# Palatable food regulates the activity of magnocellular oxytocin neurons in the supraoptic nucleus of the hypothalamus

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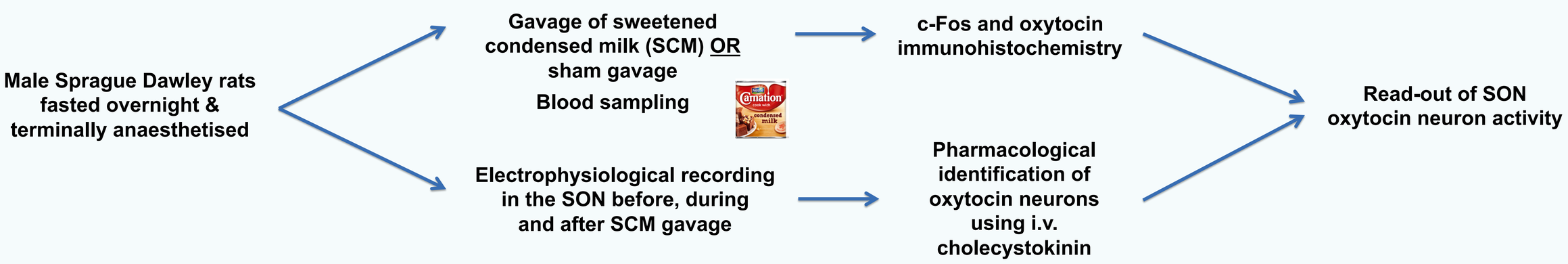
## Introduction & Hypothesis

**Alongside oxytocin's classical roles in maternal behaviour, this peptide is now considered to be key in the control of food intake. In rats, there is evidence that SON oxytocin neurons are activated by the consumption of bland food when hungry[1]. After food consumption, the peripheral hormone cholecystokinin signals satiety to anorexigenic neurons of the hypothalamic arcuate nucleus. These neurones release α-melanocyte stimulating hormone which in turn regulates oxytocin neurons in the supraoptic nucleus (SON) of the hypothalamus[2].**

***AIMS:*** **Use oxytocin- and c-Fos-like immunoreactivity alongside *in vivo* electrophysiology to study SON oxytocin neuron activity in response to *in vivo* gavage of a palatable food.**

***HYPOTHESIS*****: SON oxytocin neurons are activated in response to the direct delivery of palatable food into the stomach.**

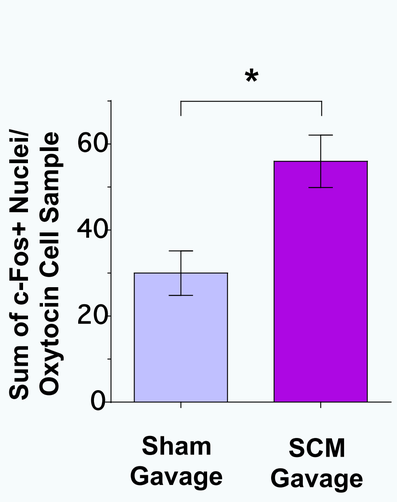
## Methods



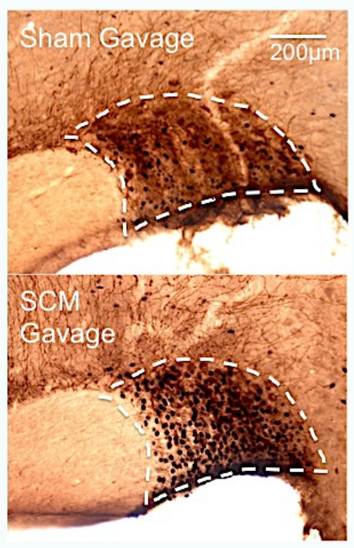

## Results

### (1) Increased c-Fos expression in SON oxytocin neurons after SCM gavage

**c-Fos expression in oxytocin neurons of the SON**



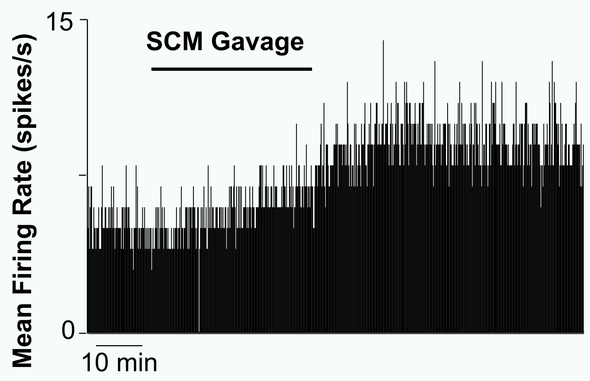

p = 0.0022
Mann Whitney test
(n = 6 per group)



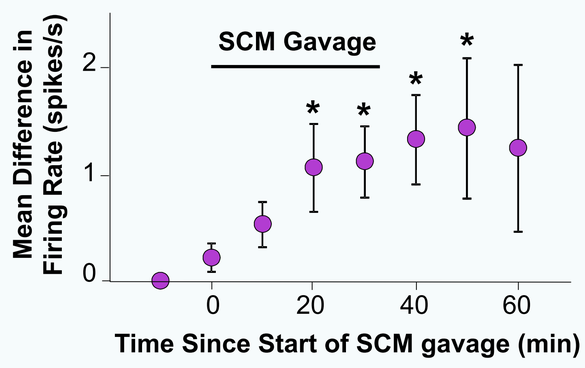

**c-Fos-Oxytocin-like immunohistochemistry**
**Black – c-Fos**
**Brown – oxytocin**

### (2) Increase in firing of SON oxytocin neurons with SCM gavage

**Example of mean firing rate in a single SON oxytocin cell**



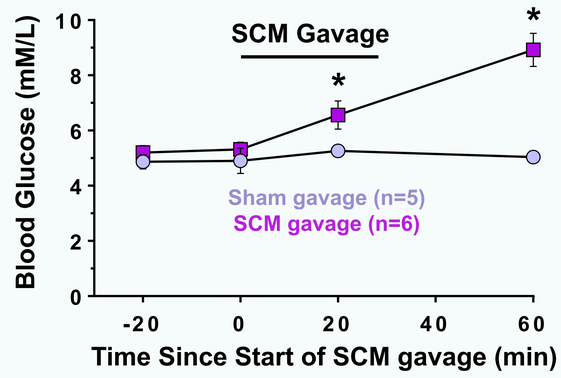

**Mean difference in firing rate for all SON oxytocin cells**




*p < 0.05 compared to t = -10
Kruskal-Wallis test p = 0.0016
(n = 9)

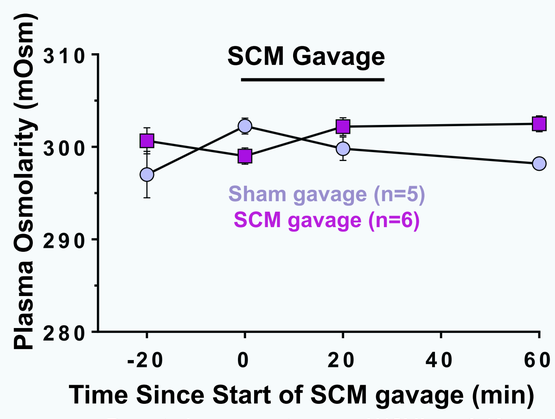
### (3) Blood glucose increases with SCM gavage but plasma osmolarity does not

**Blood glucose before, during and after SCM gavage**




*p < 0.05, Repeated measures two-way ANOVA with Bonferroni p = 0.006

**Plasma osmolarity before, during and after SCM gavage**




Repeated measures two-way ANOVA p = 0.12

## Conclusion

**SCM gavage results in increased c-Fos-like immunoreactivity and a sustained increase in the firing rate of SON oxytocin neurons. This increase in SON neuron activity is not due to a change in osmolarity. Instead, SON oxytocin neurons may be regulated by satiety-related peripheral signals released in response to food in the stomach, providing further evidence for oxytocin's role in the control of food intake.**

1. Johnstone et al., 2006. *Neuronal activation in the hypothalamus and brainstem during feeding in rats*. Cell Metab, 4(4), 313-321.

2. Sabatier, et al., 2003. *Alpha-melanocyte-stimulating hormone stimulates oxytocin release from the dendrites of hypothalamic neurons while inhibiting oxytocin release from their terminals in the neurohypophysis.* J. Neuroscience, 23:10351.

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