The Three Stages of MS

By Ashton Embry

Many people think MS has two stages: relapsing remitting (R-R MS) and secondary progressive (SPMS). I see the MS disease process as having three distinct stages. The often ignored and perhaps most critical stage of MS, is the first one which can be called Pre-clinical MS (PCMS).

The MS disease process usually begins in early childhood and is most likely precipitated in a genetically susceptible child by a cross-reactive, viral infection in combination with a low vitamin D level (i.e. winter to early spring). The viral infection results in the activation of immune cells which are sensitized to both the virus and to a protein in myelin, the substance that coats the axons in the central nervous system. The occurrence of a low vitamin D level at this time ensures that the viral infection is not well controlled and this lack of control allows a substantial pool of memory immune cells to develop. These memory immune cells, which are sensitized to the myelin protein as well as the virus, are basically an MS time bomb that most often explodes 20 – 30 years later.

During the long Pre-clinical stage of MS various cross-reactive proteins from infectious agents and foods sporadically activate the myelin-sensitive memory cells especially during times when vitamin D levels are low. These immune cells cause minor inflammatory reactions in various areas of the brain but such inflammation is very diffuse and is not detectable with an MRI scan. The damage associated with these minor inflammatory autoimmune episodes does not cause any noticeable symptoms but even at this stage nerve axons are damaged and destroyed. With each activation, the pool of problematic memory cells expands and the next episode of autoimmunity is potentially more extensive and damaging. During the long Pre-Clinical stage the immune system is gradually altered such that autoimmunity becomes less well controlled and the potential for more substantial autoimmune reactions increases. Eventually a triggering event such as a cross-reactive viral infection during a time of low vitamin D level will precipitate a large enough autoimmune attack that symptoms become very apparent and the person sees a neurologist. A second, clinically apparent attack plus the appearance of distinct lesions on an MRI scan, usually results in a diagnosis of MS. The second stage of MS consists of well-defined attacks separated by periods of remission (R-R MS). New symptoms and disabilities often appear during an attack and many resolve during the following remission as the regulatory side of the immune system regains control. However there is sometimes a small incremental increase in disability by the time the next attack happens. An MS attack is caused by greatly increased, poorly controlled inflammation in the brain due to amplified autoimmune activity. The inflammation is often focused in distinct lesions although diffuse inflammation also occurs in the brain. The swelling associated with the inflammation results in most of the new symptoms. The inflammatory action, besides causing demyelination, also damages the nerve axons themselves and this can result in long-term disability. The more frequent and extensive the autoimmune-driven inflammatory action, the greater the axon damage and the greater the long-term disability.

Inflammation is not the only disease process which is contributing to increased disability during the R-R stage. When the nerve axons are damaged and severed in the lesions and possibly elsewhere in the brain and spinal chord, they degenerate along their entire length. This degeneration of the axons is perhaps the main cause of long-term disability. Thus the R-R stage is characterized by episodes of focal inflammation and by slow, progressive degeneration of nerve axons. Considerable disability accumulates due to these processes, especially the latter one.
The third and final phase of MS is known as Secondary Progressive MS (SPMS). It is characterized by few if any distinct attacks and by an insidious, slow and steady increase in disability. All the damage that occurred to the demyelinated nerve axons during the first two stages results in a slow and continuous degeneration of the long axon strands. This means less and less nerve impulses from the brain reach the muscles they are destined for and this translates into increased disability. Autoimmune inflammation continues during this time but is much more diffuse.

The long MS disease process which can essentially last a lifetime starts with low level inflammation, progresses to greatly increased inflammation and associated nerve axon degeneration and ends with continual nerve axon degeneration. Most of the accumulated disability is due to the degeneration component of the disease process. To avoid reaching the phase of steady degeneration and experiencing a downward spiral into serious disability, one must shut off the inflammatory process as early as possible. The ideal would be for susceptible persons to use a few nutritional strategies such as adequate vitamin D in childhood. However few do this because most believe that MS will never affect them.

More rigorous nutritional strategies and perhaps even one of the MS drugs are needed once MS is diagnosed. The sooner they are instituted the better the chance to short circuit the disease process and the appearance of long-term disability. Because many people do not experience serious symptoms in the early years of R-R MS they often do nothing and falsely believe that MS is no big deal. They do not realize that the disease process and associated damage are continuing. A major attack 5–10 years after diagnosis interval often results in disabilities which do not resolve. Suddenly they want to “do something” about their MS. Nutrition can still be very helpful at this time but because of the extent of inflammatory damage and ongoing degeneration, the chances of returning to a disability-free state are low.

Once one is in the SPMS stage it is critical to use the nutritional strategies and to have a good exercise program with associated mind concentration. The goal of such a program is to “rewire” the brain and have nerve impulses use new routes and thus restore abilities. Again the chances of halting disease progression at this time are low because of widespread degeneration of the axons.

Overall it is important to understand the stages in the evolution of the MS disease process, why disabilities occur and accumulate and what strategies are needed to try to counter the processes going on in each stage. The main lesson learned from this is that the sooner one starts using nutritional strategies the better the chances of halting disease progression and avoiding any problematic disabilities. It would be wonderful if every child was given adequate vitamin D from birth onward. MS would become a very rare disease if this was done.