Dr. Peter Seland
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Dear Dr. Seland:

I was pleased to hear that you will be addressing the annual Meeting of the Calgary Branch and that you will be discussing our Society's research effort. I am keenly interested in this topic and I am hoping that it might be possible for you and I to meet before or after the annual meeting (essentially at any time which is convenient for you) to discuss the need for a research effort in the relationship between diet and MS.

As you may already know I have been looking into the concept of dietary factors playing a significant role in MS since 1995. This resulted from an effort to determine which proposed causes of MS can be considered as plausible (i.e. reasonable in regard to current epidemiological and immunological data and no available data which negate it) and which can be put aside as implausible (available data negate it or make it highly unlikely).

I found that the majority of proposed causes could be rendered implausible by our current, extensive data base for MS. For example the hypothesis that MS is caused by dental amalgams is frequently raised but to me the available data such as (1) many with MS have no amalgams, (2) it does not fit with the established N-S gradient in genetically homogenous populations, (3) MS brains have slightly less Hg than controls, (4) MS is not more prevalent in people who are exposed to excess Hg, indicate that it is implausible and should not be taken seriously at this time.

This exercise of examining each proposed cause in the harsh light of the available data resulted in the number of plausible causes being reduced to three: (1) specific infectious agent (e.g. HHV-6) within the CNS, (2) random infectious agents precipitating an autoimmune reaction through molecular mimicry of CNS auto-antigens, (3) dietary proteins precipitating an autoimmune reaction through molecular mimicry of both childhood infectious agents (e.g. HHV-6, EBV, HZ) and CNS auto-antigens (three way cross reaction).

My scientific philosophy is that every plausible cause of MS should be vigorously researched. MS is a devastating, enigmatic disease and such a combination demands that every potentially worthwhile avenue of research be explored. Thus to me no plausible cause should be ignored.

I am sure that we are in agreement that the two viral-driven hypotheses are plausible. Their apparent lack of compatibility with the N-S gradient pattern (e.g. Australia, New Zealand, North America) is of some concern but not enough to deny them plausible status. I am pleased that our Society supports research which addresses these potential causes.

This brings us to my third plausible cause, diet, and it would appear that you and your colleagues on the Medical Advisory Committee do not consider it to be plausible. This disagreement needs to be resolved with our available scientific data base being the obvious means to a resolution. I have outlined my reasons for believing the diet hypothesis is plausible in a letter to Sarah Pepall and a copy should have been given to you. On the oft chance it has not yet reached you, I will briefly summarize my reasoning:

1. There is a unifying concept, often referred to as genetic discordance, which predicts that foods recently (<5000 years) introduced into our diets (e.g. dairy, gluten) have the potential to cause biochemical malfunctions (disease) in some persons due to their genetic structure. The basis of this concept is the fact that the genetic structure of homo sapiens is 40,000+ years old and is compatible with a food supply of fruits, vegetables and lean, wild meat. The newly introduced foods contain anti-nutrients and proteins with which our genes may not be compatible and which thus can cause biochemical malfunctions. This overarching concept is certainly applicable to diseases such as CHD, CVD and various cancers. It is not unreasonable to assume it may well apply to autoimmune diseases including MS.

2. Given that some "new" foods may be involved in autoimmune disease, the next question is "is there a reasonable pathogenic model for these foods to cause MS". Researchers in the USA have recently formulated a reasonable and theoretically possible model involving proteins from the new foods. The model includes (a) the escape of intact food proteins fragments through an abnormally permeable intestine into the circulatory system, (b) molecular mimicry of childhood infectious agents and CNS self-antigens by the food proteins, and (c) consequent activation of the immune system against auto-antigens in the CNS. I have found no evidence that such a pathogenesis is not possible.

3. Epidemiological studies have revealed highly significant correlations ($p<.001$) between the consumption of new foods such as milk and saturated fat and MS prevalence. Also, to me, the well documented N-S gradient in genetically homogenous populations such as Australia and New Zealand is best explained by dietary changes which correlate to climatic belts.

4. Dr. F.W. Scott of the Nutrition Research Division of Health Canada has conclusively demonstrated that proteins from such "new" foods as wheat, soy and milk precipitate cell- mediated, organ-specific autoimmunity in genetically susceptible rats (Type 1-diabetes). I sent a copy of a summary paper of this "smoking gun" research to the MS Society for you but again, on the chance it has not reached you, I am including a copy herein.

5. Notably your colleague, Dr. George Ebers, on the basis of a recent epidemiological study, has stated in a refereed publication "In sum these data strongly indicate that the environmental factor is affecting the population risk. Accordingly, factors which influence large populations such as diet ... deserve careful reconsideration". To me, the endorsement by Dr. Ebers that diet is a potential factor in MS should not be ignored.

6. There are numerous published accounts of "success" by using a diet which excludes or greatly reduces consumption of the "new" foods (e.g. The Roger MacDougall Story). There is no doubt that such anecdotal data mean nothing by themselves but, in combination with the above considerations, they provide some additional support to the plausibility of the diet hypothesis.

To me the above six points are sufficient to consider the diet hypothesis as plausible and in need of further investigation. I realize that you and your colleagues know a great deal more about MS than I and might well be able to cite references which render the hypothesis untenable or highly unlikely. If so, I would be very appreciative of receiving such references. I have no desire to pursue an implausible hypothesis.

I was hoping that we could meet and you could briefly summarize the reasons why you and your committee do not consider the diet hypothesis worthy of any research funding. Of course, if you see some merit in such a hypothesis and agree that it is plausible, perhaps we could discuss potential research initiatives and how the MS Society could best promote them.

To me, the upside of such a research initiative is huge. If diet revision proves to be an effective therapy for slowing or halting MS progression, it could have several applications. It would provide a safe, no cost alternative for those unable or unwilling to use the current drugs. It could be used in concert with current drug therapies and this would likely result in much increased efficacies. At any rate it would definitely

help to bridge the gap between our current therapies and future (10+ years?), much more effective ones which would completely nullify autoimmune reactions and even possibly stimulate axon and myelin regeneration.

Of course if diet revision proves to be an ineffective therapy then doctors will at last be able to provide persons with MS with reliable information on that frequently asked question "Does diet play a role in MS?" The current answer provided by our Society and neurologists of "we do not know" clearly is not adequate. Thus diet research will significantly benefit persons with MS regardless of the final outcome of the research.

I hope you are agreeable to a short meeting with me to discuss this matter. I believe it would be beneficial for all concerned.

I would appreciate it if you could let me know sometime in the next few weeks by fax (403- 292-4961), phone (403-292-7125), email (**embry@gsc.nrcan.gc.ca**) or letter (5119 Brockington Rd. NW, Calgary, AB T2L 1R7) if you are amenable to this proposed meeting before or after the Calgary Annual Meeting.

Thank you very much for considering this material and my request to meet with you.

Yours truly,

Ashton F. Embry