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Response

Wolpoff's substantive criticism of our paper is that we recognized far more species of *Homo* than are compatible with the "majority interpretation of Pleistocene human evolution." In response, we should like to point out that we never actually claimed that our interpretation is the majority one. Rather, we made it clear (in the second column of page 65) that there are two schools of thought regarding the number of species of *Homo*, and that we were deliberately opting for the more speciose of the taxonomies favored by these schools. We suggested that there were theoretical and practical reasons for recognizing multiple *Homo* species, and cited a paper by Tattersall in which those reasons are explained. In short, Wolpoff may disagree with our taxonomy and reject our reasons for choosing it, but he cannot say that we presented a misleading account of current views on specific diversity in *Homo*.

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Wound Healing

The excellent work of Vishwanath R. Iyer and his colleagues, described in the report "The transcriptional program and the response of human fibroblasts to serum" (1 Jan., p. 83), will surely lead to important advances in wound healing. However, some of the comparisons to wound healing that are made by the authors and in the accompanying News of the Week article (E. Pennisi, 1 Jan., p. 17) do not appear to be fully warranted.

Reexposing starved, pure cultures of fibroblasts to dilute serum is only superficially similar to wound healing, where fibroblasts (i) are not alone, (ii) are not serum starved, and (iii) are exposed to an environment which, although based in serum, is highly modified.

The implication should not be given that fibroblasts are commonly thought to be passive responders in wound healing. We know that fibroblasts participate actively in wound healing. We know that they condition the environment with a variety of substances ranging from lactate to growth factors. However, fibroblasts are not prime movers, either. In wound healing, the temporal relationship is injury, fol-



"Reexposing starved, pure cultures of fibroblasts to dilute serum is only superficially similar to wound healing."

lowed by inflammation, followed by fibroplasia and angiogenesis. Without inflammation, fibroplasia is severely limited. In terms of spatial relationships, macrophages lead fibroblasts and endothelial cells into the blood or fibrin clot or the residual connective tissue matrix. It is well understood that fibroblasts replicate much of what macrophages and lymphocytes do, but to a lesser degree.

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Critical Volume Fraction: Second Model

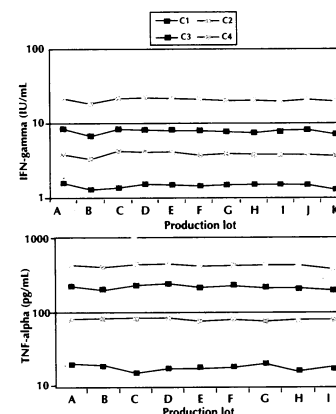
With respect to the report I co-authored with S. F. Ackley and V. I. Lytle, "The percolation phase transition in sea ice" (18 Dec. 1998, p. 2238), I would like to thank Jay Janzen for making me aware of his work on the critical volume fraction ϕ_c for conduction in a compressed powder of large polymer particles and much smaller metal particles. Had I been aware of two of his papers (1, 2) (which were

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