**Abstract**

To promote the participation in academic communication, this conference will set up poster and oral presentation. The Organizing Committee will select the poster and oral presentation (winners of award for excellence in neuromodulation) from the abstracts, and the conference committee will inform the acceptance of the submission after review.

(1)   The content should be not officially published before March 2024.

(2)   The deadline for submission is 18th March, 2024, and overdue abstracts will not be accepted for presentation.

      (3) Submission website: <https://meeting.cns.org.cn/Neurotechnology/>.

     (4) Please complete the registration for attendance, and then log in to your user account and click on "User Center" to add or modify your abstract.

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This award is open to first authors as undergraduate, graduate student, or postdoctoral researcher. To apply, please complete the registration for attendance, and then submit your resume along with a letter of recommendation to **cnsnm2024@outlook.com**. Please ensure that the subject line is clearly marked as 'CNSNM2024 Award for Excellence in Neuromodulation'.

All award recipients will be expected to deliver a 5-minute oral presentation at the conference on 27th April. This presentation opportunity serves to further showcase their work and facilitate networking among peers and professionals in their respective fields.

More information can be found on website: <https://meeting.cns.org.cn/Neurotechnology/>.

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* **Whether or not to participate in the poster presentation or award for excellence in neuromodulation?**

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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