COMMENT. Subpial cortical lesions in MS are related to an inflammatory process in the meninges. Cortical demyelination occurs mostly in progressive MS, and is invisible by conventional MRI.

Abnormal T-cell reactivities in childhood inflammatory demyelinating disease. (Banwell B et al. **Ann Neurol** Jan 2008;63:98-111). Peripheral T-cell proliferative responses to self-, dietary, and control antigens were evaluated in children with CNS inflammatory demyelination, recent-onset type 1 diabetes mellitus, nonautoimmune neurologic disorders, and healthy children. Those with inflammatory demyelination, CNS injury, and diabetes showed heightened T-cell reactivities to self-antigens. Nonspecific T-cell dysregulation is an early feature of childhood onset MS and diabetes. The study highlights the possible relation between dietary antigens (eg cow-milk reactivities) and autoimmune diseases. High milk consumption and early weaning to foreign protein diets have been proposed as a possible MS risk factor (Malosse D et al. **Neuroepidemiology** 1992;11:304-312). In the present study, the incidence of MS and diabetes was not different in patients exposed to infant formula and those breast-fed exclusively, but the analysis was limited.

DEGENERATIVE DISEASE

NEONATAL DIAGNOSIS AND TREATMENT OF MENKES DISEASE

Infants diagnosed with Menkes disease early by plasma neurochemical methods and treated early, within 22 days after birth, with copper replacement therapy, had a 92% survival rate vs 13% in those treated late, Median follow-up in 12 newborns treated early was 4.6 years compared to 1.8 years in 15 diagnosed and treated late. Abnormally low copper dependent, dopamine-B-hydroxylase activity was identified by measuring plasma catecholamine levels in infants at risk. Response to treatment occurred only in patients with ATP7A mutations that permit some residual copper transport. (Kaler SG, Holmes CS, Goldstein DS et al. Neonatal diagnosis and treatment of Menkes disease. **N Engl J Med** Feb 7, 2008;358:605-614). (Reprints: Dr Kaler, National Institute of Child Health and Human Development, National Institutes of Health, Bldg 10, Rm 5-2571, 10 Center Dr, MSC 1832, Bethesda, MD 20892).

COMMENT. Menkes disease is an X-linked recessive infantile neurodegenerative disease caused by deficiency of a copper-transporting ATPase, ATP7A. Enzymes that require copper as a cofactor (dopamine-B-hydroxylase, cytochrome coxidase) are decreased. Symptoms are delayed for 6 to 8 weeks after birth. The disease is characterized by hypotonia, seizures, failure to thrive, and death by 3 years of age. Biochemical markers such as low serum copper and ceruloplasmin are unreliable in the neonatal period since they are low in normal neonates and overlap with the values found in Menkes disease. A molecular diagnosis, involving measurement of dopamine, norepinephrine and other catecholamines in plasma, is necessary to identify cases before symptoms develop and for copper replacement therapy to be successful.