High-fat diet switches the expression of two neurotransmitters released by vagal afferent neurons in the NTS resulting in increased food intake in rats
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Hypothesis:
1) CART (Cocaine and Amphetamin Related Transcript) is released by the vagus nerve into the NTS (Nucleus Tractus Solitarius) in response to feeding and inhibits food intake
2) MCH (Melanin Concentrating Hormone) is released into the NTS by the vagus nerve in response to fasting and stimulates food intake
3) MCH is expressed even after a meal in the NTS of high-fat diet fed rats
4) CART and MCH are synthesized in the cellular body of the vagal neurons, in the nodose ganglia

Proposed research accomplished
We injected different concentrations of CART antibodies and MCH receptor 1 antagonist into the brainstem of 21 high-fat diet fed and 8 low-fat diet fed Sprague Dawley rats. We performed the injections through a cannula previously placed in the NTS of the rats during a surgery. The food intake of each was monitored 2 hours after the injection.
After euthanasia, we sectioned their brainstem in 100 micrometers thick slices to control the cannula placement.
We performed a fasting-refeeding experiment on 10 low-fat diet fed rats. They were sacrificed at different time-frames after the refeeding, and their nodose ganglia was sampled to perform a Western Blot.

Results
The injection of CART antibodies increased food intake in low-fat diet fed rats, but not in high-fat diet rats. MCH receptor 1 antagonist decreased food intake in high-fat diet fed rats but not in low-fat diet rats. Thus we proved
- endogenous CART decreases food intake
- endogenous MCH increases food intake
- CART and MCH act at the level of the NTS
We have also shown that expression of CART is high whereas expression of MCH is low in the NTS of low-fat diet fed rats after a meal. On the contrary, there is no CART and a high level of MCH in the NTS of high-fat diet fed rats after feeding.
The Western Blot we performed didn’t allow us to formulate any conclusion about the amount of CART nor MCH at the level of the nodose ganglia after the fasting-refeeding experiment.