

The Diet Hypothesis for the Cause of MS

By Ashton Embry

The cause of MS is unknown but it must be realized that there is an incredibly large data base on MS which has been collected over the past 40 years. This data base consists of at least 25000 published scientific articles in medical journals, symposium volumes and textbooks. These data constrain POSSIBLE causes very thoroughly and thus we know that most proposed causes are not tenable. Proposed causes which cannot be eliminated by the current data base are PLAUSIBLE causes and are the ones which drive the research effort.

The two broad categories of plausible causes are:

1. MS is an autoimmune disease, that is one's own immune system is attacking the myelin in the CNS. This is by far the most widely held view and almost all research and all current drug therapy is based on the premise that MS is an autoimmune disease.
2. MS is an infectious disease, that is a virus or bacteria infects the CNS and provokes an immune response which results in myelin destruction. This concept is held only by a few researchers and no clinical treatment is based on this concept. Importantly, despite an intense search with very sophisticated technology, no infectious agent has ever been identified. Overall it would appear most likely that MS is an autoimmune disease and the evidence for this is very strong.

Given that MS is autoimmune, the big question as to cause is what activates the immune system to attack self tissue in the CNS. Again almost all researchers agree that the immune system is activated against self in the periphery, that is in the blood outside of the CNS. Furthermore the source of the activation almost assuredly must be a foreign protein. The most obvious source of foreign proteins which can activate the immune system are infectious agents.

There are two possible ways infectious agents can activate the immune system against self. One is by what is known as a super-antigen which can activate many different T cell lines at once. Some of these activated T cells could be those which recognize a myelin protein as foreign and this would then lead to an attack on myelin.

The second way infectious agents can activate the immune system against self is by **MOLECULAR MIMICRY** which involves the similarity of part of the molecular structure of the infectious agent with part of a protein molecule of myelin. Thus when the immune system is activated against the infectious agent it also becomes activated against myelin. There is a great deal of evidence to support the concept of molecular mimicry being the key process in MS and many other autoimmune diseases. For example a researcher recently found a very specific antibody in the spinal fluid of a number of PwMS. This antibody was found to be reactive with both a protein in myelin AND Epstein Barr virus. The odds of such an occurrence being by chance are astronomic. Thus we can feel relatively confident that MS is an autoimmune disease caused by molecular mimicry between myelin proteins and foreign proteins.

Another source of data for determining MS cause is epidemiology which means where does the disease exist most commonly and what types of people are most likely to get it. The most important finding is that MS is dominant in Caucasians in temperate climates. Another more specific finding is fishing communities have much less MS than farming communities (when latitude and genetics are held constant). This presents a big problem for the concept that molecular mimicry is caused by common infectious agents which do not exhibit the same geographic variance as MS does.

The disease process data have recently been reconciled with the epidemiological data by the diet hypothesis for MS.

I thought it might be helpful if I briefly outlined the diet hypothesis which forms the background and the rationalization for recommending diet revision as a worthwhile therapy for combating MS. The basic tenet of the hypothesis is that dietary factors play a big role in MS onset and progression. The distinctive geographic distribution of MS and numerous anecdotal accounts (e.g. Roger MacDougall) have always suggested that diet may be involved in MS but a big stumbling block was that there was no obvious way that diet could be a driver of MS. This plus the lack of any financial incentive to test diet resulted in essentially no interest in the topic of diet and MS.

The diet model for MS cause is as follows:

1. Infection with one or more childhood illnesses(e.g. Epstein-Barr,HHV-6) which results in an autoimmune reaction against tissue in the CNS by molecular mimicry. Such autoimmune reactions are suppressed before any demyelination occurs. However memory cells against the infectious agent are produced and such memory cells can be seen as an autoimmune time bomb because they are also potentially autoaggressive.
2. With time intestinal permeability increases due to various factors including food allergies, alcohol consumption, candida overgrowth and non steroidal anti-inflammatory drugs. The consumption of gluten and legumes also increases gut permeability through the action of lectins (glycoproteins).
3. With a **leaky gut**, intact food proteins begin to escape the gut as do gut bacteria(e.g. E.coli).These antigens precipitate an autoimmune response by molecular mimicry of the childhood infectious agents and self antigens in the CNS. This results in activation of the autoaggressive memory cells. These initial reactions are most commonly successfully suppressed by a reasonably well functioning immune system before any clinically detectable damage is done.
4. The chronic activation of the autoaggressive memory cells by food and bacteria mimics which are continually escaping through a leaky gut finally results in a failure of the suppressor side and a major autoimmune attack occurs. This is eventually suppressed and depending on the strength of the immune system and its ability to suppress autoimmune reactions the individual experiences a benign,a relapsing-remitting (younger, healthier immune system) or a progressive course (older, degraded immune system).

The **Paleolithic aspect** of the concept comes in when the question is asked why would foods cause such reactions. Clearly foods which humans have eaten throughout their development(2 million years) would not cause such reactions because all those with incompatible genes would have been eliminated by negative selection long before now. Thus the foods which cause such destructive damage must have been added to the human diet relatively recently such that natural negative selection has not had sufficient time to remove the incompatible genes from the human gene pool.

Importantly the consumption of the new foods correlates extremely well with MS prevalence. Furthermore those who had reported success with diet revision had done so by removing these new foods from their diets. Thus data from a variety of independent sources is converging on the concept that protein rich foods recently added to the human diet are causing MS by molecular mimicry.

Of course the animal studies which show that such foods can indeed cause autoimmunity is icing on the cake.

Only a proper clinical trial will test the validity of the concept.