DEPARTMENT OF BIOCHEMISTRY AND MOLECULAR BIOLOGY
HARVARD UNIVERSITY

7 Divinity Avenue
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Dr. Kenneth W. Hunter Jr.
Uniformed Services University of
Health Sciences
4301 Jones Bridge Road
Bethesda, MD 20418

Dear Dr. Hunter:

Thank you very much for writing to me. I had read about your work on anti-T2 antibody in 'U.S. Medicine' and intended to contact you. Yes, I would like to employ CIEIA in testing for T2 in samples of agricultural products and other materials Tom Seeley and I brought back from Thailand in March. But it won't be until August or September that we can get started, since I will be away from mid-June until early August. If it is agreeable to you, I'd like to visit your lab to learn the technique next time I come to the Washington area, probably in September. I enclose several items related to the yellow rain problem. One of them shows the remarkable seasonal clustering of the collection times of the environmental and biomedical samples reported to contain trichothecenes. Is this an artifact of sampling times or some uncontrolled variable in the analyses themselves or does it reflect true seasonality of occurrence? It is not inconsistent with the seasonality of fusarium contamination of Bangkok foods reported by Shank, Wogan and Gibson. It suggests that further samples collected in February-April are particularly worth analyzing. Yet I have some doubt as to whether the analyses are correct. As I understand it, the half-life of T2 in the blood of swine and cattle after intravenous injection is about 15 minutes. Moreover, after intubation at near-lethal dosage, no T2 at all is found in the blood of swine, cattle or cats, at a detection limit of 1ppb. How is it then that Mirocha finds up to 110 ppb of T2 and an average of about 25ppb in the blood of persons alleged to have been attacked days to weeks before sampling? Unlike swine, cattle and cats, can people withstand blood levels of many ppb for many days and still be walking around? Is it possible that Mirocha is detecting something other than T2 which is not even a metabolite of T2 but which is seasonal and is associated with

illness? I see no other explanation but neither have I much confidence in it. Also, Edgewood, and I believe, Porton find no trichothecenes in any of about 100 environmental samples they have looked at.

Under separate cover, I am sending seven clones of D. melanogaster heat shock genes. These include clones of the small heat shock genes which are under developmental control. Two, hsp26 and hsp28, are expressed in oogenesis. It is reported that all four are expressed in response to ecdysterone in isolated imaginal discs and also in pupae. Can you induce Leischmania to differentiate in response to any stimulus besides temperature elevation?

Sincerely yours,

Matthew Meselson

Matthew Meselson
Professor of Biochemistry
and Molecular Biology

MM/db

Enclosures