given 40 mg of esomeprazole. Blood samples for fasting gastrin and pharmacokinetic analyses were taken at scheduled time-points for eight hours after the first and fifth doses. Liquid chromatography was used to analyse esomeprazole, and radioimmunoassay was used to quantify gastrin concentrations [8].

Pharmacokinetic study and microbiota analyses

There were 30 volunteers in total. Females showed a greater baseline gastrin (pM) than males, with 12 (IQR 10–15) vs. 7 (IQR 4–11) (p = 0.03). Gastrin levels in the study cohort increased from 10 (IQR 6–14) to 15 (IQR 13–20) (p = 0.0002). From day 1 to day 5, esomeprazole serum levels increased by an average of 299.8 ng/mL (p.001)[9]. There were no significant sex-related differences in the pharmacokinetic characteristics of esomeprazole when males and females were compared. On day 5, there was no significant association between the AUC and the gastrin level (p = 0.15). After four days of PPI medication, serum gastrin levels in healthy participants increased considerably. From day 1 to day 5, serum esomeprazole levels increased significantly. There was no sex-related difference in gastrin and esomeprazole concentrations, and no significant sex-related variation in pharmacokinetic parameters [10].

In a two-part randomised crossover trial, eight healthy dogs were used to investigate the pharmacokinetic characteristics of esomeprazole following intravenous (IV) and oral (po) administration. The dogs were fasted for at least 12 hours before receiving esomeprazole intravenously (dose range 0.93–1.48 mg/kg) or orally (dose range 0.95–1.50 mg/kg). The dogs were given an alternative therapy after a one-week washout period. Plasma esomeprazole concentrations were measured using ultra-high-performance liquid chromatography–mass spectrometry after serial blood samples were taken at preset time points. Analyses of no compartmental pharmacokinetics were carried out. The area under the plasma concentration/time curve (AUC) and maximal plasma concentration (C_{max}) were then standardised to a 1.0 mg/kg esomeprazole dose, resulting in AUC/dose. For the IV and po formulations, the dose-normalized peak plasma concentration (C_{max}) values were 4.06 g/mL (2.47–4.57 g/mL) and 1.04 g/mL (0.31–1.91 g/mL), respectively. For the po formulation, the median (range) time to peak concentration (T_{max}) was 105 minutes (45–360 minutes). The IV formulation had a median (range) plasma terminal half-life (t12) of 45.56 minutes (39.43–64.20 minutes) while the enteric-coated po formulation had a median (range) plasma terminal half-life (t12) of 63.97 minutes (44.02–109.94 minutes). Po bioavailability was 63.33% (range 32.26%–79.77%) on average (range 32.26%–79.77%). Both the po and IV formulations were well tolerated in clinical trials, with few adverse effects reported.

Stability

The stabilities of Olsalazine, 5-ASA, and N-Ac-5-ASA under various conditions were presented. All stability data were within a ± 15.0% deviation range, suggesting that no significant stability-related problems occurred during routine sample analysis and sample storage.

Conclusion

In the present study, pharmacokinetic and gut microbiota analyses revealed the effect of L. acidophilus on the metabolism of Olsalazine from three levels. At the level of the pharmacokinetic characteristics, L. acidophilus barely had an influence on the pharmacokinetic parameters of Olsalazine, 5-ASA, and N-Ac-5-ASA. And most remarkably, the exposures of Olsalazine, 5-ASA, and N-Ac-5-ASA in plasma and feces were not affected by L. acidophilus. At the level of metabolic microbiota, L. acidophilus did not alter the total relative abundance of azoR and NAT-associated gut microbiota families, genera, and species. At the level of metabolic enzymes, L. acidophilus did not change the