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## ACCEPTED MANUSCRIPT

# 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

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