

# THE ROCKEFELLER UNIVERSITY

*pro bono humani generis*

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Dr. W. C. Bushell  
280 Riverside Drive, #2K  
NY NY 10025

Dear Dr Bushell

I respond to yours of June 24.

As you must have had in mind to write me, I am sympathetic to your interest in fever, and believe we hardly begin to understand its role in the dynamics of host-parasite relationships. (Andre Lwoff waxed eloquent about it). And I did give Neal Miller strong support for the research he conducted here. There was a longstanding tradition of the use of induced fever for the treatment of syphilis.

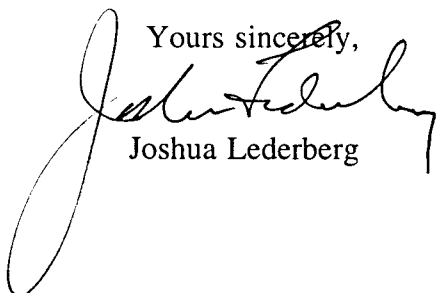
I'm a little puzzled about hyperthermia per se contributing to oxygen-availability. That is more likely secondary to circulatory enhancement. Or is the dissociation of O<sub>2</sub> from Hb significantly enhanced with a small rise in temperature?

However before leaping to all the complexities of biobehavioral approaches, I would have to take a reductionist stance and try to dissect exactly what fever (or is it vasodilation, or other components of the inflammatory response) is doing in pathogenesis. I believe this dissection will be easier with the use, say, of diathermy and other physical interventions. When/if the virtues and possible side-effects are settled, one might then go on to the subtleties of the biobehavioral instruments. We obviously disagree about that strategy.

At present, I am in theoretical exploration of how fever might contribute to the nucleation of fibrillogenic proteins, as in Alzheimer's, prion diseases and Huntington's. If you run into any epi- data that might bear on that question, please pass it on. Other aspects of inflammation have long been linked to those disorders.

I wish you well, but for now I'll pass as far as conference participation is concerned, but I hope you'll keep me informed.

*I have accumulated some  
further bibliography and share  
it with you.*

Yours sincerely,  
  
Joshua Lederberg