Supplementary Figures

Five supplementary figures:

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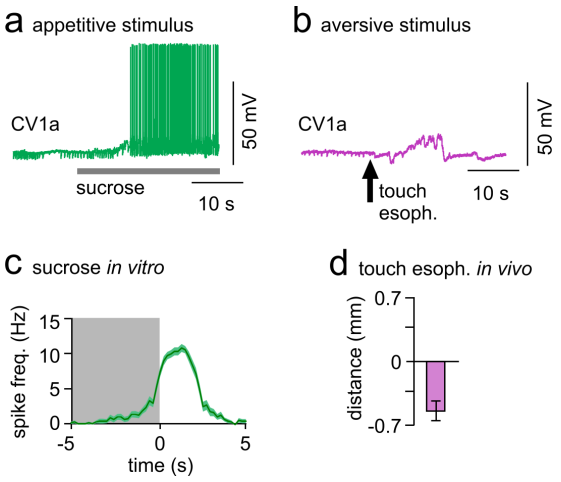
Supplementary Figure 2. Hunger does not prevent sensory driven egestion.

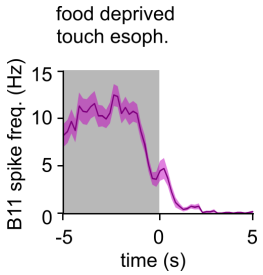
Supplementary Figure 3. Activity of PRN during ingestion and egestion.

Supplementary Figure 4. Sulpiride does not affect other aspects of feeding behavior.

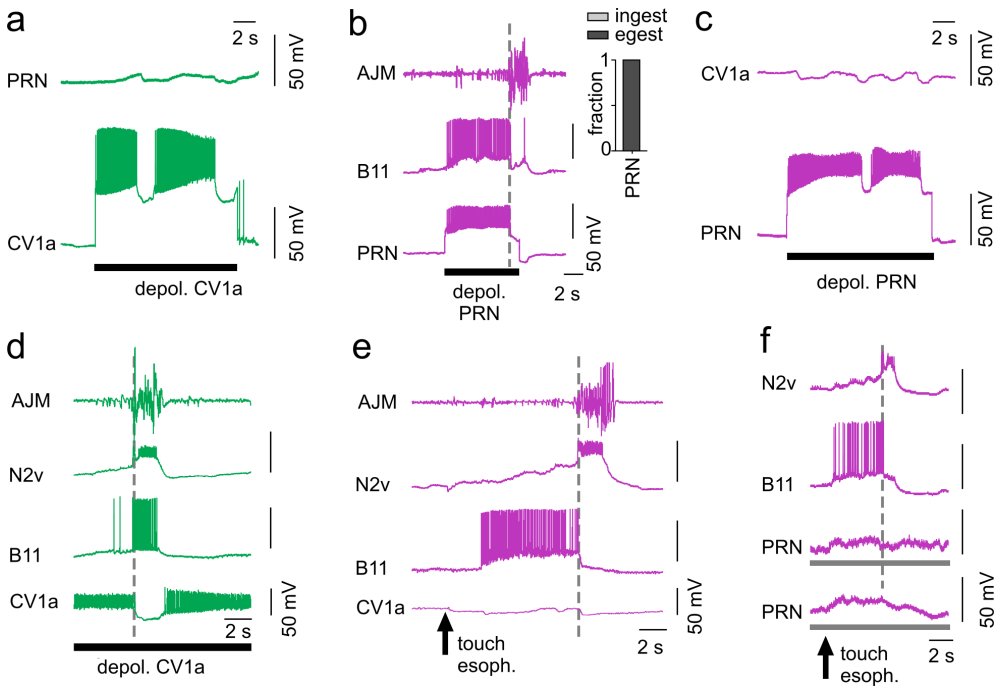
Supplementary Figure 5. Sulpiride does not alter RM firing properties.



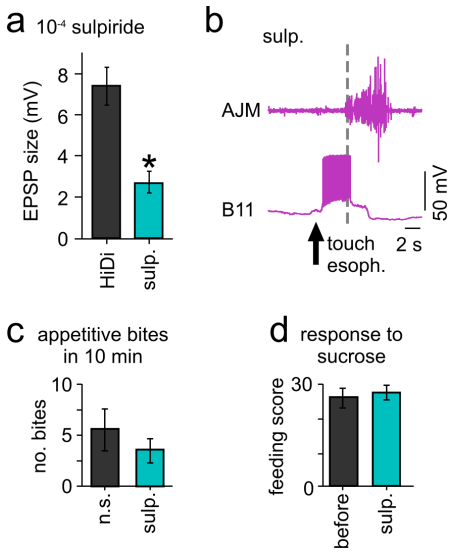
Supplementary Figure 1. Initiation of ingestion and egestion cycles. (**a**) Using a lip-CNS preparation the response of a single CV1a to sucrose solution was tested. CV1a was quiescent prior to sucrose application. Upon sucrose application, CV1a depolarizes and fires a burst of action potentials (n = 4). (**b**) Representative trace of a CV1a's response to tactile stimulation of the esophagus. CV1a shows no spiking activity during the sensory driven cycle (n = 10). (**c**) Average frequency of SLRT activity during sucrose driven cycles *in vitro* using a lip-buccal mass preparation (n = 42 cycles from 3 preparations). There was no significant difference in SLRT activity between cycles driven by sucrose (0.57 ± 0.13) or CV1a (0.77 ± 0.05, Mann Whitney test, p = 0.22). Line and shading represent mean and ± SEM respectively. (**d**) Bar chart of average radula movement in response to tactile stimulation of the esophagus *in vivo* (n = 6). Data represents mean ± SEM.



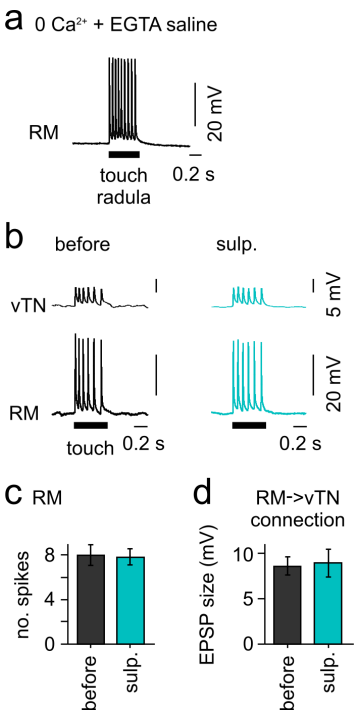
Supplementary Figure 2. Hunger does not prevent sensory driven egestion. Plot of the spike frequency of B11 activity in response to touch to the esophagus in preparations from food-deprived animals (n = 62 cycles from 8 preparations). B11 difference score = -0.8 ± 0.06. Line and shading represent mean ± SEM respectively.



Supplementary Figure 3. Activity of PRN during ingestion and egestion. (**a**) During CV1a driven ingestion cycles, PRN shows to spiking activity. Black bar represents duration of depolarizing current to CV1a. (**b**) Artificial activation of a single PRN is sufficient to drive a motor program, co-recorded with a B11 and AJM. B11 activity occurs prior to the onset of AJM burst and all cycles were egestive (n = 36 cycles from 8 preparations). Gray dotted line represents onset of AJM burst. Black bar represents duration of depolarizing current to PRN. (**c**) The ingestion-driving interneuron, CV1a, was not active during PRN driven cycles. Black bar represents duration of depolarizing current to PRN. (**d-e**) Representative traces of N2v activity during ingestion (d) and egestion (e) cycles. An N2v was co-recorded with a CV1a, B11 and AJM. During the CV1a driven ingestion cycle, the N2v plateau coincides with AJM activity as well as the high frequency activity in B11, signifying the onset of the retraction phase. CV1a activity ceases in the retraction phase due to inhibition[28]. During an egestion cycle driven by touch to the esophagus, N2v activity remains in phase with AJM, however, B11 activity occurs prior to the onset of both N2v and AJM. CV1a shows no activity during the cycle, as in Supplementary Figure 1B. Thus, N2v activity can be used to measure B11 activity against in a similar manner as AJM activity. (**f**) PRN is not necessary for touch to the esophagus driven egestion. Artificial hyperpolarization of both left and right PRN does not prevent egestion cycles (n = 3). B11 activity occurs prior to the onset of the N2v plateau. Gray bars represent duration of hyperpolarizing current to PRN. Gray dotted line represents onset of retraction phase.



Supplementary Figure 4. Sulpiride does not affect other aspects of feeding behavior. (**a**) Sulpiride reduces the PRN→B11 excitatory synaptic connection (see Fig. 4b for representative traces). EPSP size is significantly reduced after perfusion of 10^{-4} M sulpiride for 10 mins (n = 9, HiDi = 7.4 ± 0.9 mV, sulpiride = 2.7 ± 0.5 mV, paired t-test $p < 0.01$). (**b**) Sulpiride does not block the ability of the *in vitro* preparation to produce sensory triggered egestion cycles (n = 3). Representative trace showing that touch to the esophagus elicits an egestion cycle in the presence of sulpiride, recorded on a B11 and AJM. B11 activity precedes the AJM burst onset. (**c**) Appetitive food searching behavior in a novel environment was measured over a 10 min period in saline and sulpiride injected animals. There was no significant difference between the two groups (saline: 5.69 ± 2 bites, n = 16, sulpiride: 3.6 ± 1.2 bites, n = 15, Mann Whitney test, p = 0.63). (**d**) Feeding response to sucrose was not altered by sulpiride injection (saline: 26.1 ± 3 bites, n = 16, sulpiride: 27.7 ± 2.2 bites, unpaired t-test, p = 0.68.).



Supplementary Figure 5. Sulpiride does not alter RM firing properties. (**a**) Representative trace of a RM's response to tactile stimulation of the radula in zero Ca^{2+} + EGTA saline which blocks chemical synaptic transmission. RM somatic spikes persist, suggesting that RM is a mechanosensory neuron and that the response in RM is not due to a polysynaptic connection. (**b**) Sulpiride does not affect sensory processing of tactile stimuli. Representative traces of RM's response to tactile stimulation to the radula and its excitatory connection with vTN before (black trace) and during (teal trace) sulpiride application. (**c-d**) Statistical analysis of data from (b). There was no significant difference in the number of RM spikes elicited between conditions (before = 8 ± 0.9, during = 7.8 ± 0.7, n = 5, Wilcoxon signed rank test $p > 0.05$), amplitude of the spikes (before = 38.3 ± 1.9 mV, during = 37.1 ± 3.8 mV, n = 5, Wilcoxon signed rank test $p > 0.05$), interspike interval (before = 0.09 ± 0.01 s, during = 0.08 ± 0.01 s, n = 5, Wilcoxon signed rank test $p > 0.05$) or size of EPSP on vTN (before = 8.6 ± 1 mV, during = 8.9 ± 1.5 mV, n = 5, Wilcoxon signed rank test $p > 0.05$).