

Author's reply

Much of the criticism of our therapeutic approach seems to be based on interpretation of the mechanism of Celacade. Our hypothesis relates to the now well accepted anti-inflammatory sequelae of ingestion of apoptotic cells by antigen-presenting cells. Clearly many (most?) cell types in addition to professional phagocytes engulf apoptotic cells with anti-inflammatory consequences, and presumably the removal of most of the billions of cells undergoing apoptosis daily will be affected by neighbouring cells.¹ Defective clearance of apoptotic cells seems to be associated with inflammation.²

By the intramuscular injection of a bolus of apoptotic cells, we hypothesise that we mimic the events leading to the resolution of acute inflammation,³ clearly a very potent anti-inflammatory response. The factors to which blood is exposed in the Celacade process include ultra-violet light and the gaseous oxidising agent ozone—stresses known to induce apoptotic cell death, delivered at a dose that we have found does not significantly affect the white blood cell count, suggesting that there is minimal cell rupture and release of proinflammatory factors after blood processing. Any release of stress proteins, some of which seem to have anti-inflammatory properties,⁴ could contribute to the overall anti-inflammatory effects of Celacade. The effects of Celacade have been explored extensively in animals,⁵ with results clearly showing an anti-inflammatory response in vivo.

Velio Bocci hypothesises that the effects of his treatment include vasodilatation, increased delivery of oxygen to tissues, and upregulation of antioxidant enzymes. Both our theories have a commonality in that inflammation leads to endothelial activation, a reduction in endothelial nitric oxide production, and, hence, increased vasoconstriction. A systemic reduction in inflammatory mediators

should reduce this vasoconstrictive drive.

The use of a saline placebo can be justified after the demonstration in animal models of an absence of significant differences in responses to saline and whole blood.

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- 1 Cvetanovic M, Mitchell JE, Patel V, et al. *J Biol Chem* 2006; **281**: 20055–67.
- 2 Savill J, Dransfield I, Gregory C, et al. *Nat Rev Immunol* 2002; **2**: 965–75.
- 3 Serhan CN, Savill J *Nat Immunol* 2005; **6**: 1191–97.
- 4 Frostgard J, Pockley AG. In: Henderson B, Pockley AG, eds. *Molecular chaperones and cell signalling*. Cambridge: Cambridge University Press, 2005: 195–219.
- 5 Bolton AE. Biologic effects and basic science of a novel immune-modulation therapy. *Am J Cardiol* 2005; **95** (suppl): 24C–29C.

Open-access journals are delivering high impact, and more...

We welcome *The Lancet's* Editorial on open-access publishing (March 8, p 785),¹ but question the claim that "Although BioMed Central has grown substantially during the past 3 years, it has yet to capture the quality end of the research sector".

Impact factors are the most commonly used journal quality metric, and *Malaria Journal* (ranked number 1 in tropical medicine) is one of many examples of BioMed Central journals with impressive impact factors.

Unfortunately, however, Thomson Scientific's failure to track many new open-access journals means that these impact factors still do not provide the full picture.

The SCImago Journal Rank (SJR) is an alternative citation metric based on data from Elsevier's more comprehensive Scopus service, which covers more than 13 000 journals. In the most recent SCImago rankings, two BioMed Central journals, *Journal of Biology* and

Genome Biology, are ranked in the top 0.5% of all journals listed, ahead of all five PLoS titles. More than half of ranked BioMed Central journals are in the top 15% of the SJR listings, indicating that a typical BioMed Central title is substantially more highly cited than the average traditional journal.

We also note that, important as high-impact journals are, they do not fully address the challenge of communicating research results. *BMC Research Notes* and the *Journal of Medical Case Reports* are two examples of titles that explicitly seek to publish material, such as negative clinical trial results, that is unlikely to deliver a high impact factor. This material nevertheless forms a crucial part of the scholarly record.

MN and MC are both employees of BioMed Central, the publisher referred to in the article, and MC has a financial interest in the company.

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- 1 The Lancet. Clinical knowledge: from access to action. *Lancet* 2008; **371**: 785.

Department of Error

Cholesterol Treatment Trialists' (CTT) Collaborators. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90 056 participants in 14 randomised controlled trials of statins. Lancet 2005; **366**: 1267–78—In this Article (Oct 8), although the correct numbers were analysed in the figures, and the findings are therefore unaltered, there were some minor inaccuracies in the table describing contributing trials (p 1268). The number of patients whose baseline history of vascular disease was listed as "None" should have been **6586 (>99%)** in the AFCAPS/TexCAPS trial and **8037 (78%)** in the ALLHAT-LLT trial, whereas the number whose baseline history was listed as "Other vascular" in the ALLHAT-LLT trial should have been **1788 (17%)**. The total number of patients with other vascular disease and no vascular disease should therefore have been **15 043 (17%)** and **40 444 (45%)**, respectively.