Neuroinflammation linked to L-dopa induced dyskinesia (LID)

San Diego, September 30, 2016 — NeurMedix, Inc., a clinical-stage biopharmaceutical company that engages in developing products for the treatment of neurological and neuro-degenerative disorders, today announced that Neuroinflammation linked to L-dopa induced dyskinesia (LID).

• According to a publication in Experimental Neurology today (Mulas, et al, Exp Neurol. 2016 Dec;286:83-92), daily administration of levodopa (L-DOPA) to animals with Parkinson’s disease (PD) increases neuroinflammation (activation of microglia, cytokines such as tumor necrosis factor alpha, and oxidative stress caused by inducible nitric oxide synthase) leading to levodopa-induced dyskinesia (LID). The article, published by Dr. Anna Carta and coworkers, “Differential induction of dyskinesia and neuroinflammation by pulsatile versus continuous L-DOPA delivery in the 6-OHDA model of Parkinson’s disease” concludes that neuroinflammation is to blame. The authors found increased microglial activation, pro-inflammatory cytokine production (tumor necrosis factor alpha [TNFa]) and inducible nitric oxide synthase (iNOS, responsible for increased oxidative stress) in rats with PD given pulsatile levodopa. This procedure that is the gold standard for treating PD, is known to generate oxidative stress producing metabolites that can increase neuroinflammation and progression of PD.

• Significance (Christopher Reading, Ph.D., Chief Scientific Officer): This work adds strength to the anti-inflammatory mechanism of NE3107 in PD and its activity in decreasing LID in the marmoset model of PD and LID. NeurMedix has demonstrated significant preservation of neurons in two studies funded by the Michael J Fox Foundation. In both mice and marmosets with PD we found significant improvement in disease scores for PD, and when LID was induced in marmosets, NE3107 treatment decreased the severity of LID. In the mouse model, where neuroinflammation was analyzed, NE3107 significantly decreased inducible nitric oxide synthase (iNOS), tumor necrosis factor alpha (TNFa) and interleukin 1 beta (IL-1b).