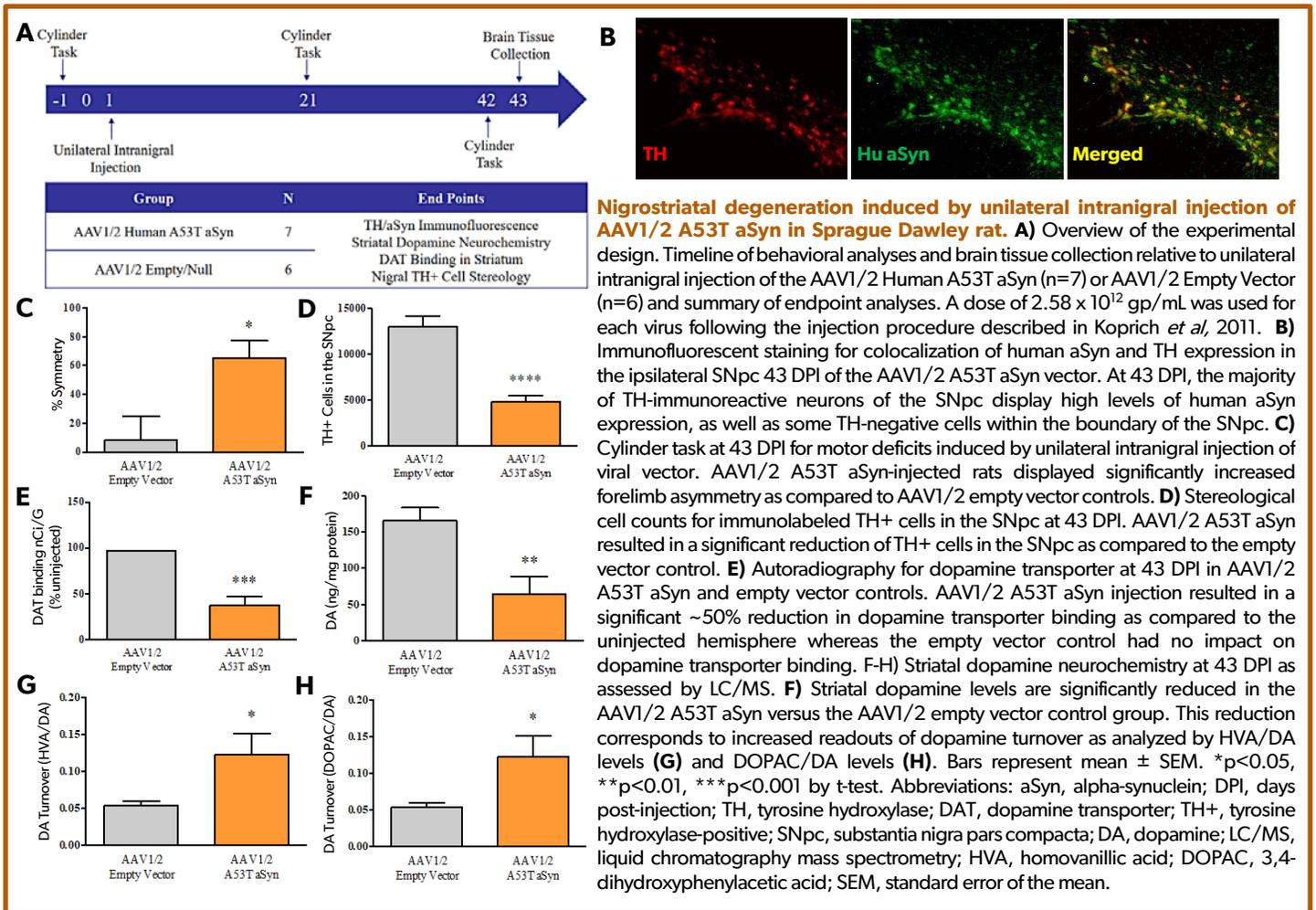


## HUMAN A53T ALPHA-SYNUCLEIN VIRAL VECTOR

MJFF has partnered with GeneDetect and Vigene Biosciences to make available the well-characterized adeno-associated viral vector expressing human A53T alpha-synuclein to model Parkinson's disease. This viral vector has been characterized in the mouse, rat, and non-human primate for ability to express alpha-synuclein, induce nigrostriatal degeneration, and model Parkinson's disease pathology (see Publications section). The current lot has been validated to ensure performance similar to the previously published batches. Viral vectors were designed and generated by GeneDetect, validated by Atuka, Inc., and are now available for purchase at [Vigene Biosciences](http://VigeneBiosciences.com). Validation data is included below.

Transgene	Viral Vector Nomenclature	Catalog #
Human A53T aSyn	AAV1/2-CMV/CBA- <b>Human A53T aSyn</b> -WPRE-BGH-polyA	GD1001-RV
Empty Vector Control	AAV1/2-CMV/CBA- <b>Null/Empty</b> -WPRE-BGH-polyA	GD1004-RV



**Publications:**

- Koprich *et al.* (2010). Expression of human A53T alpha-synuclein in the rat substantia nigra using a novel AAV1/2 vector produces a rapidly evolving pathology with protein aggregation, dystrophic neurite architecture, and nigrostriatal degeneration with potential to model the pathology of Parkinson's disease. *Molecular Neurodegeneration*, 5:43.
- Koprich *et al.* (2011). Progressive neurodegeneration or endogenous compensation in an animal model of Parkinson's disease produced by decreasing doses of alpha-synuclein. *PLoS ONE*, 6(3): e17698.
- He *et al.* (2015). Treatment with trehalose prevents behavioral and neurochemical deficits produced in an AAV alpha-synuclein rat model of Parkinson's Disease. *Molecular Neurobiology*, 53(4): 2258-2268.
- Koprich *et al.* (2016). Towards a non-human primate model of alpha-synucleinopathy for development of therapeutics for Parkinson's disease: optimization of AAV1/2 delivery parameters to drive sustained expression of alpha-synuclein and dopaminergic degeneration in Macaque. *PLoS ONE*, 11(11): e0167235.
- Ip *et al.* (2017). AAV1/2-induced overexpression of A53T-alpha-synuclein in the substantia nigra results in degeneration of the nigrostriatal system with Lewy-like pathology and motor impairment: a new mouse model for Parkinson's disease. *Acta Neuropathologica Communications*, 5:11.
- Musacchio *et al.* (2017). Subthalamic nucleus deep brain stimulation is neuroprotective in the A53T alpha-synuclein Parkinson's disease rat model. *Annals of Neurology*, 81(6): 825-836.
- Gleave *et al.* (2017). Sirtuin 3 rescues neurons through the stabilisation of mitochondrial biogenetics in the virally-expressing mutant alpha-synuclein rat model of parkinsonism. *Neurobiology of Disease*, 106: 133-146.