

To the Editor:

Drs. Logan and Baeulne suggested that treatment with enteric-coated peppermint oil reduced small-intestinal bacterial overgrowth in a patient with irritable bowel syndrome (*Altern Med Rev* 2002;7(5):410-417). That conclusion was based on the results of breath hydrogen tests administered before and after treatment with peppermint oil. While breath hydrogen excretion during the first 60 minutes of the test was lower after treatment than at baseline, the opposite was true at 120 minutes (personal communication, Logan AC).

The authors suggested that hydrogen excretion at 120 minutes is more reflective of colonic bacteria than of small-intestinal organisms; therefore, the data at 120 minutes were not included in their report. However, if peppermint oil induces a delay in gastric emptying, it would presumably delay the rise in breath hydrogen excretion, which might erroneously be interpreted as an antimicrobial effect, if only the first 60 minutes of the test were considered.

Although peppermint oil has been reported to have antimicrobial activity *in vitro*, the same effect may not occur *in vivo*. Garlic, for example, has been shown to inhibit the growth of *Helicobacter pylori in vitro*, but has little or no antibacterial effect in a person who is infected with *H. pylori*.¹

While peppermint oil is clearly beneficial for patients with irritable bowel syndrome, its efficacy could be explainable solely on the basis of its antispasmodic effect.

Sincerely,
Alan R. Gaby, MD

Reference

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NOTICE TO OUR READERS: Effective January 2003 *Alternative Medicine Review* will be published quarterly. Issues will be February (1st Quarter), May (2nd Quarter), August (3rd Quarter), and November (4th Quarter). Subscription rates will remain the same.

Response to Letter to the Editor:

We appreciate the comments of Dr. Gaby regarding our observation of improved clinical symptoms and reduced hydrogen production in a patient with irritable bowel syndrome.¹

We certainly agree that the efficacy of peppermint oil may be due strictly to its effect on motility. The lactulose hydrogen breath test has obvious limitations as a means to accurately determine *in vivo* antimicrobial activity.

Dr. Gaby contends that our interpretation of the post-treatment breath hydrogen data may have been erroneous due to peppermint oil inducing a delay in gastric emptying. Human research clearly shows, however, that peppermint oil accelerates the gastric emptying rates in both healthy adults and in those with dyspepsia.² If peppermint oil induced a delay in gastric emptying, it would be contraindicated in patients with IBS, dyspepsia, and chronic fatigue syndrome, as a delay in gastric emptying is evident in such conditions.²⁻⁴

An additional point to consider is that the follow-up lactulose hydrogen breath test was administered six days after the peppermint oil treatment was completed. Investigators have examined the pharmacokinetic parameters of menthol, the main component of peppermint oil. Results indicate that when 180 mg of enteric-coated peppermint oil is orally administered, the half-life of the menthol is 3 1/2 hours.⁵ Therefore, it is unlikely that after six days of non-treatment, peppermint oil was still exerting a significant effect on the gastric emptying time of the lactulose drink.

As for the *in vivo* anti-*Helicobacter pylori* effects of garlic, the limited clinical research has been the subject of criticism.^{6,7} More recent research indicates that the anti-*H. pylori* effect of garlic may be very dependent on the content of a phenolic phytoalexin called allixin.⁸ The allixin content of a garlic bulb is a product of growth conditions and is highly variable. International researchers continue to investigate the anti-*H. pylori* effects of garlic as no firm conclusions can be drawn at this time concerning its clinical use.^{9,10}

In the meantime, our results suggest that enteric-coated peppermint oil may, through a number of mechanisms, have some effect on small intestinal bacteria. Based on the *in vitro* data, a direct antimicrobial effect is possible. However, the reductions in breath hydrogen may also have to do with alterations in pH or perhaps motility related migration of bacteria resulted in decreased small intestinal bacterial numbers and increased counts in the large intestine.

It is hoped that quality research will determine if botanical interventions can live up to the *in vitro* promise. Although clinically useful, neither the lactulose hydrogen breath test or the urea breath test for *H. pylori* are quantitative regarding bacterial counts. Current methods to more accurately determine bacterial numbers in the stomach and small intestine are more invasive but may be required to provide the much-needed answers regarding *in vivo* antimicrobial activity.

Respectfully,

Alan C. Logan, ND, FRSH

Tracey M. Beaulne, ND

CFS-FM Integrative Care Centre; Toronto, ON

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