Important to realize of this group, esomeprazole, lansoprazole, omeprazole, pantoprazole and rabeprazole. They are similar in their action but they differ in their drug interaction and the way they are broken in the liver and that's explain the longer effect of some PPIs, therefore may be taken less frequently (Samloff IM, *et al*,. 1981).

In particular, the available PPIs that are approved in the United States includes omeprazole (prilosec®), lansoprazole (Aciphex®), pantoprazole (Protonix®), Esomeprazole (Nexium®) (FDA prescribing information). Notably, PPIs suppress daytime, nocturnal, and meal-stimulated acid secretion. The degree of acid inhibition with PPIs does not correlate with plasma concentration but to the area under the curve (AUC). The slower a PPI is cleared from the plasma, the more of it is available to be delivered to the proton pump (Kawamura M *et al.*, 2003). As an illustration PPIs inhibit only active proton pumps, because not all pumps are active at any given time, so a single dose of a PPI does not inhibit all pumps and does not result in profound inhibition of acid secretion . Acid secretion by these proton pumps will therefore be inhibited with subsequent PPI doses, taking 5-7 days to achieve a steady state with a PPI. Surprisingly There is no complete acid inhibition because of continuous synthesis of new proton pumps (Egan LJ *et al*., 2003). If PPIs given twice daily steady-state inhibition of gastric acid secretion will be achieved more rapidly and will be more complete (Bensancon M *et al.,* 1997).

Another key point, PPIs are weak bases, incompletely absorbed, with a short half-lives 0.6-1.9 hours (Lew EA, *et al.*, 1999). They accumulate and activate in an acid environment at the secretory canalicular surface of the parietal cell. In this environment, the inactive benzimidazole of the PPI is converted to a cationic tetracyclic sulfonamide, that covalently binds to cysteine residues on the alpha subunit of the H^+/K^+ ATPase enzyme,