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“Functional and Pathological Changes Associated with SIV-induced Peripheral Nerve Disease”

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Background: The most common form of HIV-induced neuropathy is Distal Sensory Polyneuropathy (DSP), which affects 30-35 % of patients with AIDS. To develop rational therapeutics for affected individuals, it is important to understand the mechanism by which HIV causes peripheral neuropathy. One of the major questions that would direct therapeutic efforts is whether DSP starts with injury to the distal nerves or whether damage of the neuronal cell bodies in dorsal root ganglia occurs first, with subsequent degeneration of the distal portion of their axons. We previously described in our SIV/macaque model lesions in the peripheral nervous system similar to those observed in HIV-infected patients with DSP, including loss of DRG sensory neurons.

Design: In this study we examined the relationship between dorsal root ganglia (DRG) damage and sensory nerve function by measuring C-fiber conduction velocities in the sural nerves and epidermal nerve fiber density in the skin of SIV-infected macaques and uninfected control macaques.

Results: SIV-infected macaques had significantly lower sensory C-fiber conduction velocity (CV) in the sural nerves than uninfected animals ($p = 0.01$). The extent of conduction velocity decline correlated strongly with the increase in macrophage infiltration in the DRG ($p = 0.006$). Although significant declines in density of epidermal fibers also developed in SIV-infected macaques ($p = 0.03$), ENF loss did not correlated with changes in the CV.

Conclusion: These findings suggest that primary injury to the neuronal in the DRG alters functional properties of sensory nerves in HIV-infected individuals and might be the initiating event in HIV-induced sensory neuropathy.