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Contributions of HIV infection in the hypothalamus and substance abuse/use to HPT dysregulation

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ver the last two decades, consequences of HIV infection of the CNS on disease severity and clinical neuropsychiatric manifestations have changed. These changes are due, in part, to improved control of peripheral infection by new anti-retroviral medications and more efficient CNS penetration of combination anti-retroviral therapies (cART). While the life spans of HIV-infected patients have been prolonged with successful cART, the spectrum of cognitive alterations observed in these patients has broadened. Recent studies report that there does not appear to be a single prototypical pattern of neuropsychological impairment associated with HIV, but includes diverse manifestations. Some comorbidities, such as substance abuse or depression likely play significant roles in the neuropsychiatric profiles of some HIV-infected patients. Newly recognized factors contributing to neurocognitive impairments include aging and unanticipated side effects from cART. Likewise, disturbances in neuroendocrine functioning are emerging as potentially important contributors to HIV-associated neurocognitive alterations. A retrospective review of clinical data from a small cohort of HIV-infected patients admitted to the psychiatric unit of an inner city hospital indicates that thyroid stimulating hormone levels were abnormal in 27% of the patients. Our data from analyses of post-mortem tissues from HIV patients show for the first time HIV infection of the hypothalamus and altered levels of thyroid hormone processing enzymes. Decreased vasopressin and oxytocin immunoreactivity in hypothalamic neurons was also observed. Thus, HIV infection of the CNS may contribute to changes in hypothalamic thyroid hormone signaling, thereby resulting in abnormal hypothalamic-pituitary-thyroid axis feedback and neuropsychiatric dysfunction.

Biography

Dr. Langford received her PhD from the University of Alabama in 1996 and completed post-doctoral training at the University of California San Diego School of Medicine in Infectious Diseases and Pathology. She joined the faculty at UCSD in the Department of Pathology in 2004, where she studied the effects of HIV infection in the CNS. In 2007, Dr. Langford joined the faculty at Temple University School of Medicine, Department of Neuroscience where she continues to study neurodegeneration in the context of HIV infection, substance abuse, and other CNS-related disorders.