“Too Hot” Or “Too Cold”: Effects of Meal Temperature on Gastric Function

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Functional gastrointestinal disorders (FGIDs) are characterized by the presence of chronic or recurrent symptoms thought to originate in the GI tract. FGIDs are associated with a complex web of predisposing factors such as genetics, early-life stress events (i.e., sexual abuse) and other psychosocial factors, and alterations in the composition of the gut microbiome and immune activation [irritable bowel syndrome (IBS)] [1]. Interestingly, although most of the FGID symptoms are related to food ingestion, the relation of food and diet with FGIDs is lacking. Recently, some studies have renewed interest in the relation of food ingestion with FGIDs; For example, the role of food sensitivity (i.e., non-celiac-disease gluten sensitivity) and symptoms related to ingestion of fermentable oligo-, di-, monosaccharides, and polyols (FODMAPs) have recently received much attention in the field of FGID research [2, 3].

The physiology of food intake is a complex process which includes sensory and motor responses that facilitate propulsion, digestion, and absorption of nutrients. Multiple receptors are responsible for food and nutrient sensing in the upper gastrointestinal tract [4]. At least three types of receptors are activated by food ingestion: mechanoreceptors, chemoreceptors, and thermoreceptors [4]. Meal ingestion activates mechanosensitive and nutrient-sensing

pathways. Gastric distension triggers stretch- and tension-sensitive mechanoreceptors that activate regions within the central nervous system, in addition to altering vagovagal reflexes that relax the proximal stomach, associated with the sensations of satiety and fullness [5].

Patients with functional dyspepsia (FD) have impaired gastric accommodation in response to meal intake [6]. Nevertheless, information regarding the influence of meal temperature is scarce and controversial; For example, some studies report that a hot meal significantly accelerates gastric emptying, although other studies have reported opposite results [7, 8]. Few studies have reported the effect of temperature on visceral sensation [9].

In this issue, Rui-Feng et al. [10] report the results of a small but well-conducted study in which the authors investigate the effects of consumption of liquid nutrients at different temperatures on gastric accommodation, sensitivity, and gastric wall compliance in patients with FD with epigastric pain syndrome (EPS). The study is notable for several reasons: (1) few studies focused on EPS subjects have been published; (2) the authors perfused a fluid nutrient solution coupled with intragastric pressure (IGP) monitoring, a new technique that is less invasive and better tolerated than barostat measurements; and (3) IGP was measured using a high-resolution manometry (HRM) system, a novel and promising method. The authors report that cold stimulation (ingestion of liquid nutrient at 8 °C) increases IGP and visceral sensitivity while reducing gastric volume, associated with epigastric discomfort and pain in FD-EPS patients.

Cold temperature may induce smooth muscle contraction, but also may stimulate transient receptor potential (TRP) channel receptors, which belong to the mammalian TRP superfamily. Menthol and capsaicin are among the agonists that may activate TRP receptors [11]. Moreover,

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temperature is a well-known activator of TRP channels; specifically, low temperature activates TRPM8 and TRPA1, and high temperature activates TRPV1,2,3,4 and TRPM4 receptors [11].

Thus, Rui-Feng et al. provide good clinical evidence that temperature, in this case cold liquids, may alter sensory gastric motor function in FD patients with EPS. Hence, the temperature of a nutrient or meal challenge should be taken into account in studies of gastric motor function.

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