metabolites pharmacodynamics and pharmacokinetics of tamoxifen

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Unable to display preview. The transport rate k42 6. Generate a file for use with external citation management software. The emphasis will be on publishing quality papers very rapidly. Gov't Research Support, U. Nevertheless, personalized treatment of tamoxifen based on genotyping has not yet met consensus. Find all citations in this journal default. The aim of this study was to develop a pharmacokinetic-pharmacodynamic model to predict the distribution of tamoxifen and endoxifen quantitatively, and to discover the anti-tumor effect patterns of tamoxifen and endoxifen. Abstract Potential mechanisms for tamoxifen resistance include loss or alteration in estrogen receptor or other transcription factors and altered tamoxifen pharmacology. Protein Families Show all items. Cytochrome Pcatalyzed N-demethylation and 4-hydroxylation. No matching affiliation detected. However, high interindividual variability in response is observed, calling for a personalized approach to tamoxifen treatment. We established a PK-PD model of tamoxifen and endoxifen to predict the tumor growth. Remember Login Login reminder. Peak levels of tamoxifen occurred hr after oral administration of tamoxifen in both species, whereas peak levels of N-desmethyltamoxifen in the immature rat did not occur until hr. Research Article Publication date:Drug Metab Dispos. Jan-Feb;19(1) Metabolites, pharmacodynamics, and pharmacokinetics of tamoxifen in rats and mice compared to the breast cancer patient. Robinson SP(1), Langan-Fahey SM, Johnson DA, Jordan VC. Author information: (1)Department of Human Oncology, University of Wisconsin Clinical. The metabolism of tamoxifen was examined in the rat, mouse, and human breast cancer patient. Large oral doses of tamoxifen (mg/kg) in the immature ovariectomized rat and mature mouse produced circulating levels of the parent compound, N-desmethyltamoxifen, and 4-hydroxytamoxifen quantifiable by HPLC. Dec 20, - The metabolism of tamoxifen was examined in the rat, mouse, and human breast cancer patient. Large oral doses of tamoxifen (mg/kg) in the immature ovariectomized rat and mature mouse produced circulating levels of the parent compound, N-desmethyltamoxifen, and 4-hydroxytamoxifen. Feb 2, - uv activation, and fluorescence detection. N-Desmethyltamoxifen and 4-hydroxytamoxifen serum levels in the mature ovariectomized mouse paralleled tamoxifen levels throughout a hr time course after a single dose of tamoxifen. On the other hand, N-desmethylta- moxifen was the predominant serum. Pharmacodynamics of Tamoxifen and Its 4-Hydroxy and N-Desmethyl Metabolites: Activation of Caspases and Induction of Apoptosis in Rat Mammary Tumors and in .. Robinson S. P., Langan-Fahey S. M., Johnson D. A., Jordan V. C. Metabolites, pharmacodynamics, and pharmacokinetics of tamoxifen in rats and mice. these metabolites. The coneentrations of tamoxifen and metabolites found in human normal and mahignant tissues confirmed and extended the conclusions made in the experimentswith These pharmacokinetic properties of tamoxifen may explain why no correlation between may cause different pharmacodynamics. Pharmacokinetics. Tamoxifen is extensively metabolized predominantly by the cytochrome P (CYP) system to several primary and secondary metabolites, some of which exhibit more antiestrogenic effects in breast cancer cells than tamoxifen itself [Articles, ,]. Tamoxifen metabolism mostly. Nov 4, - 15 days pre-treatment with red clover did not alter the tamoxifen and its active metabolite 4-hydroxytamoxifen pharmacokinetics significantly (p >). Robinson, S. P., Langan-Fahey, S. M., Johnson, D. A. & Jordan, V. C. Metabolites, pharmacodynamics, and pharmacokinetics of tamoxifen in rats and. May 5, - Various cytochrome P (CYP) enzymes have been proposed, and investigated, to affect the pharmacokinetics and pharmacodynamics of tamoxifen, since tamoxifen is bioactivated to more active metabolites (e.g. endoxifen) by these enzymes. CYP2D6 genotype showed a clear gene-exposure effect, but. Tamoxifen acts as an anti-estrogen (inhibiting agent) in the mammary tissue, but as an estrogen (stimulating agent) in cholesterol metabolism, bone density, and cell Pharmacodynamics. Tamoxifen belongs to a class of drugs called selective estrogen receptor modulators (SERMs), which have both estrogenic and.