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Interferon lambda family along with HTLV-1 proviral load, tax, and HBZ implicated in the pathogenesis of myelopathy/ tropical spastic paraparesis

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Abstract

Introduction: HTLV-1 associated myelopathy/ tropical spastic paraparesis (HAM/TSP) is a chronic neuro-inflammatory disease related to human T lymphotropic virus type 1 (HTLV-1) infection. Interferon type III (IFN-λ), which includes IL28, IL29, and IL28R, and affects the outcome of viral infections, might be complicated in the progression of HAM/TSP.

Material & Methods: The host-virus interactions in the manifestation of HAM/TSP were investigated, using IL28B, IL29, IL28R, HTLV-1 Tax, HTLV-1 basic leucine zipper factor (HBZ), and proviral load (PVL). The study groups consisted of 20 patients with HAM/TSP, 20 asymptomatic HTLV-1 carriers (ACs), and 20 healthy controls (HCs).

Results: The means of PVL, tax, and HBZ gene expressions in the HAM/TSP group ($p = 0.004$, 0.006, and < 0.0001, respectively) were significantly higher than in the AC group. The comparison of IL28B, IL29, and IL28R expression in the HAM/TSP, AC, and HC groups revealed no significant difference between the first 2, but lower concentrations in the HCs (IL28B: $p = 0.03$, 0.01; IL29: $p = 0.07$, 0.01; and IL28R: $p < 0.0001$, respectively). In the HAM/TSP group, correlations were seen between tax and HBZ ($R = 0.61$, $p = 0.004$) and between tax and IL29 ($R = 0.45$, $p = 0.04$). Negative correlations were observed between tax and IL28B ($R = -0.49$, $p = 0.02$) and between HBZ and IL28R ($R = -0.43$, $p = 0.06$). In the ACs, an inverse correlation was found between tax and IL28B ($R = -0.42$, $p = 0.06$).

Conclusion: These findings suggest that IL29, IL28B, and IL28R interfere in the infection of HAM/TSP, mainly via tax activation.

Biography

Hamid Reza Jahantigh is a PhD student in department of Veterinary Medicine in University of Bari, Italy. He has published 8 articles that have been cited about 12 times, and his H-index is 3. His main interest is in infectious diseases and works on interaction of pathogen and host.

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