Differential effects of common hepatic, gastric, and celiac branch vagotomy on food intake and pica behavior following chemotherapy in the rat

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Vomiting, nausea, and anorexia are among the main side effects of anti-cancer chemotherapies such as cisplatin. Cisplatin primarily acts on vagal afferents to produce emesis, but how this drug generates nausea and anorexia is poorly understood. Electrophysiology indicates that cisplatin activates vagal afferents of the common hepatic branch (CHB). Rats, a non-vomiting species, ingest kaolin clay (i.e., pica) when made sick by toxins. It has been hypothesized that pica behavior may model emesis in the rat. These studies examined the effects of CHB, ventral gastric (GAS), or celiac branch (CEL) vagotomies on pica and anorexia produced by cisplatin in the rat. Apomorphine, a centrally acting dopamine agonist which reliably produces emesis and/or pica, was also assessed. Results showed that cisplatin-induced pica was suppressed only by CHB vagotomy, and not by GAS and CEL vagotomy. Suppression of daily food intake and body weight following cisplatin treatment was also blunted by CHB ablation, but exacerbated by GAS or CEL vagotomy. No condition displayed altered apomorphine-induced pica. The current work is the first to show effects of vagal lesions on pica behavior in the rat. Our results clearly show that only in CHB vagotomized rats is there a diminution in ingestive behaviors indicative of cisplatin-induced sickness. This investigation may help to delineate the physiology of pica and define the neural circuitry of malaise, which may significantly impact cancer patients receiving potent pharmacotherapy.

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Inhibition of angiotensin converting enzyme protects against diet-induced obesity in rats

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Increasing evidence suggests that the renin angiotensin system (RAS) contributes to the etiology of obesity. Specifically, it has been reported that the RAS is overactive in obese humans and rodents, and it has been suggested that interfering with RAS activity might be a treatment option for obesity. To evaluate the role of the RAS in the development of diet-induced obesity we examined body weight, body composition, food intake and glucose tolerance in rats given an angiotensin converting enzyme inhibitor (Captopril; ~50 mg/(kg day)). Male Long-Evans rats were fed a high-fat diet and randomly assigned to two weight-matched groups. One group received Captopril in their drinking water at a concentration estimated to provide ~50 mg/(kg day). Captopril-treated rats gained significantly less weight than controls on the high-fat diet, and the difference was mainly attributable to differences in adipose mass. Furthermore, rats given Captopril ate significantly less high-fat diet and had improved glucose tolerance compared to controls. To determine if Captopril causes animals to defend a lower body weight, animals in both groups were fasted for 24 h and subsequently restricted to 20% of their daily intake for 2 days. When food was returned *ad libitum*, Captopril-treated and control rats returned to their respective body weights at a similar rate. Collectively, these results suggest that inhibition of the renin angiotensin system is protective against the development of diet-induced obesity.

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Effects of monotony and variety foods on feeding behavior in humans

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Variety in foods characteristics is an important determinant for its consumption. Diverse studies have indicated that the subjects tend to consume more foods when there is variety of diets. The objective of this experiment was to evaluate effects of variety or monotony of food on feeding behavior. Sixteen participants were assigned randomly to four groups. The experiment consisted of two phases. First group was exposed to monotony condition in both phases. Second group was exposed variety condition in both phases. Third group was exposed in first condition to monotony followed of variety and the fourth group was exposed to variety condition followed of monotony. Results showed that: (1) first group consumed a smaller amount of foods in both phases in comparison with second group; (2) food consumption of second group was greater during variety condition; (3) third group increased its food consumption during the second phase; and, (4) group 4, presented greater food consumption during second condition. The results suggest that variety of foods increases the answer of consumption, whereas it diminishes in monotony condition.

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The lateral parabrachial nucleus controls hypertonic, not isotonic, NaCl intake

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Preference for NaCl solutions reaches its peak at isotonic concentrations in rats. The inverted "U" shape of the preference curve is similar for hydrated and sodium-depleted rats suggesting that mechanisms that control sodium intake act changing the amplitude, not the shape of the curve. However, it is possible that some inhibitory mechanisms, like those from the lateral parabrachial nucleus (LPBN), act preferentially on the hypertonic side of the curve. The present work tested this hypothesis by either pharmacological intervention or immunohistochemistry for immediate early gene (c-fos) expression in the LPBN using two different protocols, systemic furosemide combined with low dose of captopril (FURO/CAP) and sodium depletion induced by peritoneal dialysis (PD). Methysergide (serotonin antagonist) injected into the LPBN ($n = 7$ per group) had no effect on 0.15 M NaCl intake (methysergide: 19 ± 5.2; vehicle: 19.3 ± 4.2, ml/2 h), but it enhanced 0.3 M NaCl intake (methysergide: 16.6 ± 3.5; vehicle: 6.6 ± 1.5, ml/2 h) induced by FURO/CAP without changing FURO/CAP-induced thirst. The number of positive cells for c-fos protein in the LPBN of rats made hypovolemic by PD enhanced when they ingested hypertonic (42 ± 10 vs. hydrated: 18 ± 1), but not isotonic (27 ± 10 vs. hydrated: 20 ± 8) NaCl. The results suggest that the LPBN has a preferential control over hypertonic rather than isotonic NaCl intake.

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