



Review

Potassium-Competitive Acid Blockers as a New Frontier in Acid-Related Disorders and the Role of South Korean Pharmacological Innovation

Jyotsna S. Ranbhise ^{1,2,3} , Manish Kumar Singh ^{1,2} , Hyeong Rok Yun ^{1,2}, Sunhee Han ^{1,2,3}, Sung Soo Kim ^{1,2,3,*} and Insug Kang ^{1,2,3,*}

- ¹ Department of Biochemistry and Molecular Biology, School of Medicine, Kyung Hee University, Seoul 02447, Republic of Korea; jogm25@khu.ac.kr (J.S.R.); manishbiochem@gmail.com (M.K.S.); foryou018@naver.com (H.R.Y.); sunheehan@khu.ac.kr (S.H.)
- ² Biomedical Science Institute, Kyung Hee University, Seoul 02447, Republic of Korea
- ³ Department of Biomedical Science, Graduate School, Kyung Hee University, Seoul 02447, Republic of Korea
- * Correspondence: sgskim@khu.ac.kr (S.S.K.); iskang@khu.ac.kr (I.K.); Tel.: +82-2-961-0524 (S.S.K.); +82-2-11-961-287 (I.K.)

Abstract

For over three decades, Proton Pump Inhibitors (PPIs) have served as the established primary therapy for acid-suppressive therapy; however, their clinical utility is frequently compromised by their slow onset of action, meal-time dependency, and metabolic variability driven by *CYP2C19* polymorphisms. Potassium-competitive acid blockers (P-CABs) represent a definitive pharmacological shift, offering rapid, reversible, and genotype-independent inhibition of the H^+/K^+ -ATPase. This review outlines the emergence of South Korea as a global epicenter for P-CAB innovation, predicated upon a strategic partnership between a high domestic burden of *Helicobacter pylori* infection and a rising incidence of refractory gastroesophageal reflux disease (GERD). We analyze the clinical and structural developments of indigenous Korean agents, specifically tegoprazan and fexuprazan, the latter of which features an optimized 9-h elimination half-life engineered to mitigate nocturnal acid breakthrough. A critical evaluation of recent clinical evidence is provided, including the most recent 2024 prospective Phase III data, which demonstrate that 14-day P-CAB-based triple therapy achieves significantly improved eradication rates in high-clarithromycin-resistance environments. Furthermore, we explore the emerging 2025 trend toward personalized, on-demand maintenance therapy and address critical long-term safety considerations, including the duration-dependent risk of metachronous gastric cancer identified in recent multicenter longitudinal studies. Ultimately, the South Korean clinical framework is providing the foundational evidence for a global transition toward P-CAB-centered treatment algorithms, redefining the standards of care in modern gastroenterology.



Academic Editor: Predrag Sikirić

Received: 28 June 2026

Revised: 21 July 2026

Accepted: 24 July 2026

Published: 26 July 2026

Copyright: © 2026 by the authors.

Licensee MDPI, Basel, Switzerland.

This article is an open access article distributed under the terms and conditions of the [Creative Commons Attribution \(CC BY\)](https://creativecommons.org/licenses/by/4.0/) license.

Keywords: potassium-competitive acid blockers; tegoprazan; fexuprazan; South Korea; gastroesophageal reflux disease; *Helicobacter pylori* eradication; *CYP2C19* polymorphism; on-demand therapy; nocturnal acid breakthrough; acid-related disorders

1. Introduction

The management of acid-related gastrointestinal disorders underwent a monumental shift in the late 1980s with the introduction of Proton Pump Inhibitors (PPIs). Following

the approval of omeprazole, PPIs quickly supplanted H₂-receptor antagonists (H₂RAs) as the primary therapeutic mainstay for treating Gastroesophageal Reflux Disease (GERD), Peptic Ulcer Disease (PUD), and *Helicobacter pylori* (*H. pylori*) infections. By irreversibly inhibiting the H⁺/K⁺-ATPase enzyme, PPIs offered a level of acid suppression that was previously unattainable, significantly improving mucosal healing rates and patient quality of life for over three decades [1–3]. To date, PPIs remain the foundational cornerstone of acid-related disease management globally, supported by decades of established clinical efficacy, a well-documented long-term safety profile, and significant cost-effectiveness afforded by the widespread availability of generic formulations [4,5].

However, as clinical experience with PPIs grew, so did the recognition of their pharmacological ceiling effect. Despite their efficacy, PPIs possess inherent limitations that prevent optimal therapeutic outcomes in a substantial group of patients. First, PPIs are prodrugs with a short systemic half-life (approximately 1–2 h), requiring enteric coating to survive gastric acidity. Immediate-release formulations utilizing sodium bicarbonate buffers represent a significant pharmacological exception designed for rapid onset. But the majority of conventional delayed-release PPIs are subject to the limitations of enteric protection. This requirement contributes to a delayed onset and often necessitates three to five days of repeated dosing to achieve steady-state acid suppression [6,7]. Furthermore, their strict requirement for administration 30 to 60 min before a meal to coincide [8] with the activation of parietal cell proton pumps creates a significant burden for patient compliance [9].

Beyond these general limitations, the efficacy of PPI therapy is further complicated by regional clinical challenges, particularly within East Asian populations. In South Korea, the high prevalence of *H. pylori* and a rising incidence of GERD have placed a premium on consistent and potent acid suppression. In this context, the metabolic dependency of PPIs on the hepatic cytochrome P450 2C19 (*CYP2C19*) enzyme system becomes a critical hurdle. Because genetic polymorphisms in this system lead to highly variable drug exposure, many Korean patients, specifically rapid metabolizers, experience insufficient acid control [10–12]. This unmet medical need in the Korean clinical scenario served as the primary catalyst for the domestic pharmaceutical industry to move beyond PPIs and pioneer the development of the next generation of acid blockers, Potassium-Competitive Acid Blockers (P-CABs).

This review explores the rise in P-CABs, with a specific focus on how South Korean research and development are redefining the global standards for acid-related disease management. By addressing the structural and metabolic failures of previous therapies through a novel mechanism of action, Korean P-CABs have transitioned from experimental alternatives to primary clinical tools. To understand the clinical success of these agents in the Korean market, it is essential first to examine the unique pharmacological properties that distinguish P-CABs from their predecessors at the molecular level.

2. Materials and Methods

A comprehensive literature search was conducted across the PubMed, Scopus, Google Scholar, and Web of Science databases to identify relevant clinical and pharmacological studies published through January 2025. The search strategy utilized combinations of the following keywords: ‘potassium-competitive acid blocker’, ‘P-CAB’, ‘tegoprazan’, ‘fexuprazan’, ‘*Helicobacter pylori* eradication’, and ‘gastroesophageal reflux disease’.

To capture the most contemporary clinical advancements and the rapid regulatory transition in South Korea, the inclusion criteria prioritized randomized controlled trials, multicenter Phase III studies, and meta-analyses published between 2020 and 2025. Foundational papers and landmark historical trials published before this period were selectively included to provide necessary context regarding the evolution of acid-suppressive therapy and the established molecular mechanisms of the H⁺/K⁺-ATPase. Only peer-reviewed

articles published in English and Korean were included in the primary analysis. Case reports, letters to the editor, and non-peer-reviewed conference abstracts were excluded to maintain the high evidence-based standard of the review.

3. Mechanisms and Pharmacokinetics of P-CABs

The clinical utility and rapid market penetration of P-CABs within the South Korean therapeutic scene are fundamentally predicated upon a distinct pharmacological model. The evolution from traditional PPIs to P-CABs represents a critical shift in the methodology of gastric H^+ / K^+ -ATPase inhibition, moving from irreversible covalent modification to a high-affinity, reversible ionic competition.

3.1. Molecular Mechanism: Ionic Competition vs. Covalent Binding

The divergence between conventional PPIs and P-CABs is ingrained in their distinct binding kinetics and activation requirements. PPIs are substituted benzimidazoles that act as prodrugs; they are required to undergo an acid-catalyzed cleavage within the parietal cell's secretory canaliculi to transform into active tetracyclic sulfenamides [13,14]. While this interaction is frequently described as irreversible, it is more accurately characterized as slowly reversible. Research has demonstrated that acid secretion recovery is determined by both the de novo synthesis of new pump proteins and the variable dissociation rates of the PPI from specific cysteine residues [15]. This prolonged covalent interaction nonetheless contributes to the recognized lag phase in therapeutic efficacy [16,17].

Conversely, P-CAB agents like tegoprazan and fexuprazan are acid-stable, lipophilic weak bases that bypass the requirement for acid-induced activation [18,19]. These agents function via reversible, non-covalent ionic competition with potassium ions (K^+) at the pump's K^+ -binding site [20,21]. By physically occluding the K^+ -exchange channel, P-CABs inhibit the pump in both its active and resting states. This mechanism facilitates a near-immediate inhibition of acid secretion following the initial dose, effectively eliminating the requirement for steady-state accumulation typically seen with PPI therapy [22–24]. The divergence in molecular binding kinetics and the resulting impact on the parietal cell's secretory function are summarized in Figure 1, which provides a comparative structural overview of these inhibitory pathways.

These fundamental differences in binding kinetics transitioning from a slowly reversible covalent modification to competitive ionic antagonism directly dictate the optimized pharmacokinetic and pharmacodynamic profiles of the P-CAB class. While the molecular mechanism provides the foundation for rapid acid suppression, the resulting systemic absorption, distribution, and metabolic stability further differentiate these indigenous agents in clinical practice.

3.2. Pharmacokinetic Advantages: Onset, Duration, and Meal Independence

The pharmacokinetic advantage of the P-CAB class over traditional PPIs is primarily due to their high bioavailability and the absence of a required lag phase to achieve therapeutic steady state. Typically, PPIs are susceptible to gastric acid degradation and necessitate enteric coating, which inherently delays systemic absorption. A notable exception includes immediate-release formulations that combine non-enteric-coated PPIs with buffers like sodium bicarbonate to facilitate rapid onset through immediate gastric absorption [25,26]; however, for the majority of conventional PPIs, the requirement for enteric protection results in a delayed T_{max} . In contrast, P-CABs are acid-stable molecules that facilitate rapid sequestration within the parietal cell canaliculi. Consequently, these agents exhibit significantly accelerated absorption kinetics, with a T_{max} (time to reach peak plasma concentration) typically occurring within 1.5 to 2.0 h. This rapid systemic absorption

facilitates a near-immediate onset of action, characterized by a clinically significant increase in intragastric pH within the first hour of administration [27,28]. From a pharmacodynamic perspective, this rapid systemic residence allows for a maximum inhibitory effect ($I_{\max}$) on gastric acid secretion following a single oral dose [29,30]. This contrasts sharply with the cumulative dosing required for PPIs, which typically achieve maximal $H^+ / K^+ \text{-ATPase}$ inhibition only after three to five consecutive days of administration [31–33].

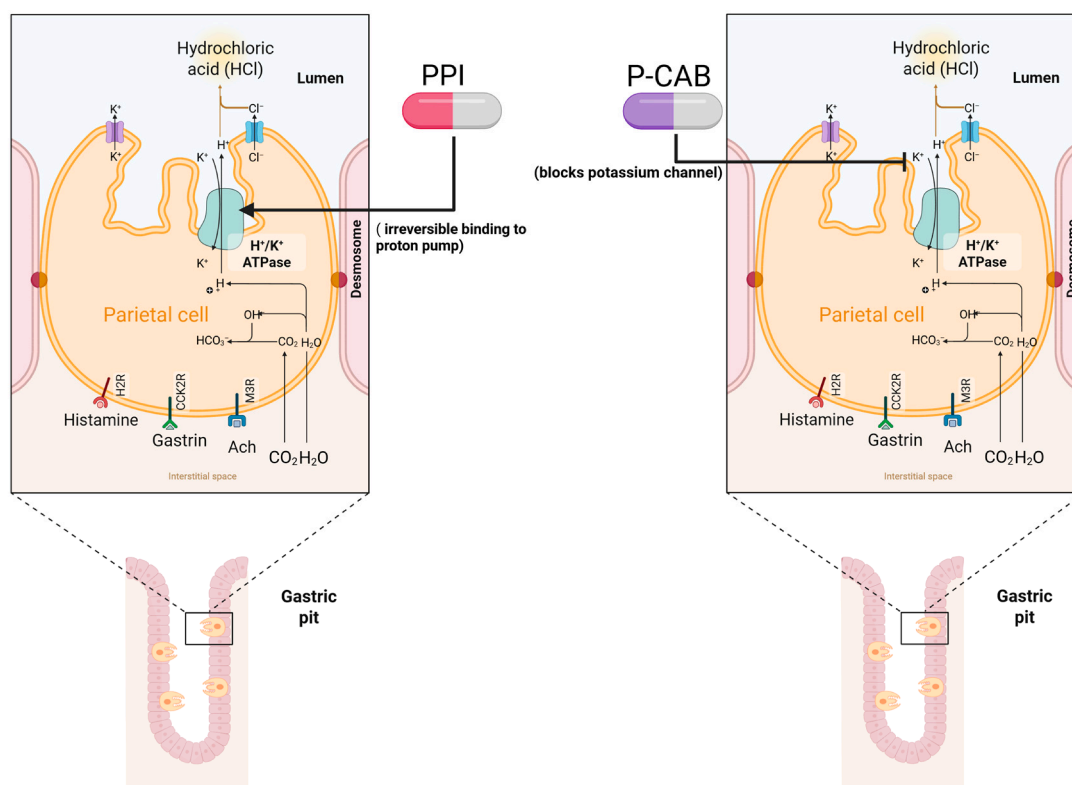


Figure 1. Comparative Molecular and Cellular Mechanisms of Gastric $H^+ / K^+ \text{-ATPase}$. Inhibition by PPIs and P-CABs. The pink capsule (left) represents the PPI standard, wherein the prodrug requires acid-catalyzed activation to form an irreversible covalent disulfide bond with the $H^+ / K^+ \text{-ATPase}$, effectively neutralizing actively secreting pumps. In contrast, the purple capsule (right) signifies the P-CAB mechanism (e.g., tegoprazan or fexuprazan), which utilizes high-affinity, reversible ionic competition with potassium ions (K^+) at the luminal exchange site of the ATPase. By physically occluding the K^+ -binding channel, P-CABs inhibit the pump in both active and resting states, facilitating a near-instantaneous onset of acid suppression and ensuring clinical efficacy independent of prandial timing. Created in BioRender. Ranbhise, Jyotsna. (2026) <https://BioRender.com/c8llblf> (accessed on 25 June 2026).

Furthermore, the pharmacodynamic profile of P-CABs is characterized by meal-independent efficacy to address one of the primary compliance barriers of traditional acid suppression [34,35]. Because P-CABs do not require the proton pumps to be in an active, secreting state to facilitate binding, unlike the prodrug mechanism of PPIs, their inhibitory capacity is dissociated from the postprandial state [36]. Pharmacokinetic evaluations have demonstrated that neither the area under the curve (AUC) nor the $C_{\max}$ (peak concentration) of P-CABs are clinically significantly altered by food intake. This allows for therapeutic flexibility that ensures consistent intragastric pH maintenance regardless of dietary timing, as demonstrated in pharmacodynamic evaluations of healthy volunteers. This is particularly advantageous in maintaining a target pH > 4.0 for extended periods, which is a critical clinical parameter for the healing of erosive esophagitis (EE) and the prevention of mucosal damage [37,38]. In addition to the clinical flexibility afforded by meal-independent

pharmacokinetics, the therapeutic predictability of the P-CAB class is further substantiated by its distinct metabolic course. Beyond the external factors of administration timing, the systemic response is further stabilized through an enzymatic pathway that bypasses the significant genetic variability inherent to conventional acid-suppressive regimens.

3.3. Predictable Metabolism and the CYP2C19 Advantage

The P-CAB class offers an enhanced inter-individual therapeutic response due to its primary metabolic pathway. The efficacy of conventional PPIs is frequently compromised by the high genetic variability of the CYP2C19 enzyme system. This variability leads to significant differences in drug exposure between rapid and poor metabolizers. In contrast, P-CABs are predominantly metabolized via the CYP3A4 pathway [39–41]. This metabolic shift results in plasma drug levels that remain more consistent across diverse genetic profiles. This helps mitigate the incidence of refractory symptoms in patients who would otherwise be predisposed to subtherapeutic PPI levels. The relative consistency in the pharmacokinetic and pharmacodynamic (PK/PD) relationship provides a more predictable pharmacological response. Consequently, P-CABs serve as a reliable clinical tool for managing high-prevalence acid-related disorders [42]. These pharmacokinetic attributes provide the foundational rationale for the strategic development of indigenous P-CABs in South Korea. In this region, pharmacological innovation has been systematically integrated into an evolving clinical framework. The distinctive pharmacokinetic and clinical parameters of these agents, including the first-in-class Japanese agent and indigenous Korean optimized molecules, are summarized in Table 1.

Table 1. Comparative Pharmacological and Pharmacokinetic Profiles of Conventional PPIs and South Korean P-CABs.

Parameter	Conventional PPIs (e.g., Esomeprazole)	Vonoprazan (Japan)	Tegoprazan (South Korea)	Fexuprazan (South Korea)
Binding Mechanism	Irreversible, Covalent	Reversible, ionic	Reversible, ionic	Reversible, ionic
Acid activation	Required (Prodrug)	Not required	Not required (Active drug)	Not required (Active drug)
Onset of action	Slow (3–5 days for steady state)	Rapid (approx. 1 h)	Rapid (approx. 0.5 h)	Rapid (approx. 1 h)
Half-life	Short (~1.5 h)	~7 h	~4 h	~9 h
Meal dependency	High (Must take 30–60 m pre-meal)	Negligible	Meal independent	Meal independent
CYP2C19 Influence	Significant (Genotype dependent)	Negligible	Negligible	Negligible

4. Advancements in P-CAB Development and Clinical Integration in South Korea

The development of the P-CAB class was pioneered by vonoprazan in Japan, which established the clinical feasibility of reversible H⁺/K⁺-ATPase inhibition. Vonoprazan is characterized by a high *pK_a* (9.3) and a steady elimination half-life of approximately 7 h. Building upon this precedent, South Korea has emerged as a prominent location for P-CAB innovation, driven by a strategic alignment between clinical necessity and targeted pharmaceutical development. While earlier prototypes demonstrated the feasibility of the class, the South Korean clinical scenario defined by a high seroprevalence of *H. pylori* and a rising incidence of PPI-refractory GERD served as the primary catalyst for the development of second-generation agents [43,44]. The Korean pharmaceutical sector has transitioned

the P-CAB class from an experimental pharmacological idea to a clinically dominant standard of care [45,46]. This was achieved through the systematic optimization of binding kinetics and elimination half-lives, resulting in the development of indigenous agents that address specific limitations of first-generation therapy [47]. The clinical exhibition of this industry-wide optimization is best exemplified by the therapeutic route of tegoprazan, the first indigenous Korean P-CAB to achieve comprehensive multi-indication approval and establish a new standard for rapid and consistent acid suppression [48,49].

4.1. Tegoprazan: Regulatory Milestones and Indication Breadth

Tegoprazan was approved by the South Korean Ministry of Food and Drug Safety (MFDS) in 2018 as the nation's 30th novel drug. While first-generation acid blockers focused predominantly on the healing of EE, the development of tegoprazan was specifically brought about to address NERD [50,51]. NERD constitutes the majority of the GERD spectrum in South Korea and is frequently characterized by a suboptimal response to traditional PPI therapy due to the transient nature of acid-reflux events [52].

Tegoprazan is a lipophilic, weak base with a pK_a of approximately 5.1 [53,54]. This chemical property allows it to highly concentrate within the acidic environment of the secretory canaliculi of the parietal cell without requiring acid-induced transformation into a reactive intermediate [55,56]. The benzimidazole core and chroman moiety allow it to engage in a high-affinity, non-covalent interaction with the gastric H^+/K^+ -ATPase [57,58]. While PPIs form a covalent disulfide bond with Cys813, tegoprazan functions through ionic competition with potassium ions (K^+) at the luminal surface of the pump's α -subunit. It specifically occludes the K^+ binding site, physically preventing the inward transport of K^+ and the outward transport of H^+ . Because this binding is reversible and avoids covalent modification, tegoprazan facilitates an accelerated onset of acid suppression. This inhibitory effect can be sustained as long as the plasma concentration remains above the threshold for effective H^+/K^+ antagonism [59–61].

Multicenter, randomized, double-blind Phase III clinical trials conducted across Korean medical institutions demonstrated that a 50 mg dose of tegoprazan maintains an intragastric $pH > 4.0$ for a mean duration of 18.4 to 20.1 h within a 24-h cycle [62,63]. These values were established in Phase I and II studies involving healthy Korean subjects and symptomatic GERD patients, respectively.

Beyond its application in reflux disorders, tegoprazan achieved a significant regulatory milestone as the first indigenous P-CAB integrated into *H. pylori* eradication protocols in South Korea. The clinical validity of this indication was established through a pivotal multicenter, randomized Phase III trial comparing a 7-day tegoprazan-based triple therapy (50 mg) against a lansoprazole-based triple therapy (30 mg) [64,65]. The trial revealed eradication rates of 62.86% in the Intention-to-Treat (ITT) analysis and 69.33% in the Per-Protocol (PP) analysis for the tegoprazan group. While these absolute rates appear modest, tegoprazan demonstrated clear statistical non-inferiority to the lansoprazole control (60.57% ITT; 67.33% PP), reflecting the challenging reality of primary clarithromycin resistance in South Korea, which has been documented as high as 32.5% to 42.1% in various metropolitan regions [66].

The pharmacological potency of tegoprazan in this context is also driven by its ability to maintain the high intragastric pH required for antibiotic stability. Pharmacodynamic evaluations in healthy Korean subjects have shown that tegoprazan-based regimens maintain an intragastric $pH > 6.0$ for more than 88% of 24 h, significantly outperforming traditional PPI-based triple therapies [67,68]. This potent acid suppression increases the replicative activity of *H. pylori*, thereby enhancing the bactericidal efficacy of amoxicillin and clarithromycin [69].

Critically, subgroup analyses confirmed that tegoprazan's efficacy remained consistent regardless of the *CYP2C19* genotype or the minimum inhibitory concentrations of the antibiotics used [70,71]. This emphasizes the agent's role as a predictable therapeutic option in the Korean clinical setting, where genetic polymorphisms frequently compromise PPI-based outcomes. Furthermore, recent real-world evidence from South Korean multicenter studies suggests that extending the treatment duration to 14 days significantly optimizes outcomes, with tegoprazan-based triple therapy achieving a PP eradication rate of 88.9% [72]. Following the foundational clinical and regulatory milestones established by tegoprazan, the South Korean therapeutic sector pursued further stabilization of acid suppression kinetics, which materialized with the development and subsequent approval of fexuprazan.

4.2. Fexuprazan: Pharmacodynamic Optimization and Kinetic Stability

The approval of fexuprazan in 2021 by the South Korean MFDS signified a critical refinement within the P-CAB therapeutic class, specifically targeting the pharmacokinetic limitation of duration of effect prevalent in earlier generations of acid-suppressive agents. While tegoprazan demonstrated the clinical feasibility of rapid, meal-independent acid inhibition, fexuprazan was formulated to maximize systemic residence time and binding stability [73]. It functions as a high-affinity $H^+ / K^+ \text{-ATPase}$ inhibitor characterized by an extended half-life ($t_{1/2} \approx 9 \text{ h}$) [74,75]. This pharmacokinetic property significantly exceeds the 1.5-h half-life of conventional PPIs and the 4-h half-life typically observed with other P-CABs, facilitating sustained pharmacological activity and consistent intragastric pH maintenance throughout the entire 24-h dosing interval [76,77].

Beyond general systemic kinetics, the extended pharmacodynamic profile of fexuprazan is of huge clinical significance in the management of nocturnal acid breakthrough (NAB), defined as an intragastric pH drop below 4.0 for more than one hour during the night. NAB has been widely discussed as a potential contributor to sleep disruption and esophageal mucosal damage in patients with GERD [78]. However, its direct impact on meaningful clinical outcomes remains a subject of ongoing investigation, as definitive prospective studies linking the prevention of NAB to improved symptomatic relief are currently limited. While pharmacodynamic data demonstrate that fexuprazan maintains a stable, high-affinity interaction with the proton pump throughout the night, further research is warranted to determine if this sustained nocturnal pH maintenance translates into enhanced long-term clinical benefits compared to traditional PPI therapy [79].

The therapeutic efficacy of fexuprazan in this context was specifically validated through a pivotal multicenter, randomized, double-blind Phase III study ($n = 322$) in patients with endoscopically confirmed EE. The trial data revealed cumulative mucosal healing rates at 8 weeks of 88.5% in the Full Analysis Set (FAS) and 97.3% in the Per-Protocol Set (PPS) for the fexuprazan 40 mg cohort. These results established the statistical non-inferiority of fexuprazan to esomeprazole (FAS 89.0%; PPS 97.9%). Notably, in patients stratified with severe Los Angeles classification grade C and D esophagitis, fexuprazan demonstrated robust healing rates and a significantly faster resolution of nighttime heartburn, effectively addressing the therapeutic ceiling effect often encountered with PPI-based regimens [80,81].

Another distinguishing feature of fexuprazan's pharmacological profile is its linear pharmacokinetic behavior. Unlike the non-linear, saturable kinetics often associated with PPI metabolism (due to *CYP2C19* dependency), fexuprazan's plasma concentration increases proportionally with the administered dose (up to 320 mg) [82]. This linearity provides clinicians with a highly predictable dose-response relationship, ensuring standardized acid suppression regardless of metabolic polymorphisms within the Korean

population. Furthermore, pharmacodynamic evaluations confirmed the absence of a food effect, with fexuprazan maintaining consistent AUC and $C_{\max}$ levels in both fasted and postprandial states, thereby removing the strict dosing constraints required for optimal PPI efficacy [83,84]. This clinical flexibility, combined with a safety profile, positions fexuprazan as a highly stable agent for the long-term management of chronic acid-related disorders. The structural and clinical refinements achieved with fexuprazan represent the current pharmacological apex of the domestic P-CAB pipeline, providing a critical foundation for examining the broader therapeutic authority and global regulatory course of these indigenous agents.

4.3. Clinical Validation and Global Regulatory Integration

The clinical impact of the South Korean P-CAB sector is further evidenced by its role as a clinical incubator for international therapeutic standards. The South Korean healthcare infrastructure, which is characterized by high-volume endoscopic screening and a rigorous regulatory framework, has facilitated extensive post-marketing surveillance and long-term safety evaluations [85,86]. These data have established a strong evidence base regarding the safety and efficacy of P-CABs in populations with high gastric cancer risk and diverse *CYP2C19* genotypes [87].

5. Discussion

The pharmacological transition from the slowly reversible covalent inhibition of the H^+/K^+ -ATPase by traditional PPIs toward the high-affinity, ionic antagonism offered by P-CABs represents a significant therapeutic evolution in gastroenterology [88,89]. This review has outlined how the South Korean therapeutic sector has emerged as a global epicenter for this innovation, predicated upon a strategic synergy between high-burden clinical necessity and targeted structural refinement. The success of the South Korean P-CAB pipeline is intrinsically rooted in its capacity to address the unmet needs of the domestic population. Recent global meta-analyses indicate that the worldwide prevalence of *H. pylori* has decreased from 58.2% in the 1980s to approximately 43.1% in the 2011–2022 period [90,91]. The South Korean clinical scenario is characterized by a persistently high seroprevalence of 51.0% in the adult population. This regional disparity, combined with a rising incidence of PPI-refractory GERD [91], created a demand for agents that supersede the ceiling effect of traditional benzimidazoles. By optimizing the molecular structure of indigenous molecules such as tegoprazan and fexuprazan, Korean researchers have effectively mitigated the metabolic variability associated with *CYP2C19* polymorphisms, which is a primary driver of therapeutic failure in the East Asian rapid metabolizer population [92]. The resulting enhanced pharmacological predictability is essential for maintaining sustained genotype-independent intragastric pH elevation (≥ 6.0). While healthy volunteer data indicate that this threshold is maintained for the majority of the 24-h period, its clinical importance is most evident in the stability of antibiotics within the gastric lumen of infected patients. This demonstrates a clear optimization, as tegoprazan validated the clinical utility of P-CABs in NERD and eradication therapy [93]. Fexuprazan presented more nuanced pharmacological adaptability, utilizing an extended 9-h elimination half-life to specifically target nocturnal acid breakthrough. The strategic evolution of this developmental pipeline, transitioning from domestic clinical necessity to international regulatory benchmarks, is synthesized in the clinical and regulatory trajectory schematic (Figure 2).

Beyond these pharmacological nuances, the South Korean experience serves as a foundational reference for the global transition toward P-CAB-dominant treatment algorithms. Although the initial 7-day Phase III trials in Korea highlighted the formidable barrier of 21–35% baseline clarithromycin resistance [94]. The subsequent clinical pivot toward 14-day

regimens has yielded significantly improved outcomes. A pivotal multicenter, randomized, double-blind Phase III trial (conducted February 2023–April 2024; $n = 382$) demonstrated that 14-day tegoprazan -based triple therapy achieved a modified Intention-to-Treat (mITT) eradication rate of 86.0%, effectively meeting non-inferiority criteria against lansoprazole (78.7%). While these results position P-CAB triple therapy as a robust first-line option, its clinical utility should be evaluated alongside current international guidelines that recommend Bismuth Quadruple Therapy (BQT) or susceptibility-guided treatment in regions with high resistance [95]. BQT remains highly effective. However, it is often limited by a high pill burden and frequent side effects. These issues can negatively impact patient adherence. P-CAB triple therapy offers a more tolerable alternative. It maintains stable pH elevation to optimize antibiotic efficacy without the complexity of BQT. Susceptibility-guided therapy remains the ideal approach for precision management. However, molecular resistance testing is not always accessible in clinical settings. In such cases, P-CABs provide a more reliable empirical platform. Their consistent pharmacological performance, regardless of the patient's *CYP2C19* genotype, ensures predictable results when resistance data are unavailable [96].

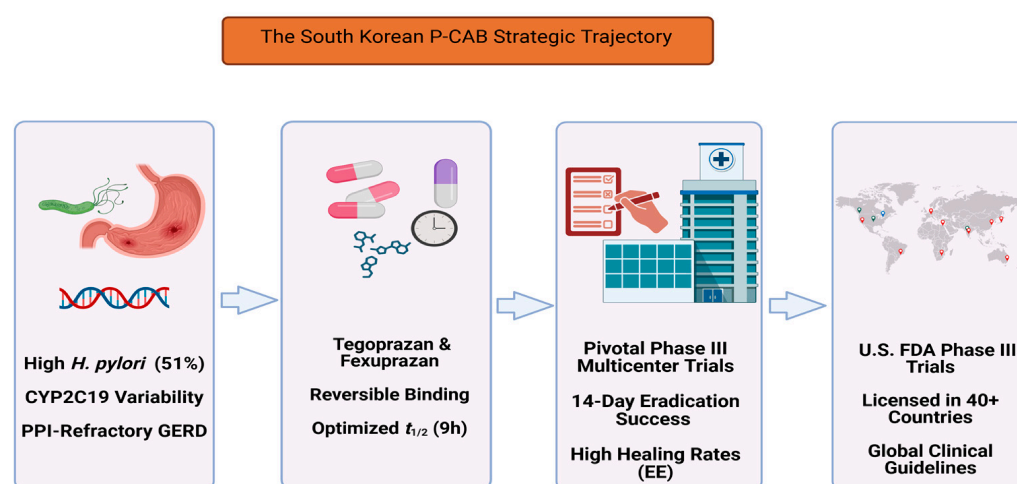


Figure 2. Schematic representation of the South Korean P-CAB clinical and regulatory trajectory. The flowchart delineates the systematic progression from addressing domestic clinical exigencies—specifically the high seroprevalence of *H. pylori* and *CYP2C19*-mediated PPI failure—toward the pharmacological optimization of indigenous agents. This trajectory has culminated in robust clinical validation through domestic Phase III trials and subsequent integration into international regulatory frameworks and global licensing agreements. Created in BioRender. Ranbhise, Jyotsna. (2026) <https://BioRender.com/9a7weng> (accessed on 21 July 2026).

Critically, in the cohort of patients with clarithromycin-resistant infections, tegoprazan exhibited a numerical trend toward high efficacy, with eradication rates reaching 47.8–50.0% compared to 35.5% in the PPI group [97,98]. While this specific subgroup difference did not reach statistical significance, it emphasizes the agent's capacity to maintain the optimal bactericidal environment ($\text{pH} \geq 6.0$) required for antibiotic stability, even in high-resistance demographics. Coupled with comparable safety and an absence of drug-related adverse effects, these findings suggest that P-CABs provide a more robust and predictable pharmacology for extended eradication therapy than conventional PPIs [99,100]. The primary clinical trials validating these therapeutic transitions across diverse acid-related pathologies are synthesized in Table 2.

Table 2. Summary of Pivotal Clinical Trials and Evidence Base for South Korean P-CABs.

Indication	Regimen Duration	Primary Result (Eradication/Healing)	Ref.
<i>H. pylori</i> Eradication	7 Days	62.86% (ITT); 69.33% (PP)	[44]
<i>H. pylori</i> Eradication	14 Days	86.0% (mITT); 91.0% (PP)	[65]
Erosive Esophagitis	8 Weeks	97.3% (PPS) Healing Rate	[101]
GERD Maintenance	On-Demand	67.7% Patient Preference	[102]

Beyond these regional pharmacological successes, the South Korean clinical experience is serving as a foundational map for the global transition toward P-CAB-centered treatment algorithms. A critical consideration in evaluating South Korean P-CAB research is the extrapolability of these data to Western and other non-Asian populations. Although the initial clinical validation was centered in cohorts with high regional disease prevalence (e.g., *H. pylori* and gastric cancer risk), the core pharmacological advantages of the P-CAB class, specifically their metabolic independence from the *CYP2C19* pathway, suggest robust global generalizability. Because *CYP2C19* polymorphisms exist across all ethnic groups (albeit at varying frequencies), the consistent therapeutic response offered by tegoprazan and fexuprazan addresses a universal clinical challenge [103]. This is further substantiated by recent Western-based Phase III trials, such as the U.S. TRIUMPH trial for tegoprazan, which reported healing rates and safety profiles in American cohorts that were consistent with the foundational data generated in South Korea [104,105]. Consequently, the Korean model of acid suppression appears highly applicable to global treatment models, particularly for patients who remain refractory to traditional PPI-based regimens regardless of their ethnic background.

The global clinical impact of the South Korean P-CAB experience is already evidenced by the evolving site of international gastroenterological guidelines. As the Korean model demonstrates the superiority of 14-day P-CAB regimens in high-resistance environments, international bodies, including the American College of Gastroenterology and the Maastricht VI/Florence consensus [95], are increasingly incorporating P-CABs into their therapeutic algorithms. This signifies a global shift where South Korean clinical data act as a predictive blueprint for Western clinicians [106], particularly in managing the growing population of patients who remain refractory to traditional PPI-based regimens regardless of their ethnic or genetic background.

However, as the clinical focus shifts toward these more potent antagonists, longitudinal research must remain focused on the long-term physiological and oncological implications of profound acid suppression. A significant 2024 multicenter retrospective cohort study from Japan (*n* = 7172) has raised critical concerns regarding the chronic utilization of P-CABs in high-risk populations. The study demonstrated that chronic P-CAB use was independently associated with an increased risk of metachronous gastric cancer (Hazard Ratio [HR] 3.89), a risk notably higher than that observed with conventional PPIs (HR 1.75). Notably, the hazard ratio escalated to 9.36 in patients utilizing therapy for more than three years, suggesting a profound duration-dependent risk path [107]. Importantly, it must be emphasized that these findings represent observational associations rather than definitive evidence of causation. Because these data are derived from retrospective cohorts, the results may be influenced by confounding variables, including the baseline severity of gastric mucosal atrophy, the presence of pre-existing intestinal metaplasia, and the physiological impact of hypergastrinemia. While P-CABs induce a more potent and

sustained elevation of serum gastrin than conventional PPIs, theoretically exerting trophic effects on the gastric mucosa. Conclusive longitudinal evidence linking this mechanism to malignant transformation in humans is currently lacking.

While current nationwide post-marketing surveillance data from South Korea indicate a favorable safety profile for short-term and maintenance use, these Japanese findings underscore the necessity for rigorous, long-term monitoring and the emphasis on the shortest effective duration of therapy. In this regard, the potential for personalized, on-demand maintenance regimens may offer a strategy to minimize cumulative systemic exposure while maintaining symptomatic relief. A landmark January 2025 prospective study from South Korea demonstrated that 67.7% of patients preferred on-demand fexuprazan therapy for GERD maintenance, with no significant difference in symptom control compared to continuous daily administration [102]. Such as-needed protocols, enabled by the rapid $T_{\max}$ of the P-CAB class, represent a significant shift toward patient-centered care and may mitigate the concerns associated with long-term profound acid suppression. Ultimately, this South Korean-led innovation represents a catalyst for a systematic transition in gastrointestinal health management. However, it is essential to note that P-CABs are intended to augment, rather than immediately replace, the existing therapeutic armamentarium. Traditional PPIs continue to play a central role in international clinical guidelines due to their proven reliability in the majority of patient populations. For many patients with mild-to-moderate GERD, the established safety experience and low economic burden of generic PPIs remain highly favorable. Therefore, the adoption of P-CABs is most clinically pertinent for unmet needs such as refractory symptoms, high-resistance *H. pylori* strains, and NAB where the specific pharmacokinetic advantages of the class justify the transition from the established PPI standard [108,109].

6. Conclusions

In conclusion, the pharmacological milestones achieved within the South Korean P-CAB sector have fundamentally shifted the therapeutic scenario for acid-related disorders. Through the development and systematic optimization of tegoprazan and fexuprazan, the inherent structural and metabolic constraints of the PPI era, specifically CYP2C19-driven variability and delayed onset of action, have been effectively mitigated. By providing rapid, meal-independent, and sustained acid inhibition, these agents offer a clinically advanced therapeutic profile for the management of the most challenging phenotypes of GERD and *H. pylori* infection.

However, it is essential to recognize that traditional PPIs currently remain the primary global standard of care for most indications, supported by an extensive history of clinical safety and cost-effectiveness. P-CABs are positioned to supplement the existing therapeutic tool by addressing specific unmet needs. As this drug class continues its global expansion, the South Korean innovation model serves as a significant catalyst for the evolving area of gastroenterology. This transition from conventional therapy must be accompanied by vigilant longitudinal surveillance to address emerging concerns regarding long-term oncological safety and to refine the role of personalized, on-demand maintenance regimens. Ultimately, the foundational evidence established in South Korea provides a robust basis for a more predictable and patient-centered future in the global management of gastrointestinal health.

Author Contributions: Conceptualization, S.S.K. and J.S.R.; resources, J.S.R., M.K.S., and H.R.Y.; data curation, J.S.R., I.K. and S.S.K.; writing—original draft preparation, J.S.R.; writing—review and editing, J.S.R., M.K.S., H.R.Y., S.H., I.K. and S.S.K.; visualization, J.S.R., S.S.K. and I.K.; supervision, I.K. and S.S.K.; project administration, I.K. and S.S.K.; funding acquisition, S.S.K. All authors have read and agreed to the published version of the manuscript.

Funding: This work was supported by the National Research Foundation of Korea (NRF) grant 415 funded by the Korean government (MSIT) (NRF-2018R1A6A1A03025124, NRF-416 2022R1A1A01067626, and RS-2025-25442355).

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: No new data were created or analyzed in this study. Data sharing is not applicable to this article.

Acknowledgments: During the preparation of this manuscript, the authors used BioRender for the purposes of creating the graphical abstract (Created in BioRender. Ranbhise, Jyotsna. (2026) <https://BioRender.com/yblq31e>) (accessed on 13 July 2026). The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest. The funders had no role in the design of the study; in the collection, analyses, or interpretation of data; in the writing of the manuscript; or in the decision to publish the results.

References

1. Andrawes, M.; Andrawes, W.; Das, A.; Siau, K. Proton Pump Inhibitors (PPIs)—An Evidence-Based Review of Indications, Efficacy, Harms, and Deprescribing. *Medicina* **2025**, *61*, 1569. [\[CrossRef\]](#)
2. Nehra, A.K.; Alexander, J.A.; Loftus, C.G.; Nehra, V. Proton Pump Inhibitors: Review of Emerging Concerns. *Mayo Clin. Proc.* **2018**, *93*, 240–246. [\[CrossRef\]](#)
3. Strand, D.S.; Kim, D.; Peura, D.A. 25 Years of Proton Pump Inhibitors: A Comprehensive Review. *Gut Liver* **2017**, *11*, 27–37. [\[CrossRef\]](#) [\[PubMed\]](#)
4. Savarino, V.; Dulbecco, P.; de Bortoli, N.; Ottonello, A.; Savarino, E. The appropriate use of proton pump inhibitors (PPIs): Need for a reappraisal. *Eur. J. Intern. Med.* **2017**, *37*, 19–24. [\[CrossRef\]](#) [\[PubMed\]](#)
5. Katz, P.O.; Dunbar, K.B.; Schnoll-Sussman, F.H.; Greer, K.B.; Yadlapati, R.; Spechler, S.J. ACG Clinical Guideline for the Diagnosis and Management of Gastroesophageal Reflux Disease. *Am. J. Gastroenterol.* **2022**, *117*, 27–56. [\[CrossRef\]](#) [\[PubMed\]](#)
6. Srebro, J.; Brniak, W.; Mendyk, A. Formulation of dosage forms with proton pump inhibitors: State of the art, challenges and future perspectives. *Pharmaceutics* **2022**, *14*, 2043. [\[CrossRef\]](#) [\[PubMed\]](#)
7. Loh, Y.Y.; Enose, A.A.; Garg, V. Enteric-coated polymers past and present—A review. *Drug Deliv. Lett.* **2022**, *12*, 85–95. [\[CrossRef\]](#)
8. Kim, S.Y.; Lee, H.; Kim, J.S.; Kim, J.H.; Jeon, H.-L. Renal Outcomes With P-CABs Versus PPIs in CKD. *Kidney Int. Rep.* **2026**, *11*, 106524. [\[CrossRef\]](#) [\[PubMed\]](#)
9. Thurber, K.M.; Otto, A.O.; Stricker, S.L. Proton pump inhibitors: Understanding the associated risks and benefits of long-term use. *Am. J. Health-Syst. Pharm.* **2023**, *80*, 487–494. [\[CrossRef\]](#) [\[PubMed\]](#)
10. Kuo, C.H.; Lu, C.Y.; Shih, H.Y.; Liu, C.J.; Wu, M.C.; Hu, H.M.; Hsu, W.H.; Yu, F.J.; Wu, D.C.; Kuo, F.C. CYP2C19 polymorphism influences *Helicobacter pylori* eradication. *World J. Gastroenterol.* **2014**, *20*, 16029–16036. [\[CrossRef\]](#) [\[PubMed\]](#)
11. Park, S.; Hyun, Y.J.; Kim, Y.R.; Lee, J.H.; Ryu, S.; Kim, J.M.; Oh, W.-Y.; Na, H.S.; Lee, J.G.; Seo, D.W. Effects of CYP2C19 genetic polymorphisms on PK/PD responses of omeprazole in Korean healthy volunteers. *J. Korean Med. Sci.* **2017**, *32*, 729. [\[CrossRef\]](#) [\[PubMed\]](#)
12. Na, J.Y.; Jeon, I.; Yoon, J.; Choi, Y.; Yoon, S.H.; Yu, K.S.; Chung, J.Y. Influence of CYP2C19 polymorphisms on the pharmacokinetics of omeprazole in elderly subjects. *Clin. Pharmacol. Drug Dev.* **2021**, *10*, 1469–1477. [\[CrossRef\]](#) [\[PubMed\]](#)
13. Ahmed, A.; Clarke, J.O. Proton pump inhibitors (PPI). In *StatPearls [Internet]*; StatPearls Publishing: St. Petersburg, FL, USA, 2023.
14. Laine, L.; Sharma, P.; Mulford, D.J.; Hunt, B.; Leifke, E.; Smith, N.; Howden, C.W. Pharmacodynamics and pharmacokinetics of the potassium-competitive acid blocker vonoprazan and the proton pump inhibitor lansoprazole in US subjects. *Am. J. Gastroenterol.* **2022**, *117*, 1158–1161. [\[CrossRef\]](#) [\[PubMed\]](#)
15. Shin, J.M.; Sachs, G. Restoration of acid secretion following treatment with proton pump inhibitors. *Gastroenterology* **2002**, *123*, 1588–1597. [\[CrossRef\]](#) [\[PubMed\]](#)
16. Tanaka, S.; Morita, M.; Yamagishi, T.; Madapally, H.V.; Hayashida, K.; Khandelia, H.; Gerle, C.; Shigematsu, H.; Oshima, A.; Abe, K. Structural basis for binding of potassium-competitive acid blockers to the gastric proton pump. *J. Med. Chem.* **2022**, *65*, 7843–7853. [\[CrossRef\]](#) [\[PubMed\]](#)
17. Geibel, J. Gastric secretions. In *Yamada's Textbook of Gastroenterology*; John Wiley & Sons Ltd.: Hoboken, NJ, USA, 2022; pp. 313–333.
18. Wong, N.; Reddy, A.; Patel, A. Potassium-competitive acid blockers: Present and potential utility in the armamentarium for acid peptic disorders. *Gastroenterol. Hepatol.* **2022**, *18*, 693.

19. Scarpignato, C.; Hunt, R.H. Potassium-competitive acid blockers: Current clinical use and future developments. *Curr. Gastroenterol. Rep.* **2024**, *26*, 273–293. [\[CrossRef\]](#) [\[PubMed\]](#)
20. Tietto, A.; Faggin, S.; Scarpignato, C.; Savarino, E.V.; Giron, M.C. Safety of potassium-competitive acid blockers in the treatment of gastroesophageal reflux disease. *Expert Opin. Drug Metab. Toxicol.* **2025**, *21*, 53–68. [\[PubMed\]](#)
21. Jankovic, K.; Gralnek, I.M.; Awadie, H. Emerging long-term risks of the use of proton pump inhibitors and potassium-competitive acid blockers. *Annu. Rev. Med.* **2025**, *76*, 143–153. [\[CrossRef\]](#) [\[PubMed\]](#)
22. Qiao, X.; Li, P. Potassium-competitive acid blockers and advances in the management of patients with acid-related diseases: A narrative review. *Front. Physiol.* **2025**, *16*, 1655102. [\[CrossRef\]](#) [\[PubMed\]](#)
23. Wang, W.; Yaseen, H.S.; Li, X.; Ahmad, M.Z.; Chen, Y.; Li, G.; Naveed, M.; Zubair, H.M.; Bibi, J. Tegoprazan: A novel, highly selective, and potent potassium-competitive acid blocker (P-CAB). *Eur. J. Clin. Pharmacol.* **2025**, *81*, 1103–1117. [\[CrossRef\]](#) [\[PubMed\]](#)
24. Antequera, C.M.; Orleck, K.; Jacob, R.; Kenneally, A.; Wright, W.L. Potassium-competitive acid blockers: Rethinking acid suppression for gastroesophageal reflux disease and *Helicobacter pylori*. *Postgrad. Med.* **2024**, *136*, 131–140. [\[CrossRef\]](#) [\[PubMed\]](#)
25. Howden, C.W. Immediate-Release Omeprazole/Sodium Bicarbonate. *Gastroenterol. Hepatol.* **2006**, *2*, 386.
26. Ramesh, S.; Zvoníček, V.; Pěček, D.; Pišlová, M.; Beránek, J.; Hofmann, J.; Dumić, A. Acid-Neutralizing Omeprazole Formulation for Rapid Release and Absorption. *Pharmaceutics* **2025**, *17*, 161. [\[CrossRef\]](#) [\[PubMed\]](#)
27. Jung, W.; Lee, J.; Jeon, H.; Sung, T.; Yun, H.y.; Lee, S.; Chae, J.w. A Mechanism-Based Multi-Level Population PK/PD Model for Potassium-Competitive Acid Blockers. *CPT Pharmacomet. Syst. Pharmacol.* **2026**, *15*, e70181. [\[CrossRef\]](#)
28. Hossa, K.; Małecka-Wojcieszko, E. Advances in gastroesophageal reflux disease management: Exploring the role of potassium-competitive acid blockers and novel therapies. *Pharmaceuticals* **2025**, *18*, 699. [\[CrossRef\]](#) [\[PubMed\]](#)
29. Koo, T.H.; Fass, R. A systematic review and meta-analysis of randomized controlled trials: The effect of potassium-competitive acid blockers on nighttime symptoms of gastroesophageal reflux disease. *Esophagus* **2026**, *23*, 275–291. [\[CrossRef\]](#) [\[PubMed\]](#)
30. Simadibrata, D.M.; Syam, A.F.; Lee, Y.Y. A comparison of efficacy and safety of potassium-competitive acid blocker and proton pump inhibitor in gastric acid-related diseases: A systematic review and meta-analysis. *J. Gastroenterol. Hepatol.* **2022**, *37*, 2217–2228. [\[CrossRef\]](#) [\[PubMed\]](#)
31. Kubo, K.; Kimura, N.; Kato, M. Potassium-competitive acid blocker-associated gastric mucosal lesions. *Clin. Endosc.* **2024**, *57*, 417–423. [\[CrossRef\]](#) [\[PubMed\]](#)
32. Shinozaki, S.; Osawa, H.; Miura, Y.; Nomoto, H.; Sakamoto, H.; Hayashi, Y.; Yano, T.; Despott, E.J.; Yamamoto, H. Endoscopic findings and outcomes of gastric mucosal changes relating to potassium-competitive acid blocker and proton pump inhibitor therapy. *DEN Open* **2025**, *5*, e400. [\[PubMed\]](#)
33. Zhang, M.; Pang, M.; Zhang, M. Efficacy and safety of potassium-competitive acid blockers versus proton pump inhibitors as *Helicobacter pylori* eradication therapy: A meta-analysis of randomized clinical trials. *Clinics* **2022**, *77*, 100058. [\[CrossRef\]](#) [\[PubMed\]](#)
34. Lim, N.R.; Chung, W.C. Intravenous proton pump inhibitors vs oral potassium competitive acid blockers before endoscopic treatment of bleeding peptic ulcers. *World J. Gastrointest. Endosc.* **2025**, *17*, 104234. [\[CrossRef\]](#) [\[PubMed\]](#)
35. Ryu, D.G.; Choi, C.W. New Therapeutic Potential of Potassium-Competitive Acid Blockers in the Management of Refractory Gastroesophageal Reflux Disease. *Korean J. Helicobacter Up. Gastrointest. Res.* **2025**, *25*, 307. [\[CrossRef\]](#) [\[PubMed\]](#)
36. Simadibrata, D.M.; Koo, T.H.; Jiménez-Castillo, R.A.; Fass, R. Potassium-competitive acid blockers and proton-pump inhibitors for the healing and maintenance of erosive esophagitis: A comprehensive and updated systematic review with network meta-analysis. *Esophagus* **2026**, 1–12.
37. Dewi, N.N.G.K.; Dewi, N.L.P.Y.; Dewi, P.I.S.L.; Pamungkas, K.M.N.; Sindhughosa, D.A.; Mariadi, I.K. Efficacy and safety of potassium-competitive acid blockers in eradicating *Helicobacter pylori* and treating gastro-esophageal reflux disease: A systematic review and meta-analysis. *Casp. J. Intern. Med.* **2026**, *17*, 14.
38. Lim, N.R.; Chung, W.C. The Efficacy of Potassium-Competitive Acid Blocker Compared With Double Dose of Proton Pump Inhibitor in Patients With Refractory Gastro-Esophageal Reflux Disease: A Case-Control Study. *Korean J. Helicobacter. Up. Gastrointest. Res.* **2025**, *25*, 350–354. [\[CrossRef\]](#) [\[PubMed\]](#)
39. Youn, M.S.; Ahn, S.H.; Kim, J.H. Pharmacogenomic profiling of the South Korean population: Insights and implications for personalized medicine. *Front. Pharmacol.* **2024**, *15*, 1476765. [\[CrossRef\]](#) [\[PubMed\]](#)
40. Tan, C.M.; Juurlink, D.N. Navigating Drug Interactions with Proton Pump Inhibitors. *JAMA Netw. Open* **2024**, *7*, e2419818. [\[CrossRef\]](#) [\[PubMed\]](#)
41. Fernandez, A.C.; Rux, C.E.; Clark, N.M. Successful treatment of refractory *Helicobacter pylori* infection with a potassium-competitive acid blocker-based regimen: A case report. *Int. J. Infect. Dis.* **2026**, *163*, 108207. [\[CrossRef\]](#) [\[PubMed\]](#)
42. Park, J.K. Optimal Use of Proton Pump Inhibitors and Potassium-competitive Acid Blockers. *Korean J. Helicobacter. Up. Gastrointest. Res.* **2023**, *23*, 180–187. [\[CrossRef\]](#) [\[PubMed\]](#)

43. Cho, J.H.; Jin, S.Y. Current guidelines for *Helicobacter pylori* treatment in East Asia 2022: Differences among China, Japan, and South Korea. *World J. Clin. Cases* **2022**, *10*, 6349–6359. [[CrossRef](#)] [[PubMed](#)]
44. Choi, Y.J.; Lee, Y.C.; Kim, J.M.; Kim, J.I.; Moon, J.S.; Lim, Y.J.; Baik, G.H.; Son, B.K.; Lee, H.L.; Kim, K.O.; et al. Triple therapy-based on tegoprazan, a new potassium-competitive acid blocker, for first-line treatment of *Helicobacter pylori* infection: A randomized, double-blind, phase III, clinical trial. *Gut Liver* **2022**, *16*, 535–546. [[CrossRef](#)] [[PubMed](#)]
45. Ahn, B.Y.; Cho, S.J. Potassium-competitive Acid Blockers: A New Therapeutic Strategy for *Helicobacter pylori* Eradication. *Korean J. Helicobacter. Up. Gastrointest. Res.* **2023**, *23*, 174–179. [[CrossRef](#)] [[PubMed](#)]
46. Sunwoo, J.; Oh, J.; Moon, S.; Ji, S.; Lee, S.; Yu, K.S.; Kim, H.; Lee, A.; Jang, I.J. Safety, tolerability, pharmacodynamics and pharmacokinetics of DWP 14012, a novel potassium-competitive acid blocker, in healthy male subjects. *Aliment. Pharmacol. Ther.* **2018**, *48*, 206–218. [[CrossRef](#)] [[PubMed](#)]
47. Yang, A.-Y.; Yoo, H.; Shin, W.; Lee, Y.-S.; Lee, H.; Kim, S.-E.; Kim, A. Size-reduced fexuprazan 20 mg demonstrated the optimal bioavailability and bioequivalence with the reference formulation. *Transl. Clin. Pharmacol.* **2023**, *31*, 40. [[CrossRef](#)] [[PubMed](#)]
48. Kim, S.H.; Cho, K.B.; Chun, H.J.; Lee, S.W.; Kwon, J.G.; Lee, D.H.; Kim, S.G.; Jung, H.Y.; Kim, J.W.; Lee, J.S.; et al. Randomised clinical trial: Comparison of tegoprazan and placebo in non-erosive reflux disease. *Aliment. Pharmacol. Ther.* **2021**, *54*, 402–411. [[CrossRef](#)] [[PubMed](#)]
49. Cho, J.-H.; Jin, S.-Y.; Park, S. Comparison of tegoprazan and proton pump inhibitors for first-line *Helicobacter pylori* eradication: A systematic review with meta-analysis. *Expert Rev. Anti-Infect. Ther.* **2025**, *23*, 227–233. [[CrossRef](#)] [[PubMed](#)]
50. Kwon, Y.H.; Kwon, J.G.; Bae, J.H.; Jo, H.H.; Kim, E.Y.; Jeon, S.W.; Chung, Y.J.; Lee, J.Y.; Park, K.S.; Lee, S.H.; et al. Evaluation of the Safety and Efficacy of K-CAB (Tegoprazan) as Symptomatic Maintenance Therapy in Patients With NERD: A Multicenter, Randomized Double-Blinded Controlled Trial. *Korean J. Helicobacter. Up. Gastrointest. Res.* **2025**, *25*, 371–376. [[CrossRef](#)] [[PubMed](#)]
51. Mermelstein, J.; Mermelstein, A.C.; Chait, M.M. Tegoprazan to treat gastroesophageal reflux disease. *Drugs Today* **2020**, *56*, 715–721. [[CrossRef](#)]
52. Savarino, E.; Zentilin, P.; Savarino, V. NERD: An umbrella term including heterogeneous subpopulations. *Nat. Rev. Gastroenterol. Hepatol.* **2013**, *10*, 371–380. [[CrossRef](#)] [[PubMed](#)]
53. Jeong, H.-C.; Kim, M.-G.; Wei, Z.; Lee, K.-R.; Lee, J.; Song, I.-S.; Shin, K.-H. Integration of a physiologically based pharmacokinetic and pharmacodynamic model for tegoprazan and its metabolite: Application for predicting food effect and intragastric pH alterations. *Pharmaceutics* **2022**, *14*, 1298. [[CrossRef](#)] [[PubMed](#)]
54. Takahashi, N.; Take, Y. Tegoprazan, a Novel Potassium-Competitive Acid Blocker to Control Gastric Acid Secretion and Motility. *J. Pharmacol. Exp. Ther.* **2018**, *364*, 275–286. [[CrossRef](#)] [[PubMed](#)]
55. Abdel-Aziz, Y.; Metz, D.C.; Howden, C.W. potassium-competitive acid blockers for the treatment of acid-related disorders. *Aliment. Pharmacol. Ther.* **2021**, *53*, 794–809. [[CrossRef](#)] [[PubMed](#)]
56. Lim, H.S.; Yoon, H.-j.; Jung, H.-K.; Hong, J.T.; Yoo, M.Y.; Jeong, E.S. Effects of tegoprazan, potassium-competitive acid blocker, on the gastric emptying and postprandial symptoms in healthy humans. *Dig. Dis. Sci.* **2025**, *70*, 1091–1098. [[CrossRef](#)] [[PubMed](#)]
57. Cerf, N.T.; Zerbetto de Palma, G.; Fedosova, N.U.; Filomatori, C.V.; Rossi, R.C.; Faraj, S.E.; Montes, M.R. How ligands modulate the gastric H,K-ATPase activity and its inhibition by tegoprazan. *J. Biol. Chem.* **2024**, *300*, 107986. [[CrossRef](#)] [[PubMed](#)]
58. Inatomi, N.; Matsukawa, J.; Sakurai, Y.; Otake, K. Potassium-competitive acid blockers: Advanced therapeutic option for acid-related diseases. *Pharmacol. Ther.* **2016**, *168*, 12–22. [[CrossRef](#)] [[PubMed](#)]
59. Carrasco-Portugal, M.D.C.; González-Hernández, G.V.; López-Cartagena, P.D.; Flores-Murrieta, F.J. Pharmacokinetics of the potassium-competitive acid blocker tegoprazan administered by the oral route in healthy Mexicans. *Int. J. Clin. Pharmacol. Ther.* **2026**, *64*, 164–168. [[CrossRef](#)] [[PubMed](#)]
60. Liu, J.; Hahn, J. Clinical pharmacokinetics of potassium competitive acid blockers: A systematic review and meta-analysis. *Front. Pharmacol.* **2025**, *16*, 1580969. [[CrossRef](#)] [[PubMed](#)]
61. Kim, D.K.; Lee, K.-H.; Kim, S.-j.; Kim, S.-J.; Lee, S.J.; Park, C.H.; Kim, B.-T.; Song, G.-S.; Moon, B.-S.; Ryu, S.-Y. Effects of tegoprazan, a novel potassium-competitive acid blocker, on rat models of gastric acid-related disease. *J. Pharmacol. Exp. Ther.* **2019**, *369*, 318–327. [[CrossRef](#)] [[PubMed](#)]
62. Han, S.; Choi, H.Y.; Kim, Y.H.; Choi, S.; Kim, S.; Nam, J.Y.; Kim, B.; Song, G.S.; Lim, H.-S.; Bae, K.-S. Comparison of pharmacodynamics between tegoprazan and dexlansoprazole regarding nocturnal acid breakthrough: A randomized crossover study. *Gut Liver* **2022**, *17*, 92. [[CrossRef](#)] [[PubMed](#)]
63. Park, S.; Yang, E.; Kim, B.; Kwon, J.; Jang, I.J.; Lee, S.H. Pharmacokinetic and pharmacodynamic exploration of various combinations of tegoprazan immediate and delayed-release formulations. *Br. J. Clin. Pharmacol.* **2023**, *89*, 2877–2887. [[CrossRef](#)] [[PubMed](#)]
64. Jung, H.K.; Kang, S.J.; Lee, Y.C.; Yang, H.J.; Park, S.Y.; Shin, C.M.; Kim, S.E.; Lim, H.C.; Kim, J.H.; Nam, S.Y.; et al. Evidence-Based Guidelines for the Treatment of *Helicobacter pylori* Infection in Korea 2020. *Gut Liver* **2021**, *15*, 168–195. [[CrossRef](#)] [[PubMed](#)]

65. Park, J.Y.; Hong, S.J.; Choi, I.J.; Baik, G.H.; Kim, S.M.; Park, J.J.; Jeon, S.W.; Kim, K.O.; Lee, S.K.; Jung, H.Y.; et al. Tegoprazan-Based Triple Therapy for *Helicobacter pylori* Eradication: A Phase III Multicenter Randomized Clinical Trial. *Helicobacter* **2026**, *31*, e70106. [\[CrossRef\]](#) [\[PubMed\]](#)
66. Kim, J.Y.; Kim, N.Y.; Kim, S.J.; Baik, G.H.; Kim, G.H.; Kim, J.M.; Nam, R.H.; Kim, H.B.; Lee, D.H.; Jung, H.C.; et al. Regional difference of antibiotic resistance of *Helicobacter pylori* strains in Korea. *Korean J. Gastroenterol.* **2011**, *57*, 221–229. [\[CrossRef\]](#) [\[PubMed\]](#)
67. Ghim, J.L.; Chin, M.C.; Jung, J.; Lee, J.; Kim, S.; Kim, B.; Song, G.S.; Choi, Y.K.; Shin, J.G. Pharmacokinetics and Pharmacodynamics of Tegoprazan Coadministered With Amoxicillin and Clarithromycin in Healthy Subjects. *J. Clin. Pharmacol.* **2021**, *61*, 913–922. [\[CrossRef\]](#) [\[PubMed\]](#)
68. Sunwoo, J.; Ji, S.C.; Oh, J.; Ban, M.S.; Nam, J.Y.; Kim, B.; Song, G.S.; Yu, K.S.; Jang, I.J.; Lee, S. Pharmacodynamics of tegoprazan and revaprazan after single and multiple oral doses in healthy subjects. *Aliment. Pharmacol. Ther.* **2020**, *52*, 1640–1647. [\[CrossRef\]](#) [\[PubMed\]](#)
69. Erah, P.O.; Goddard, A.F.; Barrett, D.A.; Shaw, P.N.; Spiller, R.C. The stability of amoxycillin, clarithromycin and metronidazole in gastric juice: Relevance to the treatment of *Helicobacter pylori* infection. *J. Antimicrob. Chemother.* **1997**, *39*, 5–12. [\[CrossRef\]](#) [\[PubMed\]](#)
70. Han, S.; Choi, H.Y.; Kim, Y.H.; Nam, J.Y.; Kim, B.; Song, G.S.; Lim, H.S.; Bae, K.S. Randomised clinical trial: Safety, tolerability, pharmacokinetics, and pharmacodynamics of single and multiple oral doses of tegoprazan (CJ-12420), a novel potassium-competitive acid blocker, in healthy male subjects. *Aliment. Pharmacol. Ther.* **2019**, *50*, 751–759. [\[CrossRef\]](#) [\[PubMed\]](#)
71. Shang, H.; Mao, H.; Wang, J.; Tian, X.; Zheng, K.; Wang, J. Optimizing acid-related disease management: Insights into potassium-competitive acid blockers and proton pump inhibitors. *Eur. J. Clin. Pharmacol.* **2025**, *81*, 1773–1786. [\[CrossRef\]](#) [\[PubMed\]](#)
72. Kang, S.; Kim, N.H.; Jeong, S.; Kim, J.W.; Moon, J.R.; Lee, Y.S.; Son, J.H. Comparison of Tegoprazan- and Lansoprazole-Based Fourteen-Day Triple Therapies as First-Line Treatments for *Helicobacter pylori* Eradication. *Korean J. Helicobacter. Up. Gastrointest. Res.* **2024**, *24*, 168–174. [\[CrossRef\]](#) [\[PubMed\]](#)
73. Lee, S.P.; Sung, I.-K.; Lee, O.Y.; Choi, M.-G.; Huh, K.C.; Jang, J.-Y.; Chun, H.J.; Kwon, J.-G.; Kim, G.H.; Kim, N. Randomized multicenter study to evaluate the efficacy and safety of fexuprazan according to the timing of dosing in patients with erosive esophagitis. *J. Neurogastroenterol. Motil.* **2025**, *31*, 86. [\[PubMed\]](#)
74. Seong, S.J.; Kweon, O.S.; Yoon, Y.R.; Kim, Y.K.; Kang, Y.N.; Jang, S.Y. Recurrent fexuprazan-induced liver injury following unintentional re-exposure. *Br. J. Clin. Pharmacol.* **2025**, *91*, 3266–3271. [\[CrossRef\]](#) [\[PubMed\]](#)
75. Kim, G.H.; Choi, M.-G.; Kim, J.I.; Lee, S.T.; Chun, H.J.; Lee, K.L.; Choi, S.C.; Jang, J.-Y.; Lee, Y.C.; Kim, J.G. Efficacy and safety of fexuprazan in patients with acute or chronic gastritis. *Gut Liver* **2023**, *17*, 884. [\[CrossRef\]](#) [\[PubMed\]](#)
76. Ramani, A.; Merchant, A.; Cash, B.D. Review of the clinical development of fexuprazan for gastroesophageal reflux-related disease. *Eur. J. Clin. Pharmacol.* **2023**, *79*, 1023–1029. [\[CrossRef\]](#) [\[PubMed\]](#)
77. Hwang, J.G.; Jeon, I.; Park, S.A.; Lee, A.; Yu, K.S.; Jang, I.J.; Lee, S. Pharmacodynamics and pharmacokinetics of DWP14012 (fexuprazan) in healthy subjects with different ethnicities. *Aliment. Pharmacol. Ther.* **2020**, *52*, 1648–1657. [\[CrossRef\]](#) [\[PubMed\]](#)
78. Zou, S.; Ouyang, M.; Cheng, Q.; Shi, X.; Sun, M. Acid-suppressive drugs: A systematic review and network meta-analysis of their nocturnal acid-inhibitory effect. *Pharmacother. J. Hum. Pharmacol. Drug Ther.* **2024**, *44*, 171–183.
79. Scarpignato, C.; Hunt, R.H. Acid Suppressant Therapy: A Step Forward with Potassium-Competitive Acid Blockers. *Curr. Treat. Options Gastroenterol.* **2021**, *19*, 94–132. [\[CrossRef\]](#)
80. Zhuang, Q.; Chen, S.; Zhou, X.; Jia, X.; Zhang, M.; Tan, N.; Chen, F.; Zhang, Z.; Hu, J.; Xiao, Y. Comparative efficacy of P-CAB vs proton pump inhibitors for grade C/D esophagitis: A systematic review and network meta-analysis. *Am. J. Gastroenterol.* **2024**, *119*, 803–813. [\[CrossRef\]](#) [\[PubMed\]](#)
81. He, E.T.; Samnani, S.; Yaghoobi, M.; Yuan, Y.; Hunt, R.H. S934 Potassium-Competitive Acid Blockers Versus Proton Pump Inhibitors in Patients With Grade C and D Erosive Esophagitis: A Meta-Analysis. *Am. J. Gastroenterol.* **2025**, *120*, S201. [\[CrossRef\]](#)
82. Kim, M.-S.; Lee, N.; Lee, A.; Chae, Y.-J.; Chung, S.-J.; Lee, K.-R. Model-based prediction of acid suppression and proposal of a new dosing regimen of fexuprazan in humans. *Pharmaceuticals* **2022**, *15*, 709. [\[CrossRef\]](#) [\[PubMed\]](#)
83. Hong, S.; Ju, J.H.; Lee, S.-H.; Hong, S.-J.; Kim, S.-h.; Ahn, G.Y.; Jung, J.H.; Hur, J.-W.; Ha, Y.-J.; Park, J.K. Comparing the efficacy and safety of fexuprazan and lansoprazole for the prevention of nonsteroidal anti-inflammatory drug-induced peptic ulcer. *Gut Liver* **2025**, *19*, 685. [\[CrossRef\]](#) [\[PubMed\]](#)
84. Oh, J.; Yang, E.; Jang, I.-J.; Lee, H.; Yoo, H.; Chung, J.-Y.; Lee, S.; Oh, J. Pharmacodynamic and Pharmacokinetic Drug Interactions between Fexuprazan, a Novel Potassium-Competitive Inhibitor, and Aspirin, in Healthy Subjects. *Pharmaceutics* **2023**, *15*, 549. [\[CrossRef\]](#) [\[PubMed\]](#)
85. Hwang, I.; Ji, S.C.; Oh, J.; Kim, H.; Cha, H.; Kim, J.; Lee, C.S.; Yu, K.S.; Lee, S. Randomised clinical trial: Safety, tolerability, pharmacodynamics and pharmacokinetics of zastaprazan (JP-1366), a novel potassium-competitive acid blocker, in healthy subjects. *Aliment. Pharmacol. Ther.* **2023**, *57*, 763–772. [\[CrossRef\]](#) [\[PubMed\]](#)

86. Wang, Y.; Dai, X.; Zhang, X. Network meta-analysis of comparing different dosages of potassium-competitive acid blocker with proton-pump inhibitor in acid-related disorders. *Clin. Transl. Gastroenterol.* **2024**, *15*, e00776. [\[CrossRef\]](#) [\[PubMed\]](#)
87. Savarino, V.; Antonioli, L.; Fornai, M.; Marabotto, E.; Demarzo, M.G.; Zingone, F.; Ghisa, M.; Barberio, B.; Zentilin, P.; Ribolsi, M. An update of pharmacology, efficacy, and safety of vonoprazan in acid-related disorders. *Expert Rev. Gastroenterol. Hepatol.* **2022**, *16*, 401–410. [\[CrossRef\]](#) [\[PubMed\]](#)
88. Oshima, T. Proton pump inhibitor versus potassium-competitive acid blocker in gastroesophageal reflux disease. *JGH Open Open Access J. Gastroenterol. Hepatol.* **2024**, *8*, e13104. [\[CrossRef\]](#)
89. Jin, T.; Wu, W.; Zhang, L.; Xuan, H.; Zhang, H.; Zhong, L. The efficacy and safety of Vonoprazan and Tegoprazan in Helicobacter pylori eradication: A comprehensive systematic review and meta-analysis of randomized controlled trials. *Ther. Adv. Gastroenterol.* **2025**, *18*, 17562848251314801. [\[CrossRef\]](#) [\[PubMed\]](#)
90. Li, Y.; Choi, H.; Leung, K.; Jiang, F.; Graham, D.Y.; Leung, W.K. Global prevalence of Helicobacter pylori infection between 1980 and 2022: A systematic review and meta-analysis. *Lancet Gastroenterol. Hepatol.* **2023**, *8*, 553–564. [\[CrossRef\]](#) [\[PubMed\]](#)
91. Jung, H.-K.; Tae, C.H.; Song, K.H.; Kang, S.J.; Park, J.K.; Gong, E.J.; Shin, J.E.; Lim, H.C.; Lee, S.K.; Jung, D.H. 2020 Seoul consensus on the diagnosis and management of gastroesophageal reflux disease. *Korean J. Med.* **2022**, *97*, 70–92. [\[CrossRef\]](#)
92. Yang, E.; Ji, S.C.; Jang, I.-J.; Lee, S. Evaluation of CYP2C19-Mediated Pharmacokinetic Drug Interaction of Tegoprazan, Compared with Vonoprazan or Esomeprazole. *Clin. Pharmacokinet.* **2023**, *62*, 599–608. [\[CrossRef\]](#) [\[PubMed\]](#)
93. Kang, S.H.; Moon, H.S.; Sung, J.K.; Kim, S.M.; Kim, K.B.; Lee, S.W.; Cho, Y.S.; Bang, K.B.; Song, K.H. Assessment of the efficacy of on-demand tegoprazan therapy in gastroesophageal reflux disease through a randomized controlled trial. *Sci. Rep.* **2025**, *15*, 168. [\[CrossRef\]](#) [\[PubMed\]](#)
94. Savoldi, A.; Carrara, E.; Graham, D.Y.; Conti, M.; Tacconelli, E. Prevalence of Antibiotic Resistance in Helicobacter pylori: A Systematic Review and Meta-analysis in World Health Organization Regions. *Gastroenterology* **2018**, *155*, 1372–1382.e17. [\[CrossRef\]](#) [\[PubMed\]](#)
95. Malfertheiner, P.; Megraud, F.; Rokkas, T.; Gisbert, J.P.; Liou, J.M.; Schulz, C.; Gasbarrini, A.; Hunt, R.H.; Leja, M.; O'Morain, C.; et al. Management of Helicobacter pylori infection: The Maastricht VI/Florence consensus report. *Gut* **2022**, *71*, 1724–1762. [\[CrossRef\]](#) [\[PubMed\]](#)
96. Rawla, P.; Sunkara, T.; Ofosu, A.; Gaduputi, V. Potassium-competitive acid blockers—Are they the next generation of proton pump inhibitors? *World J. Gastrointest. Pharmacol. Ther.* **2018**, *9*, 63–68. [\[CrossRef\]](#) [\[PubMed\]](#)
97. Jung, Y.S.; Kim, S.; Kim, H.Y.; Noh, S.J.; Park, J.H.; Park, C.H. 7-day versus 14-day tegoprazan-based triple therapy to treat Helicobacter pylori infection: Real-world evidence. *J. Gastroenterol. Hepatol.* **2022**, *37*, 1911–1918. [\[CrossRef\]](#) [\[PubMed\]](#)
98. Wei, Z.; Jeong, H.C.; Kim, M.G.; Shin, K.H. Prediction of the Drug-Drug Interaction Potential between Tegoprazan and Amoxicillin/Clarithromycin Using the Physiologically Based Pharmacokinetic and Pharmacodynamic Model. *Pharmaceuticals* **2023**, *16*, 360. [\[CrossRef\]](#) [\[PubMed\]](#)
99. Jeon, J.-Y.; Kim, S.-Y.; Moon, S.J.; Oh, K.; Lee, J.; Kim, B.; Song, G.S.; Kim, M.-G. Pharmacokinetic Interactions between Tegoprazan and Metronidazole/Tetracycline/Bismuth and Safety Assessment in Healthy Korean Male Subjects. *Clin. Ther.* **2021**, *43*, 722–734. [\[CrossRef\]](#) [\[PubMed\]](#)
100. Oshima, T.; Miwa, H. Potent Potassium-competitive Acid Blockers: A New Era for the Treatment of Acid-related Diseases. *J. Neurogastroenterol. Motil.* **2018**, *24*, 334–344. [\[CrossRef\]](#) [\[PubMed\]](#)
101. Zhuang, Q.; Liao, A.; He, Q.; Liu, C.; Zheng, C.; Li, X.; Liu, Y.; Wang, B.; Liu, S.; Zhang, Y.; et al. The efficacy and safety of fexuprazan in treating erosive esophagitis: A phase III, randomized, double-blind, multicenter study. *J. Gastroenterol. Hepatol.* **2024**, *39*, 658–666. [\[CrossRef\]](#) [\[PubMed\]](#)
102. Jung, B.W.; Park, C.H.; Eun, C.S. Preference for On-Demand Fexuprazan Therapy in Gastroesophageal Reflux Disease: A Prospective Cohort Study. *J. Pers. Med.* **2025**, *15*, 19. [\[CrossRef\]](#) [\[PubMed\]](#)
103. Hunt, R.H.; Scarpignato, C. Potassium-Competitive Acid Blockers (P-CABs): Are They Finally Ready for Prime Time in Acid-Related Disease? *Clin. Transl. Gastroenterol.* **2015**, *6*, e119. [\[CrossRef\]](#) [\[PubMed\]](#)
104. Pharmaceuticals, F.P.S. *Sebel Pharmaceuticals Announces Successful Completion of Phase 3 TRIUMpH Program of Tegoprazan in GERD with Positive 24-Week Maintenance Phase Results*; Sebel Pharmaceuticals Inc.: Roswell, GA, USA, 2025.
105. inno, N. *K-CAB, HK inno.N's New Novel Drug for GERD Treatment, Submits an NDA to the U.S. FDA; HK inno.N's Novel Drug 'K-CAB' Officially Begins the U.S. New Drug Application (NDA) Process for FDA Approval*; inno.N.: Cheongju, Republic of Korea, 2026.
106. Alsabbagh, M.; Alsabbagh Alchirazi, K.; Baliss, M.; Bazarbashi, A.; Obeidat, K.; Mohamed, A.N. S1035 Potassium-Competitive Acid Blockers vs Proton Pump Inhibitors: A Real-World Comparative Effectiveness Study. *Am. J. Gastroenterol.* **2025**, *120*, S223. [\[CrossRef\]](#)
107. Shiratori, Y.; Kim, Y.; Fukuda, K.; Suzuki, N.; Hayakawa, Y.; Tsuji, Y.; Nishida, T.; Kiyotoki, S.; Sumiyoshi, T.; Hasatani, K.; et al. Long-term use of potassium-competitive acid blockers and proton pump inhibitors and the risk of metachronous gastric cancer after H. pylori eradication: A multicenter cohort study. *Gastric Cancer* **2026**, *29*, 347–356. [\[CrossRef\]](#) [\[PubMed\]](#)

108. Miwa, H.; Shiotani, A.; Takeda, M.; Eda, M.; Kato, T.; Yajima, T. Health Care Cost Analysis of PPI or P-CAB-First Treatment in Patients With Gastroesophageal Reflux Disease. *J. Clin. Gastroenterol.* **2023**, *57*, 370–379.
109. Patel, A.; Laine, L.; Moayyedi, P.; Wu, J. AGA Clinical Practice Update on Integrating Potassium-Competitive Acid Blockers Into Clinical Practice: Expert Review. *Gastroenterology* **2024**, *167*, 1228–1238. [[CrossRef](#)] [[PubMed](#)]

Disclaimer/Publisher’s Note: The statements, opinions and data contained in all publications are solely those of the individual author(s) and contributor(s) and not of MDPI and/or the editor(s). MDPI and/or the editor(s) disclaim responsibility for any injury to people or property resulting from any ideas, methods, instructions or products referred to in the content.