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Involvement of normal prions in some human myelin diseases

We have experimentally demonstrated that cobalamin (Cbl) de ciency increases normal cellular prion (PrPC) levels in rat spinal cord (SC) and cerebrospinal uid (CSF), and decreases PrPC-mRNA levels in rat SC. Repeated intracerebroventricula administrations of anti-octapeptide repeat-PrPC-region antibodies to Cbl-de cient (Cbl-D) rats prevent SC myelin lesions, and the administrations of PrPCs to otherwise normal rats cause SC white matter lesions similar to those induced by Cbl de ciency. Cb positively regulates SC PrPC synthesis in rat by stimulating the local synthesis of epidermal growth factor (EGF), which also induce the local synthesis of PrPC-mRNAs, and down-regulating the local synthesis of tumor necrosis factor (TNF) preventing local PrPC overproduction. We have clinically demonstrated that PrPC levels are increased in the CSF of patients with sub-acut combined degeneration (SCD), unchanged in the CSF of patients with Alzheimer's disease and amyotrophic lateral sclerosis, ar decreased in the CSF and SC of patients with multiple sclerosis (MS), regardless of its clinical course. We conclude that SCD (hum and experimental) is a neurological disease due to excess PrPC without conformational change and aggregation, that the increase PrPC levels in SCD and Cbl-D polyneuropathy and their decrease in MS CNS make them antipodean myelin diseases in terms quantitative PrPC abnormalities, and that these abnormalities are related to myelin damage in the former, and impede myelin repai in the latter.

Biography

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+H ZRUNHG LQ VHYHUDO SRVLWLRQV DV IDFXOW\ RI ,QVWLWXWH RI *HQHUDO 3DWKRORJ\ DW 8QLYHUVLW\ R 3DWKRORJ\ DW 8QLYHUVLW\ RI 0LODQ IURP WR +H ZDV KRQRUHG DV $VVLVWDQW WR WKH &KDLUPDQ 0LODQ IURP WR +H KDV PRUH WKDQ 3XEOLFDWLRQV LQ KLJK LPSDFW MRXUQDOV

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