hock proteins 27 and 70: anti-apoptotic proteins with tumorigenic pro

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Abstract

Heat shock proteins (HSP) HSP27 and HSP70 are expressed in response to a wide variety of physiological and environmental insults including anticancer chemotherapy, thus allowing the cell to survive to lethal conditions. Several mechanisms account for the cytoprotective effect of HSP27 and HSP70. (1) Both proteins are powerful chaperones. (2) They both inhibit key effectors of the apoptotic machinery at the pre and post-mitochondrial level. (3) They participate in the proteasome-mediated degradation of proteins under stress conditions, thereby contributing to the so called "protein triage". In cancer cells, the expression of HSP27 and/or HSP70 is abnormally high, and both HSP27 and HSP70 may participate in oncogenesis and in resistance to chemotherapy. In rodent models, HSP27 or HSP70 over-expression increases tumor growth and metastatic potential. The depletion or inhibition of HSP27 and HS70 frequently reduces the size of the tumors and even can cause their complete involution (for HSP70). Therefore, the inhibition of HSP70 and HSP27 has become a novel strategy of cancer therapy.

See original abstract

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