

## OPEN PEER REVIEW REPORT 2

**Name of journal:** Neural Regeneration Research

**Manuscript NO:** NRR-D-20-00092

**Title:** Oxidative stress battles neuronal Bcl-xL in a fight to the death

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**Reviewer's country:** Spain

**Date sent for review:** 2020-02-25

### COMMENTS TO AUTHORS

In this article (invited paper) titled "Oxidative Stress Battles Neuronal Bcl-xL in a Fight to the Death" authors have gathered relevant and current information from several studies (some of them from their lab) regarding Neuronal Bcl-xL. They have concluded that Bcl-xL is a key protein that enhances neuronal function by regulating energy metabolism, neurotransmission, and the survival or death of neurons. Besides. They also describe how the activity and abundance of Bcl-xL is controlled by oxidative stress: ROS control the post-translational modification of Bcl-xL including its proteolytic cleavage and residue phosphorylation. Moreover, they pointed out that some specific protein modifications can alter the availability of functional Bcl-xL and its activity. On the other hand, they focused on some previous studies that have shown how ROS also play a part in the transcriptional regulation of Bcl-xL. Therefore, they affirm that understanding ROS-mediated Bcl-xL alterations should be important to further elucidate mechanisms of ROS-associated brain disease, and approaches that manipulate ROS may be an effective way to manipulate Bcl-xL function and abundance in the brain.

I consider as a major improvement of this article, in order to be better understood the text by readers, to create a figure that summarizes most of the processes that have been describe in the manuscript.

Not further actions should be done in the text from my point of view.