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MS-275 has been developed as an anti-cancer drug and it generally shows speci city to type II. e ED_{50} of val is known to be very high and controlling its dosage is necessary for the treatment of disorders including epilepsy. Although MS-275 is e ective against cancer cells, normal cells are also killed. We previously reported that nur77 gene expression via the PKA-mediated signaling pathway promoted neurite outgrowth in PC12 cells [15]. Recently, we also proposed that K-350, which is one of the 2-aminobenzamide-type HDAC inhibitors, may promote neurite outgrowth with histone acetylation using PC12 cells