

# Casein kinase 2 interacts with and phosphorylates ataxin-3

Rui-Song TAO<sup>1,2</sup>, Er-Kang FEI<sup>1,\*</sup>, Zheng YING<sup>1</sup>, Hong-Feng WANG<sup>1</sup>, Guang-Hui WANG<sup>1</sup>

<sup>1</sup>Laboratory of Molecular Neuropathology, Hefei National Laboratory for Physical Sciences at Microscale and School of Life Sciences, University of Science and Technology of China, Hefei 230027, China

<sup>2</sup>Department of Biology, Hefei Teaching College, Hefei 230061, China

\*Corresponding author

E-mail:ericfee@ustc.edu.cn

**Abstract: Objective** Machado-Joseph disease (MJD)/Spinocerebellar ataxia type 3 (SCA3) is an autosomal dominant neurodegenerative disorder caused by an expansion of polyglutamine tract near the C-terminus of the *MJD1* gene product, ataxin-3. The precise mechanism of the MJD/SCA3 pathogenesis remains unclear. A growing body of evidence demonstrates that phosphorylation plays an important role in the pathogenesis of many neurodegenerative diseases. However, few kinases are known to phosphorylate ataxin-3. The present study is to explore whether ataxin-3 is a substrate of casein kinase 2 (CK2). **Methods** The interaction between ataxin-3 and CK2 was identified by glutathione S-transferase (GST) pull-down assay and co-immunoprecipitation assay. The phosphorylation of ataxin-3 by CK2 was measured by *in vitro* phosphorylation assays. **Results** (1) Both wild type and expanded ataxin-3 interacted with CK2 $\alpha$  and CK2 $\beta$  *in vitro*. (2) In 293 cells, both wild type and expanded ataxin-3 interacted with CK2b, <http://precision-health.sibs.ac.cn/csn2019/abstract.php> but not CK2a. (3) CK2 phosphorylated wild type and expanded ataxin-3. **Conclusion** Ataxin-3 is a substrate of protein kinase CK2.

**Keywords:** Machado-Joseph disease/spinocerebellar ataxia type 3; ataxin-3; casein kinase 2; phosphorylation

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