

Dietary Proteins: A Target for Appetite Regulation

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Obesity has reached global epidemic proportions, and is a major contributor to the global burden of chronic disease and disability [1]. Regulation of food intake and management of body weight are important components of the therapeutic approaches targeting obesity. Many factors have been identified as significant determinants of the control of energy intake and body weight [2,3], including dietary macronutrient composition [4]. This has led to an increased interest in altering the proportions of macronutrients in weight-reducing diets without overtly specifying caloric intake levels. High-carbohydrate low-fat diets have been originally recommended as the macronutrient composition of choice for improved weight loss responses [5]. However, a worsening of the metabolic profile was frequently described following these diets in addition to a poor long-term compliance to such a dietary pattern [6,7]. Consequently, attention was diverted towards low-carbohydrate high-protein diets, which appeared to be more effective in inducing weight loss under ad libitum energy intake conditions [8] and to generate more favourable effects on the metabolic risk factors for cardiovascular diseases [9].

The benefits of high-protein diets with respect to food intake and body weight regulation have been attributed mainly to the established satiating properties of dietary proteins. The satiety effect of protein is greater than that of carbohydrate and fat (cal/cal) [10]. In fact, consumption of dietary proteins decreases hunger ratings, motivation to eat and subsequent food intake to a greater extent than fat and carbohydrate [11-13]. Aside from the higher thermogenic effect of dietary proteins in the postprandial period [14], protein-induced satiety has been strongly linked to the release of a number of gut-derived hormones implicated in the regulation of the ingestive behaviour including ghrelin, peptide YY (PYY), cholecystokinin (CCK) and glucagon-like peptide 1 (GLP-1) [15]. Peptide YY, CCK and GLP-1, released from different areas of the intestine, act as short-term satiety signals and were found to suppress hunger and decrease food intake in humans [16,17]. On the contrary, ghrelin, a stomach-derived hormone, stimulates hunger and food intake and was thus suggested to play a physiological role in meal initiation in humans [18]. Since suppression of food intake was only observed when protein was administered enterally, the satiating effects of proteins result mainly from an interaction of dietary proteins with gut-endocrine axis [19]. By interacting with the gastrointestinal tract via their physico-chemical properties, amino acid composition and bioactive peptides encrypted in their amino acid sequences, dietary proteins mediate the release of endogenous gut peptides by stimulating chemoreceptors and/or activating satiety hormone receptors [20].

Based on short-term studies, a meal with higher protein content was depicted as the most effective in generating optimal secretion patterns of various satiety hormones including ghrelin and PYY. In normal-weight males and females, although high-carbohydrate meals induced a greater overall postprandial ghrelin suppression than dietary protein or fat [21-23], high-protein meals had a longer-lasting suppressive effect on circulating ghrelin levels in comparison to high-carbohydrate and high-fat meals [23,24]. With respect to PYY, dietary fat, when investigated as a single nutrient, was the most potent in stimulating PYY release [25,26]. When mixed meals were used, both high-fat [27]

and high-protein [15,27] meals resulted in the greatest increase in plasma PYY levels in normal-weight individuals. The beneficial impact of high-protein meals on gut hormone release has been replicated in obese men and women [15,28] but not in other populations. In hyperinsulinemic normoglycemic men [29] and in obese dyslipidemic men with metabolic syndrome defined according to the criteria of the International Diabetes Federation [30], high-protein meals maintained a longer-lasting attenuated profile of ghrelin in comparison to high-carbohydrate and high-fat meals but did not differentially influence PYY circulating levels. It should be noted though that high-protein meals resulted in more favourable metabolic responses, characterized by lower postprandial glucose and insulin surges and reduced postprandial triglyceride responses, in hyperinsulinemic men in comparison to the other two meals [29].

The source of dietary proteins is another factor influencing satiety signals. Isocaloric test meals of casein and soy were more satiating than whey in lean healthy individuals [14]. In addition, whey protein suppressed short-term food intake to a greater extent than egg albumen and soy protein isolate in young men [31]. Protein source influences differently various physiologic and metabolic responses of the gastrointestinal tract including digestion, gastric emptying, the composition of intestinal effluents and the rate of amino acid absorption and oxidation, all of which shape the patterns of gut hormone release and the subsequent impact on satiety and food intake [20]. For instance, peptides arising from the digestion of whey and casein interact with GLP-1 receptors to suppress food intake in rats [32], whereas those from casein and soy protein decrease food intake through opioid and CCK receptors in rats [33].

In conclusion, increasing the percent of calories derived from dietary proteins in a meal appears as a potential therapeutic strategy to boost the satiating power of the diet of diverse population groups with distinct metabolic profiles by prolonging post-meal satiety sensations, delaying the onset of the next meal, increasing inter-meal intervals and consequently decreasing daily energy intakes.

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