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The short- and long-term effects of food intake on thermogenesis

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The maintenance of normal body mass is achieved by complex mechanisms, which involve a close interaction between feeding-related and thermoregulatory processes in mammals (**Fig. 1**). The balance between energy intake and total energy expenditure is crucial to maintain the optimal body mass. Food intake can be stimulated by different factors, which are directly (e.g., fasting, hunger, food cues) or indirectly associated with feeding (e.g., cold, physical activity).

Short-term thermal effects over several hours are induced following a single meal. This is distinguishable from long-term components of diet-induced thermogenesis, which can be caused by chronic overfeeding lasting one week to years.[1-3] The two major components of the short-term effects are thermic effect of feeding (TEF) and specific dynamic action (SDA). TEF originates from digestive and mechanical effects of food ingestion in the gastrointestinal tract. While the former summarizes energy expenditure due to physical (e.g., biting, chewing) and enzymatic processing of food, the latter refers to increased brown adipose tissue (BAT) thermogenesis induced by stimulation of neural (e.g., afferent vagal) and humoral factors (e.g., cholecystokinin release) in response to gastric stretch.[3] In contrast to TEF, SDA is mediated via non-vagal, non-cholecystokinin-related mechanisms and its magnitude depends from the macronutrient (protein, fat, carbohydrate) composition of the ingested food. SDA is mainly caused by postabsorptive biochemical and cellular processes and it is largest (~30%) in case of protein-rich meals.[2] The short-term thermal effects of food intake (TEF and SDA) can be summarized with the term "postprandial thermogenesis."[2]

Chronic (> 7 d) overfeeding causes long-term adaptive changes, counteracting the excess energy intake and maintains normal body mass. These adaptive changes involve skeletal muscle thermogenesis,[4] as well as BAT hypertrophy and hyperplasia,[1] both of which will lead to increased heat production, mainly via augmented mitochondrial functions. Neuroendocrinological changes (increased levels of catecholamines, thyroid hormones, steroids, etc.) also contribute to the elevation of the basal metabolic rate in response to sustained overfeeding.[1,2]

In summary, food intake induces postprandial thermogenesis over the short-term because of TEF and SDA. Sustained overfeeding leads to diet-induced thermogenesis, which is a long-term adaptive response to excess energy intake.

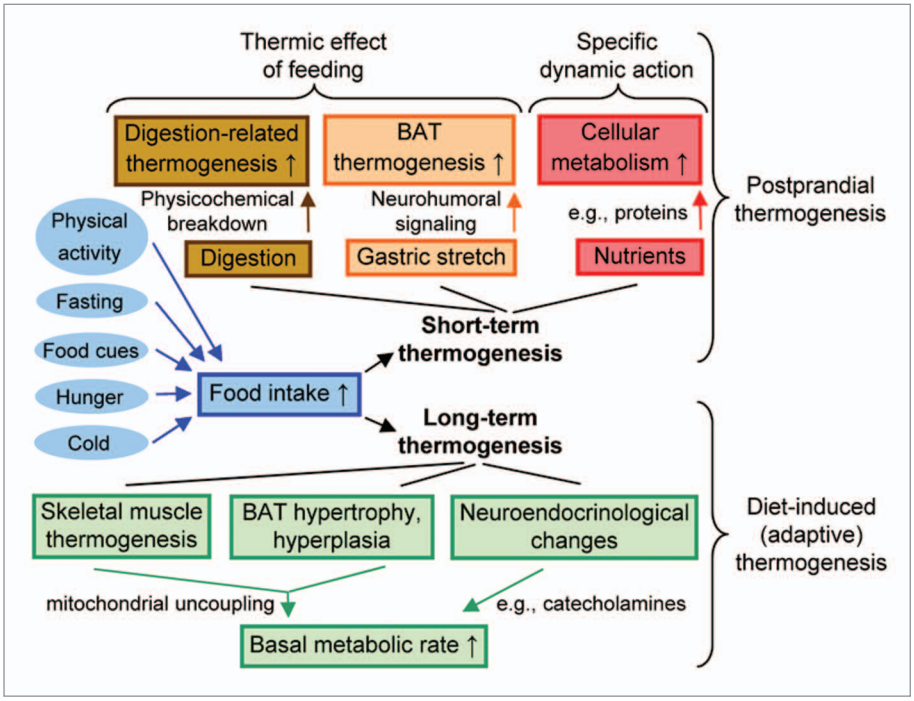

Figure 1. Short- and long-term effects of feeding on thermogenesis.

Keywords: diet-induced thermogenesis, specific dynamic action, thermic effect of feeding, postprandial thermogenesis

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Teaching Slide

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