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Stomach and Gallbladder under Fat Control?

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Ingestion of food triggers a complex interplay of various factors, for example gastrointestinal hormones and nervous reflexes, producing a concert of physiological or pathophysiological events in the upper gastrointestinal tract. Such events comprise motility, secretions of stomach and pancreas, absorption, and gallbladder contraction. The responses to meals have traditionally been considered in three phases: cephalic, gastric, and intestinal, allowing the reduction of complex events to manageable descriptions [1]. This enables us to conceptually think about defined factors and their impact on the digestive process such as physical properties of meals, meal constituents, and medications. In this context key areas of recent interest are the regulation of satiety and body weight. Of certain interest is the question how single nutrients may impact on upper gastrointestinal functions, possibly allowing clearer dietary recommendations for overweight people. This comes with the recently more and more discussed idea of the stomach significantly contributing to the quality of metabolic control, especially in those with impaired glucose tolerance and type 2 diabetes mellitus. Classic studies decades ago by Hunt and colleagues [2, 3] prompted research up to the present day addressing the question how the nutrients of a meal influence the rate of its emptying from the stomach. In this context a wealth of data has been collected concerning the impact of glucose/carbohydrates, protein, and fat [1]. Still, it seems that the meal proportion of carbohydrate and protein have only minor effects on gastric emptying, prompting more focus on the impact of fat. With fat in the stomach or duodenum, delay and also acceleration of gastric emptying have been reported, depending on the utilized experimental setting [1].

In this issue of *Digestion*, Degen et al. [4] shed more light onto this somehow controversial area while looking at it from a different perspective. They addressed the question whether fat hydrolysis is mandatory for the ability of lipid to trigger regulation of upper gastrointestinal function such as gallbladder contraction and inhibition of gastric emptying. They employed orlistat, an inhibitor of gastrointestinal lipases, as a tool to study the relationship between fat hydrolysis, CCK secretion, gastric emptying, and gallbladder contraction. Their data set the stage for CCK as a key player in the regulation of postprandial gallbladder contraction and gastric emptying dynamics. They suggest that a CCK-plasma level-dependent action is driven by intraduodenal lipolysis and, clearly, fat digestion may be important in the regulation of upper gastrointestinal tract function. This study was done in healthy, normal-weight male volunteers which already points to its limitation. Such people are not under orlistat in normal life. How will overweight individuals

behave in such studies under orlistat? Do we only look upon acute effects, bearing in mind that adaptation of gastric function to the effects of diet is under discussion [1]? A high-fat diet may not alter the stomach-emptying rate of test meals, and adaptation after a high-fat

diet may prompt acceleration rather than delay of the emptying of a test meal [5]. In any case, this work contributes to an interesting and worthwhile discussion probably preparing the way for further study.

References

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