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Monosodium luminol upregulates the expression of Bcl-2 and VEGF in retrovirus-infected mice through downregulation of corresponding miRNAs.

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The retrovirus ts1 is a mutant of Moloney murine leukemia virus (MoMuLV) that causes neurodegeneration (ND) in susceptible mice. Our previous studies showed that the antioxidant drug monosodium luminol (GVT) prevented the development of ND in ts1-infected mice. In this study, we analyzed the effect of GVT on the expression of B-cell lymphoma-2 protein (Bcl-2) and vascular endothelial growth factor (VEGF) in central nervous system (CNS) tissues of these animals. Our data showed that GVT treatment of ts1-infected mice significantly increased their expression of Bcl-2 and VEGF in brainstem compared with ts1-infected untreated mice. We also studied the expression of specific microRNAs (miRNAs) such as miRNA-15 and -16 (targeting Bcl-2), and miRNA-20 (targeting VEGF). We found that the expression of miRNAs inversely correlated with the upregulation of their target proteins in ts1-infected untreated as well as in GVT-treated-ts1-infected mice. The data showed that GVT treatment prevented ts1-induced ND at least in part by upregulating Bcl-2 and VEGF expression, which likely occurred as a consequence of downregulation of their corresponding miRNAs. Keywords: ts1 virus; monosodium luminol; Bcl-2; VEGF; miRNAs.

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