Is HSP27 a Key Molecule or a Biomarker of Cancers?

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Heat Shock Protein 27 (HSP27) and Anti-Apoptosis Activity in Cancer Cells

A molecular chaperone, heat shock protein 27 (HSP27, heat-shock 27-KD protein 1, HSPB1) is one of the small heat shock protein family. It modulates the ability of cells to respond to several types of injury, heat shock, oxidative stress and other stresses. HSP27 is expressed in almost all organisms from prokaryotes to mammals. It interacts with many proteins and can prevent a wide variety of apoptotic agents from causing cell death. HSP27 regulates apoptosis by interacting with key components of the apoptotic signaling pathway [1]. It was reported that HSP27 inhibited cytochrome c and dATP triggered activation of procaspase-9 and prevented etoposide-induced apoptosis [2], and HSP27 altered the expression of topoisomerase II and inhibited doxorubicin-induced apoptosis [3]. Furthermore, it was reported that over-expression of HSP27 in prostate cancer cells rendered cells resistant to etoposide-, diethylmaleate-, cycloheximide- or radiation-induced apoptosis, which may be mediated by the production of survival factors [4]. From our recent studies, up-regulation of HSP27 in pancreatic cancer cells has been clarified to be linked to the resistance to gemcitabine (GEM), and the down-regulation of HSP27 by using HSP27 inhibitors; siRNA for HSP27, interferon γ or KNK437 in GEM-resistant cells showed the increasing sensitivity for GEM [5-8]. They showed that the up-regulation of anti-apoptotic pathways induced by HSP27 enhanced the resistance of cancer cells to apoptosis. Enhanced resistance to apoptosis in cancer cells induced by HSP27 may be caused by many factors. HSP27 protects the cells from apoptosis by concerning with DAXX, Bid, cytochrome c, IKK, caspase-3 and etc. [9,10]. For the chemotherapies which aim at the induction of apoptosis in cancer cells, it is very important to control such factors concerning with HSP27 or itself.

HSP27 and Cancers

In these days many reports about the up-regulation of HSP27 in cancer tissues of stomach, head and neck, renal, prostate and etc have been published [11-14]. Why is HSP27 up-regulated in cancer cells? Song et al. [15] showed that constitutively activated signal transducer and activator of transcription 3 (STAT 3) up-regulated HSP27 in breast cancer tissues of stomach, head and neck, renal, prostate and etc, have been published [11-14]. Why is HSP27 up-regulated in cancer cells? The reason may be the resistance of cancer cells to the cytotoxic effect of gemcitabine. Pancreas 38: 224-226.

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References


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