Glutathione Depletion Induces Spermatogonial Cell Autophagy

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Resumen

The development and survival of male germ cells depend on the antioxidant capacity of the seminiferous tubule. Glutathione (GSH) plays an important role in the antioxidant defenses of the spermatogenic epithelium. Autophagy can act as a pro-survival response during oxidative stress or nutrient deficiency. In this work, we evaluated whether autophagy is involved in spermatogonia-type germ cell survival during severe GSH deficiency. We showed that the disruption of GSH metabolism with l-buthionine-(S,R)-sulfoximine (BSO) decreased reduced (GSH), oxidized (GSSG) glutathione content, and GSH/GSSG ratio in germ cells, without altering reactive oxygen species production and cell viability, evaluated by 2,7-dichlorodihydrofluorescein (DCF) fluorescence and exclusion of propidium iodide assays, respectively. Autophagy was assessed by processing the endogenous protein LC3I and observing its sub-cellular distribution. Immunoblot and immunofluorescence analysis showed a consistent increase in LC3II and accumulation of autophagic vesicles under GSH-depletion conditions. This condition did not show changes in the level of phosphorylation of AMP-activated protein kinase (AMPK) or the ATP content. A loss in S-glutathionylated protein pattern was also observed. However, inhibition of autophagy resulted in decreased ATP content and increased caspase-3/7 activity in GSH-depleted germ cells. These findings suggest that GSH deficiency triggers an AMPK-independent induction of autophagy in germ cells as an adaptive stress response. J. Cell. Biochem. 116: 2283-2292, 2015. (c) 2015 Wiley Periodicals, Inc.

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