



Overexpression of HSP27 in cultured human aortic smooth muscular cells reduces apoptosis induced by low-frequency and low-energy ultrasound by inhibition of an intrinsic pathway

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ABSTRACT. We investigated *in vitro* the effect of low-frequency and low-energy ultrasound (LFLEU) on apoptosis of an overexpressed HSP27 human aortic smooth muscle cell (HASMC) line. A frequency of 42.6 kHz was used in all experiments. HASMC were exposed to ultrasound and cell viability was evaluated by MTT reduction. Overexpressed HSP27-HASMC was constructed on a pcDNA3.1 vector. Apoptosis was determined 24 h after treatment by flow cytometry; gene display was evaluated with Affimax chips, and HSP27 mRNA and protein expression levels were measured by RT-PCR and Western blotting. The apoptosis rate (at 30 s) was significantly lower in

HASMC transfected with HSP27 ($7.14 \pm 1.73\%$), compared with cells transfected with a mock plasmid ($17.31 \pm 2.72\%$) or a control group ($14.23 \pm 2.77\%$), indicating a protective function for apoptosis induced by LFLEU. Gene display analysis showed that caspase-9 expression in HSP27 cell lines was downregulated and caspase-3 upregulated. However, RT-PCR and Western blotting analysis indicated that both caspase-9 and caspase-3 were inhibited at both the mRNA and protein levels. We suggest that overexpressed HSP27 is capable of protecting the LFLEU from apoptosis and that the pathway for this protection is via downregulated caspase-9 and caspase-3 expression.

Key words: HSP27; Human aortic smooth muscle cell; Ultrasound; Overexpression