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Significant differences between augmentation of kynurenine aminotransferase I and kynurenine aminotransferase II activities in various types of brain pathology after HIV-1 infection

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Kynurenic acid (KYNA), an intermediate metabolite of L-kynurenine (L-KYN), is a competitive antagonist of inotropic excitatory amino acid (EAA) receptors and a non-competitive antagonist of 7 alpha nicotine cholinergic receptors and its involvement in memory deficit and cognition impairment has been suggested. The biosynthetic machinery of KYNA e.g. the content of L-KYN and KYNA, and the activity of enzymes synthesizing KYNA, kynurenine aminotransferases I (KAT I) and kynurenine aminotransferase II (KAT II) in the frontal cortex and cerebellum of HIV-1 infected patients in relation to different types of pathology were investigated. Pathologies were classified as follows: HIV in brain (HIV); opportunistic infection (OPP); infarction of brain (INF); malignant lymphoma of brain (LY); and glial dystrophy (GD); and of control subjects. Within investigated pathologies the most frequent pathology was OPP (65%), followed by HIV (26%), LY, INF, and GD (each 22%, respectively). Further, 68% of HIV-1 patients had bronchopneumonia and the utmost incidence of bronchopneumonia was seen in the OPP and LY group by 60%. KYNA was increased significantly in the frontal cortex of LY (392% of CO; $p<0.01$) and HIV (253% of CO; $p<0.01$), and in the cerebellum of GD (291% of CO; $p<0.05$). A significant increase of L-KYN was found only in the cerebellum of LY (333% of CO; $p<0.05$). The KAT I activity increased significantly in the frontal cortex of all pathological subgroups, i.e. OPP=433%> INF > LY > HIV > GD=182% of CO, respectively. In the cerebellum, too, all pathological subgroups showed marked increase of KAT I activity (OPP=326% > LY, HIV > GD > INF=181% of CO). On contrary, the activity of KAT II was moderately, but significantly, increased in the frontal cortex of INF and OPP; in the cerebellum of HIV, OPP and LY was comparable to control, while mildly reduced in INF and GD. Interestingly, normal subjects with the diagnosis of bronchopneumonia were characterized by high KYNA metabolism in the brain, too. The present study demonstrates a different pattern of alteration of KYNA metabolism in frontal cortex and cerebellum among investigated pathological subgroups of HIV-1 infected patients. Interestingly, a marked enhancement of KYNA metabolism in the brain has been found with occurrence of bronchopneumonia. This finding indicates a notable association between impaired conditions of oxygen availability and enhancement of KYNA formation in the human brain. These observation(s) might have an impact on the understanding of pathological processes in the brain after HIV-1 infection involving the development of neuropsychiatric and neurological symptoms including memory and cognition impairment. Our recently published data on D-cycloserine about lowering KYNA synthesisstrongly supports the significance of KYNA in the clinical manifestation of bronchopneumonia and AIDS encephalopathy.

Biography

Baran Halina is presently Head of the Karl Landsteiner Research Institute for Neurochemistry, Neuropharmacology, Neurorehabilitation and Pain Treatment Mauer, Lower Austria, from 2005-2011 she was the head of the Neurochemical Laboratory Karl Landsteiner Institute Mauer and Head of Neurophysiology, Veterinary Medical University Vienna. In the year 1998-2005, she was Assoc. Prof. at Institute of Pharmacology and Toxicology, Veterinary Medical University Vienna.

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