Aggregation Responses to Platelet-Activating Factor in Normal Subjects

E.		Caffrey
E.		Madden
R.		Lambe
A.		Darragh

Institute of Clinical Pharmacology, Dublin, Ireland

E. Caffrey, MD, Institute of Clinical Pharmacology, PO Box 469, 1 James's Street, Dublin 8 (Ireland)

In a recent article Viero et al. [1] reported abnormal platelet aggregation in response to platelet-activating factor (PAF) in 6 out of 10 patients with chronic myeloproliferative disorders while all 10 controls provided normal biphasic responses. In extensive studies on a range of PAF inhibitors we have found a high degree of intersubject variability in response to PAF in platelets from healthy volunteers. Furthermore we have found that some volunteers (which include 2 of the authors) consistently demonstrate only a first-phase aggregation response, followed by rapid disaggregation in response to concentrations of PAF as high as 600 nM, while showing normal aggregation in response to other aggregating agents.

We suggest that subjects (either patients or volunteers) who show only a first-phase aggregation response to PAF do not necessarily have a platelet aggregation defect and that any apparent correlation between aggregation responses to PAF and specific disease states should be interpreted with caution.

Reference

Viero, P.; Cortelazzo, S.; Barbu, T.; Gaetano, G. de: Defective platelet aggregation induced by platelet activating factor in myeloproliferative disorders: deficiency of an aspirin-independent mechanism? Haemostasis 16: 27-33 (1986).

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Correspondence

Authors' Reply

We thank Dr. Caffrey and his/her colleagues for their interesting comments on our paper [1]. We are aware of a great individual variability in human response to PAF. In a previous study, for instance, the threshold concentration of PAF inducing irreversible aggregation within 3 min in platelet-rich plasma from 26 healthy donors ranged between 6.2 and 150 nM (median 25 nM) [2]. Irreversible aggregation by PAF could also be obtained in all control individuals tested in Bergamo by Macconi et al. [3] and in Milan by Cattaneo et al. [4]. Klo-progge et al. [5] reported that platelets collected from fasting donors better preserved their responsiveness to PAF than those collected from nonfasting donors. The lack of responsiveness to PAF in the latter group was evident in those who had taken a lipid-rich meal, whereas a lipid-poor meal had no effect. We therefore always pay attention to the fasting state of our volunteers and patients. Perhaps some factors in the 'Mediterranean' diet of Italians might contribute to the apparently better and more constant response of their platelets to PAF stimulation.

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Piera Viero, Sergio Cortelazzo, Tiziano Barbui, Division of Haematology, Ospedali Riuniti, 1–24100 Bergamo (Italy)

Giovanni de Gaetano,

Istituto di Ricerche Farmacologiche Mario Negri,

1–20157 Milano (Italy)