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Type A CCK Receptors Mediate Satiety Effects of Intestinal Nutrients

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BRENNER, L. A. AND R. C. RITTER. *Type A CCK receptors mediate satiety effects of intestinal nutrients.* PHARMACOL BIOCHEM BEHAV **54**(3) 625-631, 1996.—Previous work indicates that endogenous CCK mediates suppression of sham feeding by some intraintestinal nutrients. To test whether the mechanism involved is dependent upon action at type A or type B CCK receptors, we examined the ability of CCK_A (devazepide) and CCK_B (L-365,260) receptor antagonists to attenuate the suppression of sham feeding by intraintestinal oleic acid, maltotriose, or L-phenylalanine. Suppression by oleic acid or maltotriose was dose dependently attenuated by intraperitoneal administration of the CCK_A receptor antagonist, as was suppression by exogenous CCK. The CCK_B receptor antagonist failed to attenuate the suppression of sham feeding by these nutrients. Neither receptor antagonist attenuated the suppression of sham feeding induced by intraintestinal L-phenylalanine. These results suggest that suppression of sham feeding by intestinally infused oleic acid and maltotriose is mediated by endogenous CCK acting at CCK_A receptors.

CCK Cholecystokinin Receptors Food intake Gastrointestinal Satiety Devazepide L-365,260

DAMAGE to vagal sensory fibers by systemic or fourth ventricular application of the neurotoxin capsaicin (22,26,30), or surgical section of vagal afferent roots (25,27), attenuates suppression of sham feeding by both exogenous CCK and some intestinal nutrients. These results indicate that exogenous cholecystokinin (CCK) and some intestinal nutrients suppress sham feeding via similar, if not identical, neural substrates.

The fact that capsaicin-sensitive vagal sensory fibers are necessary for suppression of food intake by both exogenous CCK and intestinal nutrients suggests that endogenous CCK participates in control of food intake by some intestinal nutrients. In support of this hypothesis we found that selected doses of two chemically different CCK receptor antagonists attenuated suppression of sham feeding by intestinal infusion of either oleic acid or maltose, but not by L-phenylalanine (29,31). These results suggested that intestinal nutrients suppress sham feeding via an action of endogenous CCK acting at CCK type A receptors. However, data has also been presented suggesting that endogenous CCK may control food intake via an action at CCK type B receptors (7).

Therefore, in this study we compared the potential roles of CCK_A and CCK_B receptor subtypes in the suppression of sham feeding by intestinal nutrient infusion. Our results indicate that CCK_B receptors do not directly participate in suppression of sham feeding by intestinal nutrients. In addition, our current results confirm our previous conclusion that suppression of sham feeding by some nutrients depends upon CCK_A receptors. Finally, the current results demonstrate that there are some stimuli (L-phenylalanine) that suppress sham feeding via mechanisms that require neither CCK_A nor CCK_B receptor participation.

METHOD

Subjects for all experiments were adult male Sprague-Dawley rats (400-500 g, Washington State University, Lab Animal Resources) housed individually in a temperature-controlled room on a 12 L : 12 D schedule. Animals used in sham feeding experiments were anesthetized with methoxyflurane (Metofane, Pitman-Moore) and surgically implanted with chronic stainless steel gastric cannulas and duodenal catheters according to a previously detailed description (30). This preparation allows infusion of nutrient to the intestine via a remote infusion pump, while simultaneously draining ingested solution from the animal's stomach.

At the time of sham feeding experiments, gastric cannula

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screws were removed and the gastric contents gently lavaged from the stomach with warm water. A drainage tube was attached to the open cannula and the rats were placed in Plexiglas feeding cages with the drainage tube exiting through a longitudinal slot in the wire mesh cage floor. Animals were adapted to ingesting a 15% sucrose solution from graduated drinking burettes. Ingestion of sucrose was measured to the nearest 0.1 ml over 30 min.

Behavioral testing commenced only after stable 30 min baseline intakes were observed. This usually occurred after approximately six training trials within 2 weeks. All feeding experiments were performed at 0900 h every 48 h on rats that were adapted to a feeding regimen wherein pelleted laboratory rodent diet was removed at 1700 h the day prior to testing and returned at 1200 h immediately following testing. Thus, all experiments were conducted on animals deprived of food for 16 h.

In these experiments we tested the effects of the CCK_A receptor antagonist, devazepide (MK-329), and the CCK_B receptor antagonist, L-365,260 (gift of Merck Sharpe & Dohme, West Point, PA). Each compound was prepared in a 1 : 100 dimethyl sulfoxide (DMSO): saline vehicle immediately prior to testing by mixing first with DMSO and then with isotonic saline. This resulted in a suspension that was stirred constantly until being drawn into syringes for injection. CCK antagonists were tested for their ability to influence suppression of sucrose intake produced by three nutrients—maltotriose, oleate, and L-phenylalanine (L-Phe) (Sigma Chemical Co., St. Louis, MO). All nutrients were delivered at a concentration of 0.08 or 0.13 kcal/ml, at a pH of 7.2–7.4. Because L-phe at 0.13 kcal/ml produced slightly more suppression of intake than either oleate or maltotriose, an additional, more concentrated maltotriose infusion that produced a suppression similar to that resulting from L-phe also was included in our experimental design. Tonicity was checked using a vapor pressure osmometer (Wescor 5130A), and adjusted to 300 mosM. A saline solution having the same osmolality and pH as the nutrient infusions was used as a control infusate. Green food coloring (Crescent Mfg. Co., Seattle, WA) was added to the infusates so that we could evaluate the success of intraintestinal infusion. The presence of a green color in the liquid that drained from the stomach indicated that infusate had refluxed from the intestine to the stomach and that intestinal infusion was incomplete. Data from tests where the infusion was incomplete were discarded.

For intestinal infusion, the free end of the intestinal catheter was exteriorized and 0.2 ml saline was flushed through each catheter to assure patency. Infusates were drawn into 10 ml syringes connected to PE 50 tubing that passed through the drainage tubes and was joined to the intestinal catheters. An intraperitoneal (IP) injection of vehicle or antagonist was given 10 min prior to presentation of sucrose. Using syringe pumps (Sage Instruments Model 351), intestinal infusions were made beginning 5 min before and ending 5 min after presentation of sucrose. Sham intake of 15% sucrose was recorded at 5-min intervals for 30 min.

The effect of the CCK receptor antagonists on suppression of sham feeding by exogenous CCK-8 was also examined. The antagonist or vehicle was injected IP 10 min prior to sucrose presentation and CCK-8 (2 μg/kg, gift of E. R. Squibb and Sons) or vehicle injected IP 5 min prior to sucrose presentation. Sham intake of sucrose was recorded at 5-min intervals for 30 min.

A separate group of rats was used for each series of experiments involving a particular nutrient infusion—oleate, maltotriose, or L-phenylalanine. For each dose of antagonist tested the experimental subjects received the following injection: infusion combinations; vehicle : saline; antagonist : saline; vehicle : nutrient; antagonist : nutrient; and vehicle : saline. When the antagonists were tested in conjunction with exogenous CCK the same experimental design was implemented with the animals receiving intraperitoneal CCK-8 or vehicle rather than intraintestinal nutrient infusions. One-way repeated measures analysis of variance (ANOVA) incorporating a general linear model approach to handle missing values (SigmaStat) was performed on raw intake data for a particular nutrient series with antagonist dose as the repeated measure. ANOVAs were followed by Bonferroni tests for planned comparisons. p-Value for significance was set at 0.01 in all analyses.

The data are presented graphically as percentage suppression of 30-min sucrose intake. This was calculated for each rat according to the following formula: % suppression = [1 − (experimental/baseline)] × 100.

RESULTS

In no case did duodenal infusion of saline or intraperitoneal injection of vehicle solutions significantly affect sham feeding of sucrose when compared to no injection or no infusion (data not shown). Intraintestinal infusion of either oleate (0.08 kcal/ml), maltotriose (0.13 kcal/ml or 0.345 kcal/ml), or L-phenylalanine (0.13 kcal/ml) significantly decreased sham intake of sucrose ($p < 0.001$) (Table 1), as did intraperitoneal injection of CCK-8 (2 μg/kg, $p < 0.001$) (Table 3).

Intraperitoneal administration of the CCK_A receptor antagonist, devazepide, dose dependently attenuated the suppression of sham sucrose intake induced by intraintestinal oleate infusion, $F(6, 32) = 4.423$, $p = 0.002$ (Table 1, Fig. 1). Bonferroni tests for planned comparisons indicated that a dose of 300 μg/kg devazepide and higher prior to intraintestinal oleate significantly increased sham sucrose intake above the oleate alone condition (0 μg/kg devazepide), and that doses of 75 μg/kg and higher prior to oleate infusion produced sham intakes that were not significantly different from intakes following intraintestinal saline infusion alone (Table 1).

In a similar manner, devazepide dose dependently attenuated the suppression of sham sucrose intake induced by intraintestinal maltotriose infusion (0.13 kcal/ml), $F(6, 38) = 9.202$, $p < 0.001$ (Table 1). Bonferroni tests for planned comparisons indicated that doses of 25 μg/kg devazepide and higher prior to intraintestinal maltotriose significantly increased sham sucrose intake above the maltotriose alone condition (0 μg/kg devazepide), and that doses of 75 μg/kg and higher prior to maltotriose infusion produced sham intakes no different from intakes following intraintestinal saline infusion alone (Table 1). In neither the oleate nor the maltotriose group did any dose of devazepide over the range 1–600 μg/kg significantly affect sham sucrose intake when administered alone (data not shown, $p = 0.59$ and 0.06, respectively).

Administration of devazepide over the dose range of 75–600 μg/kg failed to attenuate the suppression of sham sucrose intake induced by intraintestinal infusion of L-phenylalanine, $F(3, 18) = 0.815$, $p = 0.502$ (Table 1, Fig. 2). Similar to the results found in both the maltotriose and oleate groups, no dose of devazepide tested had any effect on sham sucrose intake when administered in the absence of nutrient infusion (data not shown, $p = 0.246$).

Devazepide was also administered to a group of subjects receiving intraintestinal infusion of 0.345 kcal/ml maltotriose

TABLE 1

SHAM INTAKE OF SUCROSE FOLLOWING INTRAPERITONEAL ADMINISTRATION OF THE CCK_A RECEPTOR ANTAGONIST, DEVAZEPIDE (MK-329), PRIOR TO INTRAINTESTINAL NUTRIENT INFUSION

	Sucrose			
Devazepide (μg/kg)	Oleate (0.08 kcal/ml)	Maltotriose (0.13 kcal/ml)	Maltotriose (0.345 kcal/ml)	L-Phenylalanine (0.13 kcal/ml)
0	27.70 ± 3.15 (12)	34.64 ± 3.80 (10)	18.28 ± 3.35 (8)	19.62 ± 4.36 (8)
1	29.88 ± 11.22 (6)	34.48 ± 6.31 (9)	12.68 ± 3.67 (6)	
25	33.31 ± 7.23 (8)	40.78 ± 5.88* (9)	17.63 ± 4.84 (6)	
75	33.44 ± 8.33 (7)	47.55 ± 4.91* (8)	25.22 ± 7.24 (6)	19.30 ± 5.9 (6)
150	40.00 ± 4.21 (10)	39.96 ± 6.12* (7)	39.15 ± 6.02* (6)	
300	42.08 ± 4.91* (4)	43.43 ± 5.42* (5)	38.13 ± 6.31*(6)	25.36 ± 8.18 (8)
600	37.90 ± 4.75* (3)	46.67 ± 7.34* (6)	34.82 ± 5.42* (5)	16.49 ± 4.37 (7)
saline : saline	44.09 ± 3.72 (12)	50.41 ± 3.4 (10)	49.94 ± 3.28 (8)	40.01 ± 3.4 (8)

Devazepide was administered IP 5 min prior to intraintestinal nutrient infusion (10 ml/10 min). Values are mean 30 min sham intakes in milliliters ± SEM beginning 5 min after the start of intraintestinal infusion.

*$p < 0.01$, significantly different from intraintestinal nutrient infusion alone.

Saline : saline represents the baseline condition − saline injection coupled with saline infusion. Numbers in (parentheses) indicate n for each experiment.

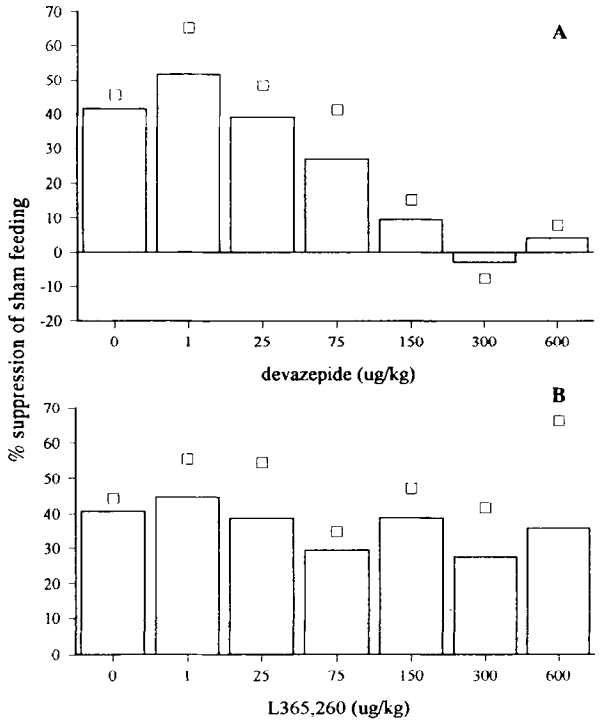

FIG. 1. (A) The CCK_A antagonist, devazepide, dose dependently attenuates suppression of sham feeding induced by intraintestinal oleate (0.08 kcal/ml). IP injection of 300 μg/kg or 600 μg/kg of devazepide 5 min prior to intraintestinal oleate significantly attenuated oleate-induced suppression of sham sucrose intake ($p < 0.001$). A dose of 75 μg/kg or higher prior to oleate infusion produced sham intakes that were not significantly different from intakes following intraintestinal saline infusion alone. (B) The CCK_B antagonist, L-365,260, had no effect on oleate-induced suppression of sham feeding at any of the doses tested ($p = 0.843$). (□) indicate standard error of the mean (SEM).

since this concentration gave suppressions similar to those seen with L-phenylalanine infusion. Devazepide dose dependently attenuated the suppression of sham intake induced by this higher concentration of maltotriose, $F(6, 29) = 12.801$, $p < 0.001$ (Fig. 3). Bonferroni tests for planned comparisons indicated that at this maltotriose concentration, a dose of 150 μg/kg devazepide or higher significantly increased sham sucrose intake above the maltotriose alone condition (0 μg/kg devazepide), and that this same dose or higher produced sham intakes no different from intakes following intraintestinal saline infusion alone (Table 1).

Administration of the CCK_B receptor antagonist, L-365,260, over the dose range of 1–600 μg/kg was ineffective for attenuation of the suppression of sham sucrose intake induced by intraintestinal oleate, $F(6, 20) = 0.441$, $p = 0.843$ (Fig. 1), maltotriose, $F(5, 28) = 1.89$, $p = 0.13$ (Fig. 3), or L-phenylalanine (300 μg/kg, $t(6) = 1.014$, $p = 0.35$) (Table 2).

The suppression of sham sucrose intake resulting from intraperitoneal injection of a 2 μg/kg dose of sulfated CCK-8 was dose dependently attenuated by administration of the CCK_A receptor antagonist devazepide, $F(6, 33) = 10.757$, $p < 0.001$ (Fig. 4). There was a significant increase in sham intake above the CCK-8 alone condition (0 μg/kg devazepide) at a dose of 1 μg/kg devazepide or higher, and at doses of 10 μg/kg and higher, sucrose intake was no different from intake following vehicle : saline injection (Table 3).

DISCUSSION

The complete dose-response studies presented here confirm our previous conclusion that suppression of sham feeding by some nutrients depends upon CCK_A receptors. Our results also demonstrate that CCK_B receptors do not directly participate in suppression of sham feeding by intestinal nutrients. Furthermore, the lack of efficacy of either the CCK_A or CCK_B receptor antagonists for attenuation of the suppressive effects of intraintestinal L-phenylalanine indicates that suppression of sham feeding by this nutrient occurs via a mechanism(s) that requires neither CCK_A nor CCK_B receptor participation.

Because the suppression of intake produced by L-phenylalanine was more pronounced than that resulting from either 0.08 kcal/ml oleate or 0.13 kcal/ml maltotriose infusion, the

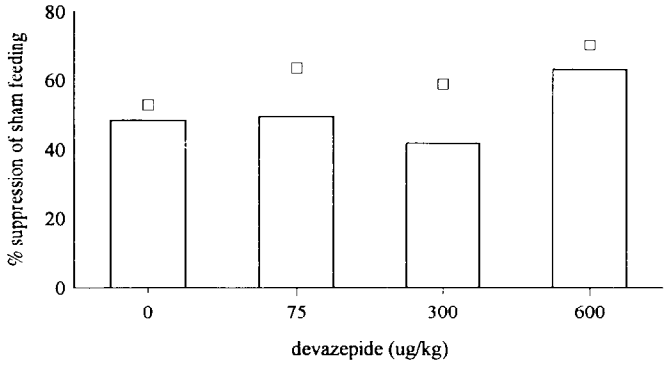

FIG. 2. The CCK_A antagonist, devazepide, fails to attenuate suppression of sham feeding by intraintestinal L-phenylalanine (0.13 kcal/ml). L-Phenylalanine significantly suppressed sham feeding, and IP injection of 75–600 μg/kg devazepide had no significant effect on this suppression ($p = 0.50$). (□) indicate standard error of the mean (SEM).

ineffectiveness of the CCK_A receptor antagonist for attenuation of the suppressive effects of L-phenylalanine could be interpreted as a problem of insufficient devazepide dosage to overcome this higher level of suppression. However, the dose-dependent attenuation of a similar level of suppression of sham feeding produced by intraintestinal infusion of a high concentration of maltotriose—a nutrient, which, like L-phenylalanine, does not elevate plasma CCK in the rat (4)—supports our earlier conclusion that the mechanism responsible for L-phenylalanine-induced suppression of sham feeding is independent of endogenous CCK (29).

Our results concerning the ability of devazepide to dose dependently attenuate the suppression of sham feeding by exogenously administered CCK are similar to those reported by other investigators for real feeding animals (7,13,21). Unlike these reports, however, administration of devazepide either alone or in concert with infused nutrient or exogenous CCK resulted in no significant increase in sham sucrose intake above baseline levels. This disparity can be attributed to a difference in experimental paradigm—in our experiments, rats were 16-h deprived and sham feeding, a situation in which feeding is maximal and which eliminates changes in gastric distension and emptying, as well as the entry of ingested material into the intestine. Similar results were found in the sham feeding cat (1).

It is possible that devazepide increases food intake by a mechanism that is completely independent of the one activated by intestinal oleate infusion (28). Our sham-feeding paradigm may preclude detection of increased intake by devazepide administered to rats in the absence of a nutrient infusion. However, if the reduction of food intake by nutrient infusion and its increase by devazepide were due to a devazepide-induced increase in feeding and not attenuation of nutrient-induced suppression of intake, the antagonist should increase food intake regardless of the intestinal nutrient used to cause reduction. The fact that devazepide reverses suppression of sham feeding by intestinal oleate and maltotriose, but not by L-phenylalanine, indicates that devazepide does not merely act independently of intestinal stimulus to increase food intake. Although we do not wish to rule out additional mechanisms by which devazepide may alter food intake, we view our results as providing evidence for participation of endogenous

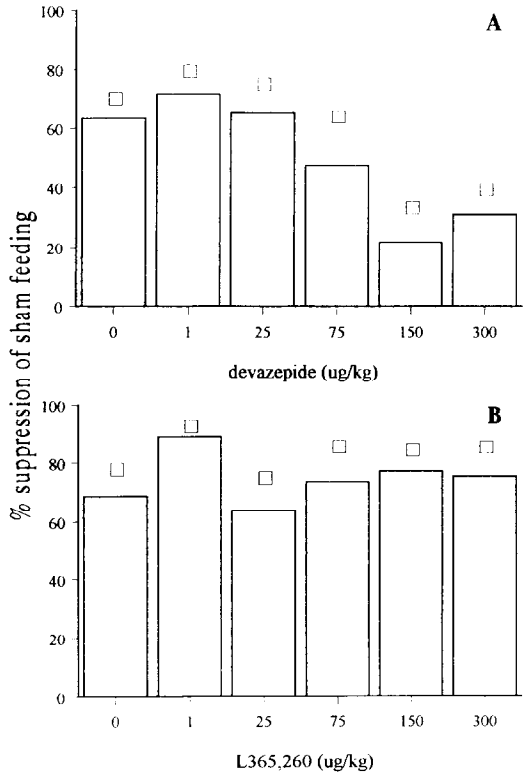

FIG. 3. (A) The CCK_A antagonist, devazepide, dose dependently attenuates suppression of sham feeding induced by intraintestinal maltotriose (0.345 kcal/ml). IP injection of 150 μg/kg or higher significantly attenuated maltotriose-induced suppression of sham sucrose intake ($p < 0.001$). A dose of 150 μg/kg or higher prior to maltotriose infusion produced sham intakes that were not significantly different from intake following intraintestinal saline infusion alone. (B) The CCK_B antagonist, L-365,260, had no effect on maltotriose-induced suppression of sham feeding at any of the doses tested ($p = 0.13$). (□) indicate standard error of the mean (SEM).

TABLE 2

SHAM INTAKE OF SUCROSE FOLLOWING INTRAPERITONEAL ADMINISTRATION OF THE CCK_B RECEPTOR ANTAGONIST, L-365,260, PRIOR TO INTRAINTESTINAL NUTRIENT INFUSION

	Sucrose Intake (ml)		
L-365,260 (μg/kg)	Oleate (0.08 kcal/ml)	Maltotriose (0.345 kcal/ml)	L-Phenylalanine (0.13 kcal/ml)
0	28.27 ± 3.17 (13)	15.08 ± 5.43 (8)	20.74 ± 5.32 (7)
1	30.93 ± 9.29 (6)	5.13 ± 1.97 (6)	
25	30.86 ± 12.97 (5)	17.43 ± 6.11 (8)	
75	34.93 ± 2.53 (3)	13.83 ± 7.16 (6)	
150	24.02 ± 4.11 (5)	10.21 ± 4.48 (7)	
300	28.55 ± 6.74 (4)	11.55 ± 5.85 (6)	16.21 ± 4.50 (7)
600	24.40 ± 10.61 (3)		
saline : saline	47.87 ± 3.98 (11)	42.32 ± 4.07 (8)	35.50 ± 2.99 (7)

L-365,260 was administered IP 5 min prior to intraintestinal nutrient infusion (10 ml/10 min). Values are mean 30 min sham intakes in milliliters ± SEM beginning 5 min after the start of intraintestinal infusion. No dose of antagonist tested produced significant attenuation of suppression induced by any of the intraintestinal nutrient infusions. Saline : saline represents the baseline condition – saline injection coupled with saline infusion. Numbers in (parentheses) indicate *n* for each experiment.

CCK in suppression of sham feeding by specific nutrients—oleate and maltotriose.

Suppression of sham feeding by exogenous CCK was significantly attenuated by devazepide at a dose of 1 μg/kg, while significant attenuation of suppression resulting from nutrient infusions required doses from 25 to 150 times higher. One interpretation of this observation suggests that intestinal infusion of oleate or maltotriose results in a higher level of endogenous CCK at one or more receptor sites than is achieved by intraperitoneal injection of our 2 μg/kg dose of exogenous CCK. CCK occurs in neurons of the central and peripheral nervous systems (2,9,10,20), as well as in intestinal endocrine cells. We previously compared the efficacy of intestinal nutrients for suppressing food intake with their efficacy for elevating plasma CCK concentrations and found no consistent relationship between a nutrient's ability to elevate plasma CCK and its ability to suppress sham feeding (4). Consequently, we conclude that the mechanism of CCK's participation in nutrient-induced suppression of sham feeding does not depend upon stimulus-induced release of CCK into the systemic circulation, but rather, endogenous CCK's action is neuronal or paracrine in nature.

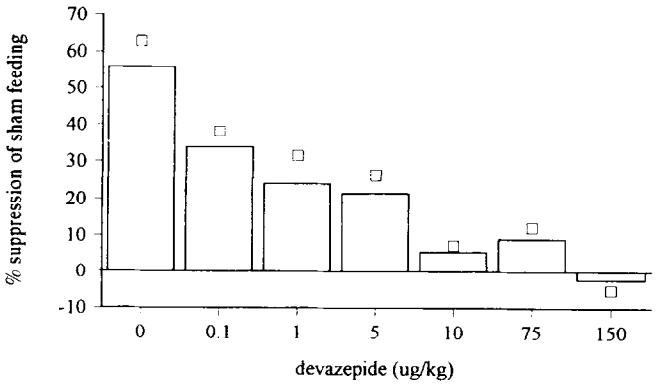

FIG. 4. The CCK_A antagonist, devazepide, dose dependently attenuates suppression of sham feeding induced by intraperitoneal CCK-8 (2 μg/kg). IP injection of 1 μg/kg devazepide and higher 5 min prior to intraperitoneal CCK-8 significantly attenuated CCK-induced suppression of sham sucrose intake ($p < 0.001$). Doses of 10 μg/kg or higher prior to CCK-8 injection produced sham intakes that were not significantly different from intakes following vehicle : saline injection alone. (□) indicate standard error of the mean (SEM).

TABLE 3

SHAM INTAKE OF SUCROSE FOLLOWING INTRAPERITONEAL INJECTION OF THE CCK_A RECEPTOR ANTAGONIST, DEVAZEPIDE, PRIOR TO INTRAPERITONEAL CCK-8

Devazepide (μg/kg)	Sucrose Intake (ml)
	CCK-8
0	23.87 ± 4.54 (11)
0.1	34.00 ± 7.30 (6)
1	40.36 ± 7.20* (8)
5	43.25 ± 5.04* (6)
10	52.43 ± 4.45* (6)
75	46.70 ± 3.59* (6)
150	56.14 ± 3.33* (7)
saline : saline	50.62 ± 2.92 (11)

Devazepide was administered 5 min prior to IP CCK-8 (2 μg/kg). Values are mean 30 min sham intakes in milliliters ± SEM beginning 5 min after IP CCK-8 injection.

*$p < 0.01$, significantly different from IP CCK-8 injection alone.

Saline : saline represents the baseline condition – saline injection coupled with saline infusion. Numbers in (parentheses) indicate *n* for each experiment.

Our experiments do not distinguish between a peripheral or central site for the receptor population(s) whose blockade results in attenuation of nutrient-induced ingestive effects. Experiments in which the CCK_B receptor antagonist, L-365,260, exhibited more potent stimulation of food intake in satiated rats than did the CCK_A receptor antagonist, devazepide (7),

suggested a central site of mediation for the role of endogenous CCK in food intake because type B CCK receptors predominate in the brain (14). Our inability to demonstrate any efficacy of the CCK_B receptor antagonist for attenuation of nutrient-induced suppression of food intake would argue against such a conclusion, especially because the CCK_B receptor antagonist employed has been shown to readily penetrate the brain (16). However, CCK_A receptors also exist in the rat brain. CCK_A receptor density is especially dense in the area postrema, medial subnucleus of the nucleus of the solitary tract, and the interpeduncular nucleus (15). Evidence that CCK is released from some brain areas during feeding and gastrointestinal stimulation (6,11,24), and reports of alterations in food intake produced by intracerebral applications of CCK agonists (8) and antisera (5) suggest that endogenous CCK could be exerting its effects at central neuronal CCK_A receptors. Devazepide does penetrate the blood-brain barrier (17). However, no information is available comparing the relative accessibility of the antagonist to central verses peripheral sites of action. Nevertheless, it is possible that higher devazepide doses are required to abolish suppression of sham feeding by intestinal nutrients than are required to abolish suppression of sham feeding by exogenous CCK because CCK_A receptors involved in suppression of sham feeding by intestinal nutrients are less accessible to peripherally administered CCK antagonists such as devazepide.

There is a strong case for specific vagal mechanisms sensitive to exogenous CCK and potentially capable of mediating behavioral effects of endogenous CCK. Electrophysiological evidence indicates that vagal fibers respond to exogenously administered CCK and that these CCK responsive fibers belong to a subpopulation of neurons that is damaged or destroyed by the neurotoxin capsaicin (18). The CCK-responsive fibers appear to be at least partially distinct from gastric mechanoreceptive fibers (19,23) and to activate intestinal mucosal neurons that may be chemoreceptive (3). As mentioned previously, both surgical and chemical destruction of vagal sensory fibers attenuates suppression of feeding by exogenous CCK and by oleate and maltose. These results, taken together with the fact that vagal fibers express CCK_A receptors (12), are consistent with participation of peripheral CCK in control of food intake by intestinal oleate, maltose, and maltotriose. However, the nature of peripheral CCK's participation remains uncertain.

In summary, administration of the type A CCK receptor antagonist, devazepide, dose dependently attenuates the suppression of sham feeding by intraintestinal infusion of oleate or maltotriose, but not L-phenylalanine. In contrast, administration of the B receptor antagonist has no effect on the suppression of sham feeding induced by intraintestinal infusion of any of these nutrients. These results suggest that the mediation of the satiety effects of oleate and maltotriose is dependent upon the interaction of endogenous CCK with type A CCK receptors.

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