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Brain-derived neurotrophic factor (BDNF) and polysialylated-neural cell adhesion molecule (PSA-NCAM) in the human brainstem precerebellar nuclei from prenatal to adult age

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Occurrence and distribution of the neurotrophin Brain-derived neurotrophic factor (BDNF) and Polysialylated-neural cell adhesion molecule (PSA-NCAM), a neuroplasticity marker known to modulate BDNF signalling, were examined by immunohistochemistry in the human brainstem precerebellar nuclei at prenatal, perinatal and adult age. Western blot analysis performed in human brainstem showed for both molecules a single protein band compatible with the molecular weight of the dimeric form of mature BDNF and with that of PSA-NCAM. Detectability of both molecules up to 72 hours post-mortem was also assessed in rat brain. In neuronal perikarya, BDNF-like immunoreactivity (LI) appeared as intracytoplasmic granules, whereas PSA-NCAM-LI appeared mostly as peripheral staining, indicative of membrane labelling; immunoreactivity to both substances also labeled nerve fibers and terminals. BDNF- and PSA-NCAM-LI occurred in the external cuneate nucleus, perihypoglossal nuclei, inferior olive complex, arcuate nucleus, lateral reticular formation, vestibular nuclei, pontine reticulotegmental and paramedian reticular nuclei, and pontine basilar nuclei. With few exceptions, for both substances the immunostaining pattern detected at prenatal age persisted later on, though the staining intensity and density of immunoreactive structures appeared often higher in pre- and full-term newborns than in adult specimens. The results obtained suggest that BDNF operates in the development, maturation, maintenance and plasticity of human brainstem precerebellar neuronal systems. They also imply a multiple origin for the BDNF-LI of the human cerebellum. The codistribution of BDNF- and PSA-NCAM-LI in analyzed regions suggests that PSA-NCAM may modulate the functional interaction between BDNF and its high and low affinity receptors, an issue worth further analysis, particularly in view of the possible clinical significance of neuronal trophism in cerebellar neurodegenerative disorders.