

Hunter



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BETHESDA, MARYLAND 20814  
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PEDIATRICS

TEACHING HOSPITALS  
WALTER REED ARMY MEDICAL CENTER  
NATIONAL NAVAL MEDICAL CENTER  
MALCOLM GROW AIR FORCE MEDICAL CENTER  
WILFORD HALL AIR FORCE MEDICAL CENTER

Seven clones:

Send P P W 229  
P P W ~~22~~ 244  
88.1 & 188  
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88.4

Matthew Meselson, Ph.D.  
Department of Biochemistry  
and Molecular Biology  
Harvard University  
Cambridge, MA 02138

Dear Dr. Meselson:

This letter concerns two unrelated topics. First, my laboratory has been engaged in a study of differentiation in the pathogenic human protozoan parasite Leishmania mexicana. During the course of these studies we found that the parasite, while undergoing temperature-induced differentiation from promastigote to amastigote stage in vitro, produced what we think are the classic heat shock proteins seen in many other species. At first blush this might seem to be a rather ordinary finding, but Leishmania presents a more interesting picture. If these are indeed heat shock proteins, they are produced during the active and successful differentiation of this parasite; an event with a notable change in morphology. A second observation deals with the fact that unlike other organisms in which heat shock has been studied, the elevation of habitat temperature is part of the normal life cycle for the leishmanial parasite and is thus continuous rather than transient. The parasites must adapt or die. The control of heat shock protein synthesis under these conditions may be somewhat unconventional.

In any case, we are writing a small NIH grant and wonder whether you might donate cDNA probes for the 70KD and 83KD heat shock genes. Dr. Milton Schlesinger has kindly provided antisera to these proteins for blot confirmation, and the cDNA probes would be of similar utility. If such probes can be provided, a letter stating such would be greatly appreciated (prior to July 1 if possible).

The second topic of this letter deals with T-2. My laboratory has, under Army contract, prepared a mouse monoclonal antibody specific for this mycotoxin. The antibody has an affinity of  $3.7 \times 10^6$  liters/mole against T-2, and though it cross-reacts well with HT-2, it does not recognize diacetoxyscirpenol, monoacetoxyscirpenol, or deoxynivalenol. This antibody can be manipulated in a competitive inhibition enzyme immunoassay (CIEIA) to detect as little as  $1.0 \times 10^{-7}$  M T-2. The Army, possibly due to its abiding interest in bees, has contracted to produce rapid, field adaptable, antibody-based "dipstick" detectors for T-2.

Be that as it may, I wonder whether you might like to employ the antibodies as as a rapid, field adaptable enzyme immunoassay adjunct to the conventional physico-chemical quantification methods? It would be my pleasure to provide these reagents and instructions for performing the CIEIA.

As an individual privy to most of the information extant regarding T-2 as a potential CW agent, I tend to believe that the most important application of our detection system is in the agricultural arena.

I hope we can chat some time about our common interests.

Sincerely,



Kenneth W. Hunter, Jr., Sc.D.  
Associate Professor of Pediatrics  
and Preventive Medicine/Biometrics