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DEVELOPMENT AND EVALUATION OF OMEPRAZOLE BUCCAL FILM

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ABSTRACT
Buccal tablets were prepared using mucoadhesive polymers like Chitosan, HPMC K4M, Na CMC & amp; Sod. alginate by direct compression technique. Buccal tablets were characterized for number of parameters like Hardness, weight uniformity, thickness, % friability, swelling index, mucoadhesive strength, surface pH, drug–excipient interaction study, drug content uniformity and In vitro drug release study. The continuous secretion of saliva and its subsequent swallowing can lead to substantial drug depletion from the dosage form and hence low bioavailability. Therefore, other transmucosal routes such as nasal, rectal, vaginal, ocular and oral mucosae are being considered as alternatives to conventional oral dosage forms for drug delivery to avoid the above disadvantages associated with conventional oral delivery (i.e., tablets, capsules, syrups, etc.). Of these routes of delivery, the buccal oral mucosa has emerged as one of the target sites for administration of drugs in a wide variety of dosage forms, particularly for those drugs targeted for local delivery in the oral cavity and systemic absorption.

KEYWORDS: Buccal Tablets, Polymers, Mucoadhesion / bioadhesion.

INTRODUCTION

The mucosa of the mouth is very different from the rest of the gastrointestinal tract and morphologically is more similar to skin. Although the permeability of skin is widely regarded as poor, it is not generally appreciated that the oral mucosa lacks the good permeability demonstrated by the intestine. These differences within the gastrointestinal tract can largely be attributed to the organization of the epithelia, which serve very different functions. A simple, single-layered epithelium lines the stomach, small intestine, and colon, which provides for a minimal transport distance for absorbents. In contrast, a stratified or multilayered epithelium covers the oral cavity and esophagus and, in common with skin, is composed of layers with varying states of differentiation or maturation evident on progression from the basal cell layer to the surface. Drugs have been applied to the oral mucosa for topical applications for many years. However, recently there has been interest in exploiting the oral cavity as a portal for delivering drugs to the systemic circulation. Notwithstanding the relatively poor permeability characteristics of the epithelium, a number of advantages are offered by this route of administration. Foremost among these are the avoidance of first-pass metabolism, ease of access to the delivery site, and the opportunity of sustained drug delivery predominantly via the buccal tissue.[1]

Omeprazole[1]

Omeprazole is a proton pump inhibitor used to treat GERD associated conditions such as heartburn and gastric acid hypersecretion, and to promote healing of tissue damage and ulcers caused by gastric acid and H. pylori infection.

Brand Names

Konvomep, Losec, Omeclamox, Omesec, Previdolrx Analgesic Pak, Prilosec, Talicia, Yosprala, Zegerid, Zegerid Reformulated Aug 2006, Zegerid with Magnesium Hydroxide

Generic Name

Omeprazole.

Drug Bank Accession Number DB00338

Originally approved by the FDA in 1989, omeprazole is a proton-pump inhibitor, used to treat gastric acid-related disorders. These disorders may include gastro esophageal reflux disease (GERD), peptic ulcer disease, and other diseases characterized by the over secretion of gastric acid. This drug was the first clinical useful drug in its class, and its approval was followed by the formulation of many other proton pump inhibitor drugs. Omeprazole is generally effective and well- tolerated, promoting its popular used in children and adults.

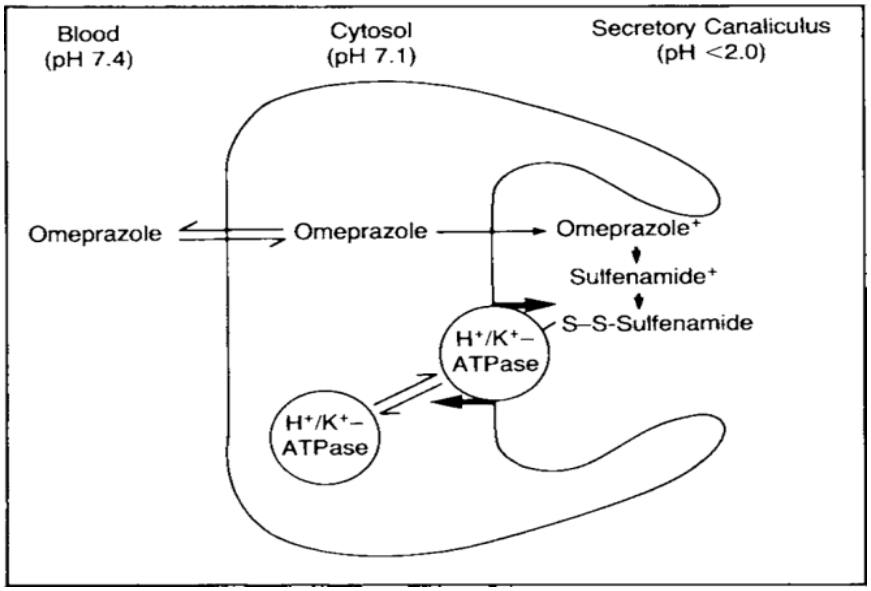

Figure 1.

Type[2]
Small Molecule

Groups
Approved, Investigational, Vet approved **Chemical Formula:** C17H19N3O3S **Indications:**
Omeprazole, according to the FDA label is a proton pump inhibitor (PPI) used for the following purposes:

- Treatment of active duodenal ulcer in adults
- Eradication of Helicobacter pylori to reduce the risk of duodenal ulcer recurrence in adults
- Treatment of active benign gastric ulcer in adults
- Reduction of risk of upper gastrointestinal (GI) bleeding in critically ill adult patients.
- Treatment of symptomatic gastro esophageal reflux disease (GERD) in patients 1 year of age and older
- Treatment of erosive esophagitis (EE) due to acid-mediated GERD in patients 1 month of age and older
- Maintenance of healing of EE due to acid-mediated GERD in patients 1 year of age and older
- Pathologic hyper secretory conditions in adults[2]

Pharmacodynamics[3]
Effects on Gastric Acid Secretion
This drug decreases gastric acid secretion. After oral administration, the onset of the antisecretory effect of omeprazole is usually achieved within one hour, with the maximum effect occurring by 2 hours after administration. The inhibitory effect of omeprazole on acid secretion increases with repeated once-daily dosing, reaching a plateau after four days.

Effects On Serum Gastrin
In studies of 200 or more patients, serum gastrin levels increased during the first 1- 2 weeks of daily administration of therapeutic doses of omeprazole. This occurred in a parallel fashion with the inhibition of acid secretion. No further increase in serum gastrin occurred with continued omeprazole administration. Increased gastrin causes enterochromaffin-like cell hyperplasia and increased serum Chromogranin A (CgA) levels. The increased CgA levels may lead to false positive results in diagnostic studies for neuroendocrine tumors Label.

Enterochromaffin-like (ECL) cell effects
Human gastric biopsy samples have been obtained from more than 3000 pediatric and adult patients treated with omeprazole in long-term clinical studies. The incidence of enterochromaffin-like cell hyperplasia in these studies increased with time; however, no case of ECL cell carcinoids, dysplasia, or neoplasia have been identified in these patients. These studies, however, are of insufficient in power and duration to draw conclusions on the possible influence of long-term administration of omeprazole in the development of any premalignant or malignant conditions

Other Effects[4]
Systemic effects of omeprazole in the central nervous system, cardiovascular and respiratory systems have not been found to date. Omeprazole, given in oral doses of 30 or 40 mg for 2-4 weeks, showed no effect on thyroid function, carbohydrate metabolism, or circulating levels of parathyroid hormone, cortisol, estradiol, testosterone, prolactin, cholecystokinin or secretin[3].

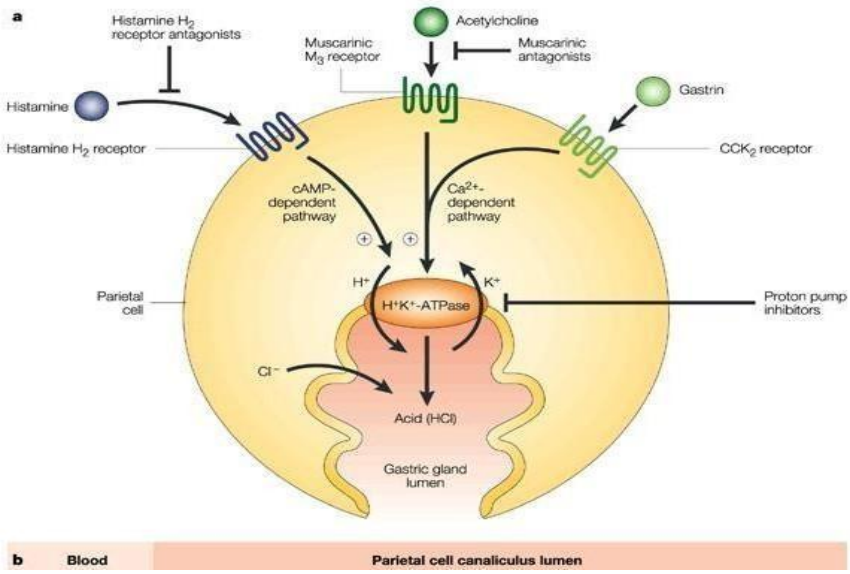

Figure 2.

Mechanism of Action[6]

Hydrochloric acid (HCl) secretion into the gastric lumen is a process regulated mainly by the H(+)/K(+)- ATPase of the proton pump 10, expressed in high quantities by the parietal cells of the stomach. ATPase is an enzyme on the parietal cell membrane that facilitates hydrogen and potassium exchange through the cell, which normally results in the extrusion of potassium and formation of HCl (gastric acid) 9. Omeprazole is a member of a class of antisecretory compounds, the substituted benzimidazoles, that stop gastric acid secretion by selective inhibition of the H+/K+ ATPase enzyme system. Proton-pump inhibitors such as omeprazole bind covalently to cysteine residues via disulfide bridges on the alpha subunit of the H+/K+ ATPase pump, inhibiting gastric acid secretion for up to 36 hours 11. This antisecretory effect is dose-related and leads to the inhibition of both basal and stimulated acid secretion, regardless of the stimulus.

Mechanism Of H. Pylori Eradication[7]

Peptic ulcer disease (PUD) is frequently associated with Helicobacter pylori bacterial infection (NSAIDs). The treatment of H. pylori infection may include the addition of omeprazole or other proton pump inhibitors as part of the treatment regimen. H. pylori replicate most effectively at a neutral pH. Acid inhibition in H. pylori eradication therapy, including proton-pump inhibitors such as omeprazole, raises gastric pH, discouraging the growth of H. pylori. It is generally believed that proton pump inhibitors inhibit the urease enzyme, which increases the pathogenesis of H. pylori in gastric-acid related conditions.

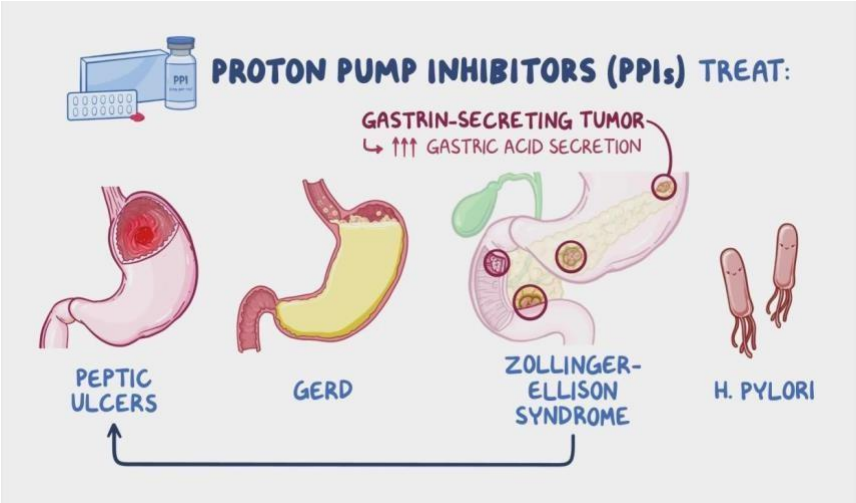

Figure 3.

Absorption[8]

Omeprazole delayed-release capsules contain an enteric-coated granule formulation of omeprazole (because omeprazole is acid-labile), so that absorption of omeprazole begins only after the granules exit the stomach. Absorption of omeprazole occurs rapidly, with

peak plasma concentrations of omeprazole achieved within 0.5-3.5 hours. Absolute bioavailability (compared with intravenous administration) is approximately 30-40% at doses of 20-40 mg, largely due to pre-systemic metabolism. The bioavailability of omeprazole increases slightly upon repeated administration of omeprazole delayed-release capsules.

Volume of Distribution
Approximately 0.3 L/kg, corresponding to the volume of extracellular water. Protein binding Approximately 95% bound to human plasma proteins.

Metabolism
Omeprazole is heavily metabolized in the liver by the cytochrome P450 (CYP) enzyme system. The main part of its metabolism depends on the polymorphically expressed CYP2C19, which is responsible for the formation of hydroxy omeprazole, the major metabolite found in plasma. The remaining part depends on CYP3A4, responsible for the formation of omeprazole sulphone.

Route Of Elimination[9]
After a single dose oral dose of a buffered solution of omeprazole, negligible (if any) amounts of unchanged drug were excreted in urine. Most of the dose (about 77%) was eliminated in urine as at least six different metabolites. Two metabolites were identified as hydroxy omeprazole and the corresponding carboxylic acid. The remainder of the dose was found in the feces. This suggests significant biliary excretion of omeprazole metabolites. Three metabolites have been identified in the plasma, the sulfide and sulfone derivatives of omeprazole, and hydroxy omeprazole. These metabolites possess minimal or no antisecretory activity.

Half-Life[10]
0.5-1 hour (healthy subjects, delayed-release capsule)
Approximately 3 hours (hepatic impairment)

Clearance
Healthy subject (delayed release capsule), total body clearance 500 - 600 mL/min Geriatric plasma clearance: 250 mL/min
Hepatic impairment plasma clearance: 70 mL/min5

SECTION SNIPPETS
Materials
Omeprazole, sodium alginate (300–400 cPs) and hydroxypropyl methylcellulose (HPMC, 2208, 4000 cPs) were supplied by Chongkun Dang (Seoul, Korea), Wako (Tokyo, Japan) and Shinetsu (Tokyo, Japan), respectively. Magnesium oxide and croscarmellose sodium were of USP grade.

Preparation Of Omeprazole Buccal Adhesive Tablets[11]
As shown in Table 1, omeprazole buccal adhesive tablets, in diameter 7 mm and hardness 6–8 kP (kilopascal), were prepared by compressing 20 mg of omeprazole, 80–95 mg of excipients such as sodium alginate, HPMC, magnesium oxide and.

Release Of Omeprazole from Buccal Adhesive Tablet[12]
To test the effect of composition ratio of sodium alginate to HPMC on the release rates of omeprazole from the buccal adhesive tablets, we determined the release kinetic parameters of omeprazole from buccal adhesive tablets composed of 20 mg of omeprazole, 30 mg of the mixture of sodium alginate and HPMC with different ratio, and 50 mg of magnesium oxide.

CONCLUSION
It is concluded that the omeprazole buccal adhesive tablet composed of omeprazole–sodium alginate–HPMC–magnesium oxide–croscarmellose sodium (20:24:6: 50:10 mg), which could be attached on human cheek without collapse and stabilize a drug in human saliva for at least 4 h, gave a fast release of omeprazole and absolute bioavailability of 13.7±3.2% in hamsters. Our results suggest that the omeprazole buccal adhesive tablet would be useful to deliver omeprazole which degrades very rapidly in acidic.

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10. NIH StatPearls: Omeprazole
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12. FDA Approved Drug Products: KONVOMEP™ (omeprazole and sodium).