Casein kinase 2 interacts with and phosphorylates ataxin-3

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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-immunoprecipition 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 CK2α and CK2β *in vitro*. (2) In 293 cells, both wild type and expanded ataxin-3 interacted with CK2b, http://precision-health.sibs.ac.cn/csn2019/abstract.phpbut 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

标题：3 号 Times New Roman

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