**ENZYME-INDUCING AEDs SHOULD NOT BE USED AS AGENTS IN THE TREATMENT OF EPILEPSY**

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Carbamazepine, phenytoin, phenobarbital and primidone stimulate the activity of a variety of monooxygenase (cytochrome P450) enzymes in addition to glucuronyl transferases and epoxide hydroxylases. Patients taking these agents metabolise at a faster rate a wide range of concomitantly administered lipid-soluble medications including oral contraceptives, warfarin, cytotoxics, cardiac antiarrhythmics, antiretrovirals, statins, immunosuppressants and other antiepileptic drugs. This can result in treatment failure e.g. breakthrough pregnancy, transplant rejection, increased cancer mortality, progressive AIDs, etc. The doses of concomitant medication often require to be increased to offset their increased clearance. Higher doses will invariable result in greater costs. Later withdrawal of enzyme inducers will increase the circulating concentrations of these drugs with the risk of serious toxicity. Induction of endogenous hormone breakdown can lead to osteoporosis and sexual dysfunction. None of the modern AEDs possesses the extensive enzyme-inducing properties of the older generation agents. However, oxcarbazepine, lamotrigine, felbamate and high dose topiramate accelerate the metabolism of oral contraceptive steroids. Enzyme induction takes place as long as the patient is taking the inducer. No one knows what health problems lie in wait down life's journey. Managing concomitant treatment with enzyme-inducing AEDs is difficult enough for the expert. Many doctors are unaware of the pitfalls of enzyme induction let alone have the skill to protect their epilepsy patients from a major iatrogenic disaster. Why take the risk of serious morbidity or even mortality by prescribing life-long treatment with an enzyme inducer when there is available a range of safer and equally effective alternative AEDs?