BCL-2 family: integrating stress responses at the ER to control cell demise

Por: Pihan, P (Pihan, Philippe)[1,2,3] ; Carreras-Sureda, A (Carreras-Sureda, Amado)[1,2,3] ; Hetz, C (Hetz, Claudio)[1,2,3,4,5]

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Resumen

In the last decade, the endoplasmic reticulum (ER) has emerged as a central organelle regulating the core mitochondrial apoptosis pathway. At the ER membrane, a variety of stress signals are integrated toward determining cell fate, involving a complex cross talk between key homeostatic pathways including the unfolded protein response, autophagy, calcium signaling and mitochondrial bioenergetics. In this context, key regulators of cell death of the BCL-2 and TMBIM/BI-1 family of proteins have relevant functions as stress rheostats mediated by the formation of distinct protein complexes that regulate the switch between adaptive and proapoptotic phases under stress. Here, we overview recent advances on our molecular understanding of how the apoptotic machinery integrates stress signals toward cell fate decisions upstream of the mitochondrial gateway of death.

Palabras clave

KeyWords Plus: ENDOPLASMIC-RETICULUM STRESS; UNFOLDED-PROTEIN-RESPONSE; MITOCHONDRIAL PERMEABILITY TRANSITION; CYTOCHROME-C RELEASE; OUTER-MEMBRANE PERMEABILIZATION; ORGANELLE-SPECIFIC INITIATION; BAX INHIBITOR-1; CYCLOPHILIN-D; MEMBER BOK; PROAPOPTOTIC BAX

Información del autor

Dirección para petición de copias: Hetz, C (autor para petición de copias)

+ Univ Chile, Biomed Neurosci Inst, Fac Med, Av Independencia 1027 Bloque B POB, Santiago 70086, Chile

Direcciones:

+ [1] Univ Chile, Biomed Neurosci Inst, Fac Med, Av Independencia 1027 Bloque B POB, Santiago 70086, C
+ [2] Univ Chile, Inst Biomed Sci, Program Cellular & Mol Biol, Santiago, Chile
+ [3] Univ Chile, Ctr Gerosci Brain Hlth & Metab, Fac Med, Santiago, Chile

[4] Buck Inst Res Aging, Novato, CA 94945 USA

[5] Harvard Sch Publ Hlth, Dept Immunol & Infect Dis, Boston, MA 02115 USA

Direcciones de correo electrónico:chetz@med.uchile.cl

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