## The effect of teas on malabsorption of carbohydrates

Dear Sir:

I read with great interest the article "An Extract of Black, Green, and Mulberry Teas Causes Malabsorption of Carbohydrate but not of Triacylglycerol in Healthy Volunteers" by Zhong et al (1) in the September 2006 issue of the Journal. Zhong et al provide an excellent introduction to the health benefits of tea extracts. Through the use of an excellent protocol design, black, green, and mulberry tea extracts were tested with special emphasis on their interference with carbohydrate and triacylglycerol absorption via their ability to induce the malabsorption of carbohydrate or fat.

The authors pointed out that no studies in humans or animals have shown that tea preparations cause the malabsorption of carbohydrates or fat. The Asian belief that drinking tea promotes good health and longevity is gaining scientific merit (2). Dullo et al (3) reported that the consumption of green tea extract elevates both the metabolic rate and the rate of fat oxidation in humans.

One or 2 additional points can be made on this topic. First, the racial composition of the study participants was not given. Would the outcomes have been influenced if the study participants were African American or Latino, given the higher prevalence of diabetes and obesity in these groups (4; E Caballero, M Heisler, NL Agbayani, unpublished observation, 2006)? In addition, can this study be replicated by using a different protocol design and a larger study group?

The ability of a tea extract to inhibit carbohydrate absorption will continue to be an important issue in the management of weight control and in the treatment of diabetes. The study also indicated that carbohydrate malabsorption induced by tea extracts could influence blood glucose concentrations. Future studies are needed to include African American and Latino populations. If such studies have promising results, this approach could positively affect the challenging health disparities of various populations.

The author had no conflict of interest to declare.

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## Vitamin D supplementation for non-Western pregnant women: the British experience

Dear Sir:

We read with interest the article by van der Meer et al (1) on Vitamin D deficiency in pregnant non-Western women in the Netherlands. We would like to share the British experience and will mainly discuss 2 aspects of the issue. First, we will address evidence of vitamin D deficiency in pregnant Asian women in the United Kingdom and the benefit of routine antenatal supplementation. Second, we will discuss the confusion that has arisen from conflicting recommendations from the Department of Health and National Institute of Clinical Excellence on antenatal vitamin D supplementation.

There has long been evidence of vitamin D deficiency in pregnant Asian women and their newborns in the United Kingdom (2, 3), as assessed by maternal and neonatal serum 25-hydroxycholecalciferol concentrations. Several studies have shown the benefit of vitamin D supplementation in pregnant women in the United Kingdom, including a double-blind randomized controlled trial that showed increases in maternal serum 25-hydroxycholecalciferol concentrations after vitamin D supplementation (4–6). Maternal nutritional status also benefited as assessed by maternal weight gain and concentrations of retinol-binding protein and thyroid binding prealbumin (4). Cord blood 25-hydroxycholecalciferol concentrations also improved (6, 7). After birth, there was a significantly lower incidence of asymptomatic and symptomatic hypocalcaemia in the supplemented group and evidence of significant improvements in weight and length of the infant up to 1 y of age (4).

Considering the abovementioned evidence, it seems logical that pregnant British Asian women should routinely be supplemented with vitamin D. Indeed, the Department of Health (DOH) recommendations, based on Committee on Medical Aspects of Food Policy (COMA), are that all pregnant and breastfeeding women should receive 10 $\mu$g (400 IU) vitamin D/d (8). However, the National Institute of Clinical Excellence (NICE) (9) recommends, based on a Cochrane review (10), that there is insufficient evidence to evaluate the effectiveness of vitamin D in pregnancy, and, in the absence of evidence of benefit, vitamin D supplementation should not be offered routinely to pregnant women. The same Cochrane review, however, recommends that vitamin D supplementation in the later part of pregnancy should be considered in vulnerable groups, such as Asian women living in Northern Europe, and possibly in those living in geographic areas with long winters (eg, the United Kingdom and the Netherlands). The contradictory statements from the DOH and NICE have led to confusion in antenatal clinics in hospitals and in

general practice. In a recent survey of general practices in the Thames Valley area and Lambeth (where 67.9% of practices had Asian or African Caribbean populations constituting >8% of the total population), none were supplementing pregnant women with vitamin D (11).

On the basis of the abovementioned evidence, we believe that, at a minimum, the DOH recommendations should be followed, and all pregnant and breastfeeding women should receive 10 μg (400 IU) vitamin D/d. We also echo the concerns raised by others concerning the recommendations of NICE (12, 13) and call for clarity.

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## Insulin secretion and capsaicin

Dear Sir:

Many studies in the medical literature have indicated that the consumption of chili-containing meals increases energy expenditure and fat oxidation. Moving toward this direction, a new study regarding the metabolic effect of a capsaicin chili–containing meal after the consumption of a bland diet and a chili-blend–supplemented diet was performed by Ahuja et al (1). The authors concluded that regular consumption of chili may attenuate postprandial hyperinsulinemia. To our knowledge, many studies have reported different effects of capsaicin on glucose metabolism, implying that several factors may be responsible for or may interact with capsaicin at a molecular level, receptor level, or both. In one of the first studies of the effects of capsaicin on glucose metabolism, Karlsson et al (2) showed in mice that both the early (1-min) insulin secretory response to intravenous glucose and glucose elimination were potentiated after capsaicin administration, whereas basal insulin concentrations were not affected by capsaicin. Similarly, in their experimental studies in rats, Gram et al (3) reported that the mean blood glucose concentration decreased and the plasma insulin concentration was unchanged during an oral-glucose-tolerance test (OGTT) after capsaicin administration, whereas the administration of a tolerable analogue of capsaicin suitable for in vivo use (resiniferatoxin) was accompanied by an increased insulin response to oral glucose (4). In addition, Akiba et al (5) showed in another study in rats that systemic administration of 10 mg capsaicin/kg (subcutaneously) dose-dependently increased insulin secretion and plasma insulin concentrations 1 h after treatment. Other experimental studies conducted by Tolan at al (6, 7) showed that purified capsaicin caused a decrease in blood glucose concentrations in dogs during an OGTT and a concomitant elevation in plasma insulin concentrations. Another study of insulin metabolism after oral application of capsaicin by Domotor et al (8) conducted in healthy human subjects showed that, although the plasma concentrations of insulin increased from 90 to 165 min after glucose loading, there were no significant differences between the results obtained with and without capsaicin administration. Although many studies of the role of capsaicin in carbohydrate metabolism have been performed on animals, most of which showed that (contrary to the results of Ahuja et al) capsaicin is associated with increased insulin secretion, most of these studies were conducted under different experimental circumstances and, although adding some evidence for a better understanding of the role of capsaicin in carbohydrate metabolism, their results are hardly comparable. However, to date, there is a paucity of human studies on the effects of capsaicin on glucose metabolism. In conclusion, it seems that several influencing and confounding factors, as well as differences in animal and human metabolisms, could at least partly explain why the results of the study by Ahuja et al contrasted with those of other studies. Although the time of a clinical application of capsaicin analogues in treating metabolic disorders such as diabetes is still distant, and even though there is a lack of more solid evidence, we believe that larger-scale clinical trials are needed.

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