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Enhanced potency of daunorubicin against multidrug resistant subline KB-ChR-8-5-11 by a pulsed magnetic field.

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Tumor cell resistance to many unrelated anticancer drugs is a major obstacle during cancer chemotherapy. One mechanism of drug resistance is thought to be due to the efflux of anticancer drugs caused by P-glycoprotein. In recent years, magnetic fields have been found to enhance the potency of anticancer drugs, with favorable modulation of cancer therapy. In this study, KB-ChR-8-5-11, a multidrug resistant (MDR) human carcinoma subline, was used as a model to evaluate the ability of pulsed magnetic fields (PMF) to modulate the potency of daunorubicin (DNR) in vivo and to determine the appropriate order of exposure to drugs and PMF using an in vitro cytotoxicity assay. Solenoid coils with a ramped pulse current source were used at 250 pulses per second for both in vivo and in vitro experiments. For the in vivo study, KB-ChR-8-5-11 cells were inoculated into thymic Balbc-nu/nu female mice. Treatment was begun when the average tumor volume reached 250-450 mm³. Treatment consisted of whole body exposure to PMF for one hour, followed immediately by intravenous (i.v.) injection of 8 mg/kg DNR designated as day 0, and repeated on days 7 and 14. Among the various groups, significant differences in the tumor volume were found between PMF + saline and PMF + DNR groups ($p = 0.0107$) at 39 days and 42 days ($p = 0.0101$). No mice died in the PMF alone group, and no toxicity attributable to PMF was found during the experimental period. For the in vitro studies, the sulforhodamine blue (SRB) cytotoxicity assay was used to determine the effect of the sequence which cells are exposed to PMF and/or DNR. Cells were exposed to PMF either before (pre-PMF) or after (post-PMF) drug was added. Results showed that the IC₅₀ was significantly different between controls and pre-PMF + DNR groups ($P = 0.0096$, $P = 0.0088$). The IC₅₀ of the post-PMF + DNR group was not found to be significantly different from control groups. Thus, the data in this report demonstrates that PMF enhanced

the potency of DNR against KB-ChR-8-5-11 xenograft in vivo, while the efficacy of DNR was potentiated in vitro by PMF exposure only when PMF exposure occurred in the presence of drug. The data in vitro suggest that the mechanism by which PMFs modulate DNR's potency may be by inhibition of the efflux pump, P-glycoprotein. Further work to determine conditions for maximum modulation of drug potency by PMFs is warranted.

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