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Review

Physiological and psychosocial age-related changes associated with reduced food intake in older persons

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ABSTRACT

Dietary intake changes during the course of aging. Normally an increase in food intake is observed around 55 years of age, which is followed by a reduction in food intake in individuals over 65 years of age. This reduction in dietary intake results in lowered levels of body fat and body weight, a phenomenon known as anorexia of aging. Anorexia of aging has a variety of consequences, including a decline in functional status, impaired muscle function, decreased bone mass, micronutrient deficiencies, reduced cognitive functions, increased hospital admission and even premature death. Several changes during lifetime have been implicated to play a role in the reduction in food intake and the development of anorexia of aging. These changes are both physiological, involving peripheral hormones, senses and central brain regulation and non-physiological, with differences in psychological and social factors. In the present review, we will focus on age-related changes in physiological and especially non-physiological factors, that play a role in the age-related changes in food intake and in the etiology of anorexia of aging. At the end we conclude with suggestions for future nutritional research to gain greater understanding of the development of anorexia of aging which could lead to earlier detection and better prevention.

1. Introduction

Aging in humans is associated with a failure to maintain energy homeostasis in response to changes in physiological and non-physiological factors, resulting in a decrease in body fat and body weight in older individuals (above 60 years of age) (Steen, 1988; Shimokata et al., 1989). The changes in the maintenance of energy homeostasis have been elucidated in several epidemiological studies. A cross-sectional follow-up study in the USA, for example, reported a decline in average daily energy intake of 1165 kcal in males and 405 kcal in females when comparing individuals of 20 and 75 years of age (Briefel et al., 1995). A similar study performed in Mexico, reported a decrease of 19.3 kcal/day/year in women and 25.1 kcal/day/year in men who were above 60 years of age (Koehler, 1994). Furthermore, an Australian study revealed that older persons lost height (1.8 cm) and weight (1.9 kg) during the 84 months of the study which was related to a significant decrease in carbohydrate, fat and protein consumption (Zhu et al., 2010). Besides changes in the amount of food and type of food intake, it is also shown that older persons eat fewer snacks between meals (de Castro, 1993), that they experience less cravings for food (Pelchat and Schaefer, 2000) and that they feel less hungry and more satiated than younger individuals (Clarkson et al., 1997). These age-related changes in food consumption lead to decreased energy intake in older persons which is associated with a reduction in body fat and body weight. These reductions can lead to a variety of health-related consequences, including a decline in functional status, impaired muscle function, decreased bone mass, micronutrient deficiencies, reduced cognitive functions, increased hospital admission and even premature death (Roberts, 2000; Ahmed and Haboubi, 2010). Overall, the reduced food intake and the decreased body fat and body weight in older persons, is referred to as anorexia of aging (Morley, 1997; Hays and Roberts, 2006; Kmiec, 2010).

Anorexia of aging is a nationwide issue with currently 16% of individuals older than 65 years who are affected (Guigoz et al., 2002; Ahmed and Haboubi, 2010). It is especially a severe problem among institutionalized older persons of which 15% of the community-dwelling and home-bound older persons, 23–62% of the hospitalized patients and up to 85% of the nursing home residents suffer from malnutrition (Guigoz et al., 2002; Ahmed and Haboubi, 2010). The development of anorexia of aging is multifactorial, involving both physiological and non-physiological aspects

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(Hays and Roberts, 2006). Preceding reviews on anorexia of aging focused primarily on the physiological changes involved in the development of this type of anorexia. Here, we will outline both the physiological (peripheral hormones, senses and central brain regulation) and non-physiological (psychological and social) changes to show that both type of factors play a significant role in the development of anorexia of aging.

2. Physiological changes

There are several physiological factors associated with reduced food intake in older persons. Some of these factors are known to be associated with feelings of hunger or satiety while the effects of others remain unknown. Most of the physiological factors involved in lower dietary intake in elderly are related to changes in peripheral hormones, senses and central brain control. Here, we will discuss the most important age-related changes in these three aspects affecting energy intake regulation and their role in the development of anorexia of aging.

2.1. Peripheral hormones

Several peripheral hormones are important in food consumption and are associated with altered energy intake in older persons, including cholecystokinin (CCK), leptin, ghrelin, insulin, peptide YY (PYY) and glucagon-like peptide-1 (GLP-1). These peripheral hormones are released in the gastrointestinal (GI) tract in response to food ingestion and they mainly influence food intake by affecting activity in key brain areas like the hypothalamus, where the blood–brain barrier is less tight due to a fenestrated capillary endothelium (Kastin and Pan, 2000; Bear et al., 2006).

2.1.1. Cholecystokinin

One of the hormones involved in short-term regulation of food intake is CCK. It is usually referred to as the satiety hormone because it induces feelings of satiety. CCK is released from inclusion (I) cells of the duodenum and jejunum in response to stimulation by certain nutrients. Furthermore, CCK is co-released with PYY from specialized gut endocrine cells (L cells) in the intestine (Sam et al., 2012). The main effect of CCK is activation of neurons in the nucleus of the solitary tract [a viscerosensory cell group in the brain stem (NTS)], possibly involving vagal afferents. This activation results in changes in eating behavior by reducing meal frequency and meal size (Bear et al., 2006). Other functions of CCK include slowing of gastric emptying (Morley, 1987), inhibiting ghrelin production (a hormone involved in increasing appetite) (Sam et al., 2012) and down regulating neuropeptide Y (NPY) gene expression (a neuropeptide stimulating food intake) (Simpson et al., 2009). When focusing on age-related changes in CCK, findings from different human studies have shown that older individuals have more CCK immuno-reactive cells in the duodenum compared to younger persons (Sandström and El-Salhy, 1999). Also, baseline plasma CCK-33 (one of the splice-variants of the CCK hormone) concentrations are higher in older than in younger adults and fasting levels of CCK have found to be increased fivefold in older individuals (MacIntosh et al., 1999). These higher levels were found to be associated with increased feelings of satiation and a subsequent reduction in food intake. Moreover, comparing underweighted older persons with healthy individuals, CCK-8 levels [a splice-variant involved in the relation between gut motility and digestive status (Buéno, 1993)] have found to be significantly higher in the underweighted subject group (Martinez et al., 1993), suggesting that increased CCK levels can reduce food intake with subsequent weight loss as a result. Contradictive results have also been observed. A study performed by Serra-Prat et al. (2009) showed that young participants had a higher increase in CCK after ingestion of a breakfast meal compared to the older participants. In addition, in the younger persons this increase remained for 3 h after which a decrease was observed, while the older persons showed a relative quick progressive decrease of CCK levels after food intake. These contradictive results might be explained by the fact that in the study reported by Serra-Prat et al. (2009) a radioimmunoassay was used that mainly assessed the active form of CCK, CCK-33-a, and no changes in gastric emptying and delivery of nutrients were monitored in the older participants. Since delayed gastric emptying is often observed in older persons (see below), CCK release can take more time in the older persons compared to younger individuals.

Overall, the higher plasma CCK concentrations in older persons observed by MacIntosh et al. (1999), the impaired CCK response after a meal, and especially the increased CCK concentration in underweighted older persons compared to healthy controls, support that changes in CCK concentrations are involved in changes in dietary intake in older persons. This effect can be mediated by CCK itself or CCK-induced inhibition of ghrelin production, which both increase feelings of satiety. However, it should be kept in mind that the abovementioned studies focused on different forms of CCK. It therefore remains difficult to outline the exact effects of increasing age on CCK levels and its precise involvement in the development of anorexia of aging. Also, abnormalities in gastrointestinal motility should be monitored more carefully in studies on CCK and anorexia of aging.

2.1.2. Leptin

Another hormone which is released by the body in response to food ingestion is leptin. This hormone is released by adipocytes and it regulates body mass by reducing appetite and increasing energy expenditure via inhibition of neuropeptide Y/agouti-related peptide (NPY/AgRP) neurons and stimulation of alpha-melanocyte-stimulating hormone/cocaine–amphetamine-regulated transcript (αMSH/CART) neurons in the hypothalamic arcuate nucleus (ARC) (Hays and Roberts, 2006). Several human studies have examined age-related effects on leptin and most of these studies show increased circulating leptin levels in older individuals (Ruhl et al., 2004; Zoico et al., 2004). Furthermore, a study on the effects of re-nutrition (increased nutrition in underweighted individuals) in older persons, found that only leptin showed significant changes; levels were increased after 6 weeks of successful re-nutrition. This makes this hormone a candidate biological marker for monitoring the efficacy of re-nutrition in malnourished older individuals (Nivet-Antoine et al., 2011). However, Roberts et al. (1997) found no effect of age on the relationship between circulating leptin and body fat mass. Therefore, they concluded that changes in leptin concentration are not linked to changes in body fat in older humans. These contradictive results suggest that, although leptin might be involved in malnutrition and re-nutrition in older persons, other mechanisms seem to be more important in the development of anorexia of aging.

2.1.3. Insulin

Another peripheral signal involved in food intake is insulin. This hormone is released into the bloodstream by β cells of the pancreas and it forms a necessary prerequisite for transport of glucose into body cells. Insulin regulates the levels of glucose in the blood via production of glucose transporters (Ferrannini et al., 1999) and, similar to leptin, insulin is able to inhibit NPY/AgRP neurons and to stimulate αMSH/CART neurons in the ARC, resulting in reduced food intake (Kmiec et al., 2005). Moreover, it can act as a satiety signal by decreasing ghrelin levels (Serra-Prat et al., 2009). As shown by Gutzwiller et al. (1999) aging in humans is characterized by elevated insulin levels in the blood and reduced glucose tolerance followed by increased blood glucose levels. These changes in glucose tolerance, increased insulin and glucose levels might lead to