May 12, 1986

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Dear Dr. Susskind:

Many thanks for the lengthy write-up on progress to date.

As I said earlier, we are indeed fortunate to have you studying the problem.

As to the date when we shall be there, I shall need to leave it up to Dr. Pybus, as his schedule is the one @ must adjust to. So if he cannot meet with you, there would be no point in my coming, as I'm not technically trained in your areas.

I reread Wyburn-Mason's A New Protozoon and began to wonder if Stamm and Wyburn-Mason did not mistake one of the pleomorphic bacteria (that you call an L form -- and incidentally I sent material to Paul Pybus on prggenitor cryptocides described by Dr. Virginia Livingston-Wheeler) for Amoeba chromatosa. It is interesting -- perhaps more than coincidental -- that Livingston-Wheeler describes the progenitor cryptocides in every single place where Wyburn-Mason describes Amoeba chromatosa: plant gall, surface soils, cancer tumors, lymphomas, leukemias, arthritics, and so on.

Roger says the Amoeba chromatosa causes some cancer; Livingston-Wheeler says that the progenitor cryptocides causes some arthritis. Brown says that a mycoplasmic bacteria is involved with RD.

So -- could Roger's Amoeba chromatosa be a transient form of the pleomorphic organisms progenitor cryptocides which is also defined, I believe, as a mychplasmic bacteria, an L form? Certainly when A New Protozoon was written, Stamm and Wyburn-Mason did not have advantage of powerful microscopes, nor a whole lot of knowledge about pleomorphic organisms -- although Roger does mention one class of L forms, I believe.

I am sending Roger's books off to a specialist on the pleomorphic organism to compare possible characteristics.

(over)

I have no problem with changing research directions, except for one thing: I believe that Paul Pybus and myself have long claimeddthat we will not find the Amoeba chromatosa in knee effusions, where most everyone has been looking, because that is so easy to get (comparatively).

We believe that Roger and Stamm probably found the organism in tumorous tissue.

Until that is searched out, I don't believe we can yet say that Roger and Stamm were mistaken.

And yes -- I believe that physician's clinical experience demonstrates that those who are newly acquired with RD symptoms are the ones who respond fastest and best. RD patients who are burned out, do not respond as well.

And, in my case, I had one joint that continued to degenerate over more than 1½ years, despite every treatment known, and despite no longer having all the normal symptoms of RD.

I stopped the destruction with EDTA chelation therapy, five times repeated. Theory: Iron from defective red blood cell acts as chemical catalyst producing a whole cascade of inflammatory actions, using the cartilage as fuel. By chelating out the iron, the action stopped.

So I'm sure that your statement about sequelae is correct in that instances, and probably many other instances. Problem with rheumatologists and RD victims is that they are used to looking at the disease as a set of manifested symptoms, and not as a distinction between damage that is done, and diseasesas an on-going process.

The other problem that I see, by changing directions (which I'm not necessarily against doing) is that one must now come up with a viable theory that explains and predicts clinical aspects as well as Roger's.

I'll wait on Paul Pybus to determine what we shall do about the visit.

Cordially,

Perry A. Chapdelaine, Sr.