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# Attenuation of oxidative stress, inflammation and apoptosis by minocycline prevents retrovirus-induced neurodegeneration in mice.

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The ts1 mutant of the Moloney murine leukemia virus (MoMuLV) causes neurodegeneration in infected mice that resembles HIV-associated dementia. We have shown previously that ts1 infects glial cells in the brain, but not neurons. The most likely mechanism for ts1-mediated neurodegeneration is loss of glial redox support and glial cell toxicity to neurons. Minocycline has been shown to have neuroprotective effects in various models of neurodegeneration. This study was designed to determine whether and how minocycline prevents paralysis and death in ts1-infected mice. We show here that minocycline delays neurodegeneration in ts1-infected mice, and that it prevents death of cultured astrocytes infected by ts1 through attenuating oxidative stress, inflammation and apoptosis. Although minocycline reduces virus titers in the CNS of infected mice, it does not affect virus titers in infected mice thymi, spleens or infected C1 astrocytes. In addition, minocycline prevents death of primary neurons when they are cocultured with ts1-infected astrocytes, through mechanisms involving both inhibition of oxidative stress and upregulation of the transcription factor NF-E2-related factor 2 (Nrf2), which controls cellular antioxidant defenses. We conclude that minocycline delays retrovirus ts1-induced neurodegeneration involving antioxidant, anti-inflammation and anti-apoptotic mechanisms.

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