
mutation or overexpression increases the aggressiveness of tumor cells and enhances resistance to PTX. We investigated the mechanism of PTX-induced apoptosis in p53-positive HSC-GT3 gastric cancer cells, in which loss of p53 function may be a key factor for resistance to PTX. HSC-GT3 cells were treated with PTX, and the apoptotic signal was evaluated by fluorescence microscopy and flow cytometry. We also examined the change in the p53 pathway and mitochondrial membrane potential ($\Delta\Psi(m)$). Expression of Bcl-2 and Bax and the release of cytochrome c from mitochondria were measured by western blot analysis. DNA fragmentation was detected by a morphologic and flow cytometric evaluation. Incubation with PTX induced caspase-dependent apoptosis in HSC-GT3 cells. Expression of p53 was induced by PTX, and it was associated with the early inhibition of G(2)-M phase transition. The loss of $\Delta\Psi(m)$ was detected in HSC-GT3 cells treated with PTX. The Bcl-2-binding proteins Bax and Bak were activated by PTX, as evidenced by increased Bax/Bcl-2 ratio, and cytochrome c was released from mitochondria to the cytosol. Expression of Bcl-2 and Bcl-X(L) was decreased in HSC-GT3 cells treated with PTX. These results demonstrate that the loss of $\Delta\Psi(m)$ and cell cycle arrest by PTX are associated with Bax activation and apoptosis, resulting in the loss of Bcl-2 and Bcl-X(L), as well as cytochrome c release. The p53-dependent apoptotic signal may contribute to the mechanism of PTX-induced apoptosis in gastric cancer cells with p53 mutations. The present invention relates generally to the control of devices that operate in a hostile environment and, more particularly, to a computerized system for the control of an actuator in a hostile environment.

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