

JNS 2691

Selective Suppression of Neurofilament Antigen Expression in the Hypothyroid Rat Cerebral Cortex

Audrius V. Plioplys, Claude Gravel and Richard Hawkes

Laboratory of Neurobiology and Department of Biochemistry, Laval University, Quebec, P.Q. (Canada)

(Received 27 November, 1985)

(Revised, received 21 March, 1986)

(Accepted 21 March, 1986)

SUMMARY

As an integral component of the cytoskeleton neurofilaments play a central role in the establishment and maintenance of neuronal form. In particular, high neurofilament concentrations are characteristic of many classes of axons in the central nervous system. Isolated neurofilaments from rat brain consist of 3 distinct polypeptides with apparent molecular weights 210K, 160K and 68K. A murine monoclonal antibody, mabN210, has been produced which specifically recognizes an epitope associated with the high molecular weight subunit and this antibody has been used to explore the regulation of neurofilament expression during brain development. It has been shown that in the rat cerebellar cortex, the expression of mabN210-immunoreactivity in basket cell axons is severely suppressed in hypothyroidism while neurofilament antigen expression in other cerebellar axons seems not to require thyroid hormones. In view of the well-known cortical deficits in hypothyroidism, these studies have now been extended to include the developing rat cerebral cortex and selected cortical afferent and efferent axons. In hypothyroid rats there is a marked suppression of mabN210-immunoreactivity in the cerebral cortex and corpus callosum and, to a lesser extent, there is a reduction in staining in the internal capsule. By contrast, hypothyroidism did not reduce mabN210-immunoreactivity in the lateral olfactory tract or the stria medullaris. In rats, serum thyroid hormone starts to rise to adult levels on postnatal day 4. It appears that axons that have attained their mature distribution prior to the onset of thyroid hormone

Address correspondence to: Dr. R. B. Hawkes, Laboratoire de Neurobiologie, Hôpital de l'Enfant Jesus, 1401, 18e Rue, Quebec, Canada G1J 1Z4.

This work was supported by grants to R. H. from the Medical Research Council of Canada and the Fonds de Recherche en Paralyse Cérébrale, by a Medical Research Council of Canada fellowship to A. V. P. and by a F. C. A. C. studentship to C. G.