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Neoplastic, infectious, and degenerative diseases modify HDAC3 expression in the human brain

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Altered expression of histone deacetylases (HDAC) may play a key role in the pathogenesis of a variety of neurological diseases. To determine whether neoplastic, infectious, and degenerative processes modify HDAC3 expression in the human brain, HDAC3 expression was defined by immunohistochemistry in sections of surgically removed tissue specimens and autopsy tissues from patients with glioblastoma, herpes simplex virus (HSV) encephalitis, Alzheimer's disease and controls. In brain tissue controls without evidence of neurological disease, neurons and ependymal cells demonstrated nuclear HDAC3 staining, weak to moderate nuclear HDAC3 staining was detected in a minority of astrocytes and oligodendroglial cells, and no HDAC3 expression was detected in vascular endothelial cells. In HSV encephalitis, significant nuclear HDAC3 expression was detected in reactive astrocytes, mononuclear inflammatory cells and some vascular endothelial cells. In Alzheimer's disease, there was focal decrease or loss of HDAC3 expression in neurons. In glioblastoma, neoplastic glial cells and endothelial cells of proliferated blood vessels demonstrated increased nuclear HDAC3 expression. These observations indicate that HDAC3 expression is modified in the human brain affected by infectious, degenerative and neoplastic processes and may lead to a better understanding of the role of HDAC3 in the pathogenesis of some of the clinically most significant neurological diseases.

Biography

Tibor Valyi-Nagy, MD, PhD, is Professor of Pathology and Director of Neuropathology at the University of Illinois at Chicago. He received clinical and research training at the Medical University of Debrecen, Hungary, University of Texas Medical Branch at Galveston, The Wistar Institute of Philadelphia and Vanderbilt University.

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