VESTIBULAR BALANCE OF FOOD INTAKE.

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ABSTRACT

The intake of the nutrients is under complex control involving signals from both the periphery and central nervous system. The purpose of this article is to review research reports related to vestibular stimulation and its role in regulation of food intake and to suggest translational research in this area. Vestibular system is having extensive interactions with hypothalamus, dorsal raphe nucleus, nucleus tractus solitarius, locus coeruleus, hippocampal formation and regulates food intake. The present review provides evidence for the relationship between vestibular stimulation and food intake. Understanding these associations will be important in developing effective treatments for obesity and related metabolic diseases.

KEY WORDS: Vestibular stimulation, Food intake.

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INTRODUCTION

Vestibular system the sixth sense was discovered by a French anatomist, Pierre Flourens. It is the sense organ for balance and located near the inner ear, protected by the same hard bones of the skull that surround the cochlea. The sense of movement and gravity of the balance (vestibular) system in the ears develops very early and begins to function at five months gestation.\[1\] The need for vestibular stimulation can be observed throughout the life. \[2\] Vestibular system is having extensive interactions with thalamus, hypothalamus, periaqueductal grey, parabrachial nucleus, cerebellum, nucleus tractus solitarius and raphe nuclei [3] autonomic system, substantia nigra, hippocampal formation [4] locus coeruleus, [5] and relieves stress, treats parkinson’s disease, relieves cancer pain, promotes sleep, improves immunity, delays brain ageing, treats endocrine disorders and improves cognition. \[6, 7,8,9,10,11,12,13,14\] Food intake and energy expenditure are controlled by complex, redundant, and distributed neural systems involving thousands of genes and reflecting the fundamental biological importance of adequate nutrient supply and energy balance. Larger portions of the nervous system of animals and humans, including the cortex, basal ganglia, and the limbic system, are concerned with the procurement of food as a basic and evolutionarily conserved survival mechanism to defend the lower limits of adiposity. \[15\] Food intake is also regulated by autonomic, endocrine and psychosocial factors. \[16, 17\] Vestibular system regulates food intake with its extensive connections with the brain structures. The purpose of this article is to review research reports related to vestibular stimulation and its role in regulation of food intake and to establish a hypothesis that vestibular stimulation regulates food intake and to suggest translational research in this area.

MATERIALS AND METHODS

Articles were selected from searches at google.com, pubmed.com, British medical journal.com, Medline, ERIC, frontiersin.org, hindawi.com, scopus.com and other online standardized journals.

Vestibular stimulation regulates food intake through vagal stimulation

Vagal nerve stimulation (VNS) can increase vagal afferent signal conduct to CNS and mimics the satiety signals leading to reduce food intake and body weight gain. \[18, 19\] long-term VNS (102 days) with low-frequency electrical impulses (0.05 Hz) decreased food intake and body weight in rats. VNS increased serum levels of ghrelin and decreased serum levels of leptin. \[20\] Activation of the vagal nerves causes release of vagally controlled gastrointestinal hormones such as gastrin and cholecystokinin (CCK). \[4\] CCK is an important satiety signal for regulation the size of meals. \[23\] Direct vestibular nucleus projections to nucleus tractus solitarius (NTS) and dorsal motor nucleus of the vagus nerve (DMX) are observed. The activity of more than one-third of NTS neurons was modulated by vertical vestibular stimulation, with most of the responsive cells having their firing rate altered by rotations in the head-up or head-down directions. Single shock vestibular stimulation evokes response from the ipsilateral but not from the contralateral vagus nerve. \[3, 4\]

Vestibular stimulation regulates food intake through insulin

Insulin signaling in the hypothalamus regulates food intake and hepatic glucose production in rodents. Insulin activates Erk1/2 signaling in the DVC to regulate energy balance. \[21\] in rodents, intracerebroventricular (ICV) injection with neuro peptide Y (NPY) would stimulate food intake while ICV insulin would reduce food intake. \[22\] Pancreatic insulin secretion is regulated by vestibular stimulation through vagus nerve. Electrical vagal stimulation produced an increase in both insulin and glucagon secretion in proportion to the pulse frequency, but an inhibition in somatostatin release in rat. \[4\]
Vestibular stimulation regulates food intake through arcuate nucleus

The arcuate nucleus of hypothalamus plays a central role in both long-term control of energy balance and body weight and short-term control of food intake from meal to meal. It has two subsets of neurons that function in opposite manner. One subset releases neuropeptide Y, most potent appetite stimulator. The other releases melanocortins, which suppress appetite. The lateral hypothalamic area and paraventricular nucleus release orexins and corticotrophin-releasing hormone respectively in response to input from arcuate nucleus. Vestibular stimulation increases serotonin release from dorsal raphe nucleus. The nucleus raphe dorsalis and median raphe send projections to the arcuate nucleus. Serotonin (5-HT) has been suggested to induce satiety and 5-HT fibers contact NPY neurons in the arcuate nucleus (ARC) and PVN, suggesting that 5-HT could inhibit the ARC-PVN projection.

Vestibular stimulation regulates food intake through HPA axis

Dysregulation in the HPA axis has been associated with upper body obesity. Controlled vestibular stimulation modulates hypothalamic-pituitary-adrenal (HPA) axis. Controlled vestibular system inhibits stress axis directly, through GABA, hippocampal formation.

Vestibular stimulation regulates food intake by promoting sleep

Sleep deprivation has been linked to both increased risk for obesity and type 2 diabetes. Sleep deprivation is suggested to be a chronic stressor that may contribute to increased risk for obesity and metabolic disease, possibly in part through HPA axis dysregulation. The vestibular system is having extensive interactions with hypothalamus, dorsal raphe nucleus, nucleus tractus solitarius, locus coeruleus, hippocampal formation and promotes sleep. Vestibular stimulation promotes sleep.

Vestibular stimulation regulates food intake through thyroid hormones

Hypothyroidism caused a decrease in the daily food intake, body weight, and body temperature. The thyroid hormones are necessary for the long-term regulation of energy intake, storage, and expenditure by different mechanisms. The feeding behavior seems to be partially dependent on a direct action of the thyroid hormones on the brain and this effect is independent of the energy expenditure in the peripheral organs. The body weight is closely dependent on the thyroid status and its maintenance seems to involve thyroid action on mechanisms other than feeding and metabolic rate. Thyrotrophin releasing hormone (TRH) neurons are located in paraventricular nucleus. Electrical and caloric stimulation of vestibular pathways results in a response in PVN (para ventricular neurons) neurons in the guinea pig. Retrograde viral tracing in the rat brain has demonstrated the presence of a direct vestibuloparaventricular projection. The hypothalamic pituitary thyroid (HPT) axis plays a critical role in mediating changes in metabolism and thermogenesis. Thus, the central regulation of the thyroid axis by Thyrotropin Releasing Hormone (TRH) neurons in the paraventricular nucleus of the hypothalamus (PVN) is of key importance for the normal function of the axis under different physiological conditions including exposure to cold and changes in nutritional status.

CONCLUSION

The present review provides evidence for the relationship between vestibular stimulation and food intake. Understanding these associations will be important in developing effective treatments for obesity and related metabolic diseases.
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