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Editorial

Benefits and Hazards of Sun Exposure (Solar Radiation) in Multiple Sclerosis

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EDITORIAL

Studies of environment factors contributing to multiple sclerosis (MS) have provided strong evidence that sun exposure, the most important source of vitamin D, is protective [1-3]. Swank et al in 1952 cited and reviewed geographical disparities in the occurrence of MS in the earlier literature and reported the prevalence of MS in Norway [4]. This varied greatly from one geographic area to another with differences of approximately 4:1 observed, with higher rates correlating with inland dairy farming and animal fat consumption. An important corollary of increased consumption of animal fat is reduced consumption of fatty fish (and fish oils containing vitamin D). Fish oils have been used since the eighteenth century to prevent rickets in Northern European coastal areas with limited sunshine [5]. More recently, Kampman confirmed the lack of a latitudinal gradient for MS in Norway, pointing out that in coastal areas vitamin D sufficiency is maintained by ingested vitamin D from oily fish; supporting a protective role for vitamin D [6]. Although omega-3 fatty acids are also present in fish oils and a potential role has been suggested in earlier studies, a recent Norwegian placebo controlled study did not show any benefit [7]. Ebers has continued to emphasize latitudinal differences in MS incidence and correlation with sunshine.

By way of contrast, the potential hazards of solar radiation are less well appreciated. Hot bath testing has been abandoned because of the risk of inducing new neurological deficits but the risk of overheating from solar radiation in MS patients is largely unrecognized [8]. To illustrate this we describe an MS patient with severe neurological deficit following exposure to solar radiation. In December 2003 a 38-year-old African American man presented with onset of gait ataxia and a mild paraparesis. Brain magnetic resonance Imaging (MRI) revealed T2 hyperintense periventricular white matter lesions at callosal-septal interface, frontal gray-white matter junction, and brachium pontis. His cervical spinal MRI was normal. Cerebrospinal fluid revealed and an increased IgG/albumin ratio and 5 oligoclonal bands. He was diagnosed with MS. After recovery from a new paraparesis 2 years later, interferon beta-1b treatment was initiated. He subsequently stabilized, and continued to walk independently.

In spring 2007, 4 years after his initial diagnosis, his father left him in a chair at the beach. Returning 4 hours later, he found his son unable to speak or to move his limbs. Exposure to direct sunlight was estimated to be 3 hours at an ambient temperature of 75^0 F. from records of the National Weather Service. He was hospitalized in a community facility, quadriplegic and anarthric, and treated with intravenous methylprednisolone 1 gm. daily for 3 weeks. After two months he was transferred to our institution, afebrile but with severe decubiti. *Neurological examination* revealed severe dysarthria and quadriplegia. Deep tendon reflexes were decreased and a bilateral sensory level to pain below cervical (C2-C3) was present. A brain MRI revealed a prominent increase in periventricular T2 signal. Numerous black holes were also present on T1 sequences without contrast enhancement. Notably, MRI of the spinal cord revealed numerous new non-enhancing lesions of the cervical cord extending from the C2 to C6 level, without spinal cord enlargement or atrophy. His decubiti healed and interferon-beta-1b was reinstituted. He slowly recovered his ability to speak and use of his right upper extremity. However, a residual dense left hemiplegia has remained and he has been wheelchair bound.

We add a cautionary note based on our experience with this and other cases that we have encountered. While sun exposure is beneficial in MS [2,3,9,10], there are well known risks of actinic skin damage with its associated increased risk of cutaneous malignancies. Importantly, solar radiation may raise core body temperatures, not only temporarily increasing neurological manifestations of MS (Uhthoff phenomenon), but also risking the induction of new neurological deficits [8]. Heating of the head, in particular, we hypothesize, may facilitate activated lymphocyte homing into the brain and cervical spinal cord, as shown by Levin et al in experimental animals [11]. Vitamin D supplementation is a safe and effective alternative to excessive exposure to soar radiation in MS.

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