inhibiting acid production of the bound cell of the active pumps (Bell NJV., *et al*., 1992). Granted that acid recovery depends on synthesis of new pumps and slow dissociation of the PPI from the cysteine. Differences in cysteine binding between drugs may thus affect the speed of acid recovery. In addition to new pump synthesis, recovery of acid secretion may be by reduction of the disulfide bond by extracellular glutathione. Secretory capacity is restored when new pumps (H^+/K^+ ATPase molecules) are converted from their inactive status in the tubulovesicle to their active form at the canalicular surface, which occurs on average in 36-72 hours (Klinkenberg-Knol EC *et al*., 1995).

Furthermore, all PPIs are metabolized in the liver by the cytochrome P450 system having two main enzymes: CYP2C19, which forms inactive 5-hydroxy and 5-O-desmethyl metabolites, and the CYP3A4 enzyme, which forms an inactive sulfone metabolite (Hatlebakk JG, *et al.*,2000). preference for one enzyme over another influences the metabolic pathway and hence, lead to differences among the PPIs group of drugs in interactions with other drugs. PPIs as a result of the reduction in gastric acidity, they may alter the absorption of other orally administered drugs. Consequently elevated gastric pH has the potential to affect the stability of drugs that are acid-labile or alkaline-labile as well as the absorption of drugs that have pH-dependent formulations. Whether these interactions are clinically significant is more difficult to determine. Although pharmacokinetic interactions at the CYP2C19 level are uncommon, there is considerable genetic variation in enzyme capacity. Efficacy of PPIs in controlling pH and healing of erosive esophagitis may have some dependence on the percentage of the molecule metabolized via CYP2C19 vs CYP3A4 and CYP2C19 polymorphism (Van Herwaarden MA, *et al*., 1999).