

JOINT EVENT

4<sup>th</sup> International Conference on **Epilepsy & Treatment**  
&  
4<sup>th</sup> World Congress on **Parkinsons & Huntington Disease**  
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**VRK2 mRNA stability and polyQ-huntingtin aggregation**

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Misfolded proteins with abnormal polyglutamine (polyQ) expansion cause neurodegenerative disorders, including Huntington's disease (HD). Recently, it was found that polyQ aggregates accumulate due to vaccinia-related kinase 2 (VRK2)-mediated degradation of TCP-1 ring complex (TRiC)/chaperonin-containing TCP-1 (CCT), which has an essential role in the prevention of polyQ protein aggregation and cytotoxicity. The levels of VRK2 are known to be much higher in actively proliferating cells but are maintained at a low level in the brain via an unknown mechanism. Here, we found that basal levels of neuronal cell-specific VRK2 mRNA are maintained by post-transcriptional, rather than transcriptional, regulation. Moreover, heterogeneous nuclear ribonucleoprotein Q (hnRNP Q) specifically binds to the 3' UTR of VRK2 mRNA in neuronal cells to reduce the mRNA stability. As a result, we found a dramatic decrease in CCT4 protein levels in response to a reduction in hnRNP Q levels, which was followed by an increase in polyQ aggregation. Taken together, these results provide new insights into how neuronal hnRNP Q decreases VRK2 mRNA stability and contribute to the prevention of HD, while also identifying new prognostic markers of HD.

**Biography**

Kim K T completed his PhD from the University of Massachusetts, Amherst in 1989. During 1982–1985, he worked as a Research Investigator at Genetic Engineering Research Center, Korea Institute of Science and Technology. From 1997–1998, he was a Visiting Scientist at the Department of Physiology & Biophysics at the University of Washington. His major stream of research involves the modulation of second messengers in neuronal cells, regulation of neurotransmitter secretion, and transcriptional regulation of the enzymes involved in the biosynthesis of neurotransmitters.

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