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J Bioenerg Biomembr. 2012 Feb;44(1):51-60. doi: 10.1007/s10863-012-9411-x. Epub 2012 Feb 9.

Bromopyruvate mediates autophagy and cardiolipin degradation to monolyso-cardiolipin in GL15 glioblastoma cells.

Davidescu M, Sciaccaluga M, Macchioni L, Angelini R, Lopalco P, Rambotti MG, Roberti R, Corcelli A, Castigli E, Corazzi L.

Source

Department of Internal Medicine, Laboratory of Biochemistry, University of Perugia, Via del Giochetto, 06122 Perugia, Italy.

Abstract

The GL15 glioblastoma cell line undergoes viability loss upon treatment with bromopyruvate. The biochemical mechanisms triggered by the antiglycolytic agent indicate the activation of an autophagic pathway. Acridine orange stains acidic intracellular vesicles already 60 min after bromopyruvate treatment, whereas autophagosomes engulfing electron dense material are well evidenced 18 h later. The autophagic process is accompanied by the expression of the early autophagosomal marker Atg5 and by LC3-II formation, a late biochemical marker associated with autophagosomes. In agreement with the autophagic route activation, the inhibitory and the activator Akt and ERK signaling pathways are depressed and enhanced, respectively. In spite of the energetic collapse suffered by bromopyruvate-treated cells, MALDI-TOF mass spectrometry lipid analysis does not evidence a decrease of the major phospholipids, in accordance with the need of phospholipids for autophagosomal membranes biogenesis. Contrarily, mitochondrial cardiolipin decreases, accompanied by monolyso-cardiolipin formation and complete cytochrome c degradation, events that could target mitochondria to autophagy. However, in our experimental conditions cytochrome c degradation seems to be independent of the autophagic process.

PMID:
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- Cardiolipins/metabolism*
- Cell Line, Tumor
- Cell Survival/drug effects*
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- Microscopy, Electron, Transmission
- Microtubule-Associated Proteins/metabolism

- Pyruvates/pharmacology*
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Substances

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