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Human CYP2D6 and metabolism of m-chlorophenylpiperazine

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Abstract

Background: Metabolic drug–drug interactions can occur between drugs that are substrates or inhibitors of the same cytochrome P450 (CYP) isoenzymes, but can be prevented by knowing which isoenzymes are primarily responsible for a drug's metabolism. m-Chlorophenylpiperazine (mCPP) is a psychopharmacologically active metabolite of four different psychiatric drugs. The present experiments were designed to identify the CYP isoenzymes involved in the metabolism of mCPP to its main metabolite p-hydroxy-mCPP (OH-mCPP).

Methods: The rate of production of OH-mCPP from mCPP was correlated with isoform activities in a panel of human liver microsomes, was assessed using a panel of individual complementary DNA-expressed human CYP isoenzymes, and was investigated in the presence of a specific inhibitor of CYP2D6.

Results: OH-mCPP production correlated significantly with CYP2D6 activity in human liver microsomes. Furthermore, incubations with microsomes from cells expressing CYP2D6 resulted in OH-mCPP formation, whereas no mCPP was formed from incubations with microsomes from cells expressing other individual isoforms. Finally, when the specific CYP2D6 inhibitor quinidine was preincubated with either human liver microsomes or cells expressing human CYP2D6, there was a concentration-dependent decrease in the production of OH-mCPP.

Conclusions: These results confirm that CYP2D6 is the isoform responsible for the p-hydroxylation of mCPP, and indicate that caution should be exercised in coprescribing inhibitors or substrates of CYP2D6 with drugs that have mCPP as a metabolite.

Keywords: [Cytochrome P450](#), [m-chlorophenylpiperazine](#), [microsomes](#), [trazodone](#), [nefazodone](#), [metabolism](#)

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