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PII: S1357-2725(17)30265-0
DOI: https://doi.org/10.1016/j.biocel.2017.10.007
Reference: BC 5236

To appear in: *The International Journal of Biochemistry & Cell Biology*

Received date: 18-7-2017
Revised date: 10-10-2017
Accepted date: 13-10-2017

Please cite this article as: Zhao, Xinyuan., Xing, Fengjun., Cong, Yewen., Zhuang, Yin., Han, Muxi., Wu, Zhiqiang., Yu, Shali., Wei, Haiyan., Wang, Xiaoke., & Chen, Gang., Antimony trichloride induces a loss of cell viability via reactive oxygen species-dependent autophagy in A549 cells.*International Journal of Biochemistry and Cell Biology* https://doi.org/10.1016/j.biocel.2017.10.007

Antimony trichloride induces a loss of cell viability via reactive oxygen species-dependent autophagy in A549 cells

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Highlights

- Antimony trichloride activates autophagy in A549 cells.
- Antimony trichloride enhances autophagic flux and p62 gene expressions.
- Antimony trichloride induces autophagy through ROS in A549 cells.
- Inhibition of autophagy attenuates Sb-induced A549 cell viability loss.

Abstract

Antimony (Sb) is one of the most prevalent heavy metals and frequently leads to biological toxicity. Although autophagy is believed to be involved in metal-associated cytotoxicity, there is no evidence of its involvement following exposure. Moreover, the underlying mechanism of autophagy remains unclear. In this study, treatment with antimony trichloride caused autophagy in a dose- and time-dependent manner in A549 cells but did not affect the level of *Atg5* or *Atg7* mRNA expression. Furthermore, Sb enhanced autophagic flux while upregulating p62 gene and protein levels. The classic mechanistic target of rapamycin (mTOR) pathway is not involved in Sb-induced autophagy. However, Sb-induced autophagy and the upregulation of p62 were inhibited by treatment with the antioxidant N-acetylcysteine (NAC). Subsequent analyses demonstrated that the inhibition of autophagy protected A549 cells from a loss of cell viability, while the activation of autophagy by rapamycin had the opposite effect. These data suggest that reactive oxygen species-dependent autophagy mediates