Photo

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**Brief biography** (200 words maximum):

Guoqiang Xu received his BSc (1996) and MSc (1999) from Fudan University, Shanghai, and PhD (2002) from the University of Akron, Ohio, USA. He worked as a postdoctoral associate and research assistant professor at the Department of Chemistry and Chemical Biology and Weill Medical College of Cornell University. He developed several proteomic approaches to profile protein ubiquitination and proteolytic processing. He then joined the College of Pharmaceutical Sciences, Soochow University in 2013 as a professor. He is the editorial board of BMC Genomics (proteomic sections). His research interest is to investigate the roles of ubiquitin proteasome system and ubiquitin-like modification in the regulation of neuronal development and cancer. He has published more than 100 papers in journals such as Oncogene, J Biol Chem, Hum Mol Genet, Nat Biotechnol, Nat Commun.

**Abstract (~200 words):**

**Role of the E3 ligase substrate receptor cereblon on neuronal development**

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Mental retardation accounts for about 1~3% of the world population and the majority of them are caused by genetic defects. Recently, mutations in the cereblon (CRBN) encoding gene were found in children with mental retardation. Although it is well-known that CRBN forms an E3 ubiquitin ligase complex with Cullin 4-RING E3 ligase (CRL4-CRBN), the precise molecular mechanism by which CRBN and its mutants regulate neuronal development and intelligent disability remains unknown. Here, using quantitative proteomics, we identified many novel substrates of this E3 ligase. Through biochemical and animal work, we found that CRBN regulates neurite outgrowth by promoting the ubiquitination and downregulating its substrates. However, the missense mutation differentially regulates a specific substrate and thus the neurite outgrowth. Our work demonstrates the phenotype of the CRBN deficiency mice and elucidates the molecular mechanism by which CRBN mutation causes defect in neuronal development.