immunol* 1992; **2:** 219–228) we found that "the model has proved useful for studying the disease since it reproduces all the main features of it, namely, alveolitis, granuloma formation, the production of precipitins and a delayed hypersensitivity response". However, Dr Tremblay and coworkers used an experimental model developed in mice which has both advantages and limitations (e.g. bronchoalveolar T helper response compared with the elevation in suppressor/cytotoxic lymphocytes in humans). Furthermore, they used an intranasal route for administration of antigen whereas in our work we used intratracheal administration. Moreover, the protocol of administration of the antigen and BN 52021 is very different in both papers. These differences could probably explain the discrepancies observed in the results obtained for total and differential counts.

(b) Concerning the criticism about the use of the 'same antigen', we should point out that *Faenia rectivirgula* has been used for a long time in experimental models of extrinsic allergic alveolitis by many groups.[1–3] Specifically, we use the protocol of Schuyler and Crooks[4] as indicated in our paper. Indeed, as mentioned in our paper (ref.

ISSN 0962-9351 print/ISSN 1466-1861 online/99/030181-02 © 1999 Taylor & Francis Ltd

17), one of us (Prof. J.M. Lopez Novoa) has used BN 52021 (provided by Dr P. Braquet) prior to 1993 (the date of publication of Dr Tremblay's paper). We are therefore very surprised that Dr Tremblay sees fit to draw attention to our use of this antigen when it is so widely used by others, and strongly refute the implication in his final sentence that we intentionally omitted to cite his work.

(c) The methods used for the evaluation of the role of PAF in the pathogenesis of EAA is very different in both papers. Their results include two main aspects: the number and type of cells recovered in BAL, and the production of eicosanoids by alveolar macrophages. In our work we evaluated several parameters of parenchymal lung disease (not only alveolitis) and we measured PAF directly. So, with the exception of BAL lavage (a technique used in all papers that investigate the pathogenesis of interstitial lung disease) the rationale of the work is totally different.

In conclusion, while we recognize the omission of their very interesting paper from our paper, we strongly deny that this was in any way deliberate.

References

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2. Wilkie, BN. Experimental hypersensitivity pneumonitis. Humoral and cell-mediated immune. Response of cattle to Microspolyspora Faeni and clinical response to aerosol challenge. *Int Archs Allergy Appl Immun* 1976; **50:** 359–373.
3. Zaidi SH, Dogra RKS, Shanker R, Chandra SV. *J Pathol* 1971; **105:** 41–48.
4. Schuyler M, Crooks L. Experimental hypersensitivity pneumonitis in guinea pigs. Kinetics and dose response. *Am Rev Respir Dis* 1989; **139:** 996–1002.