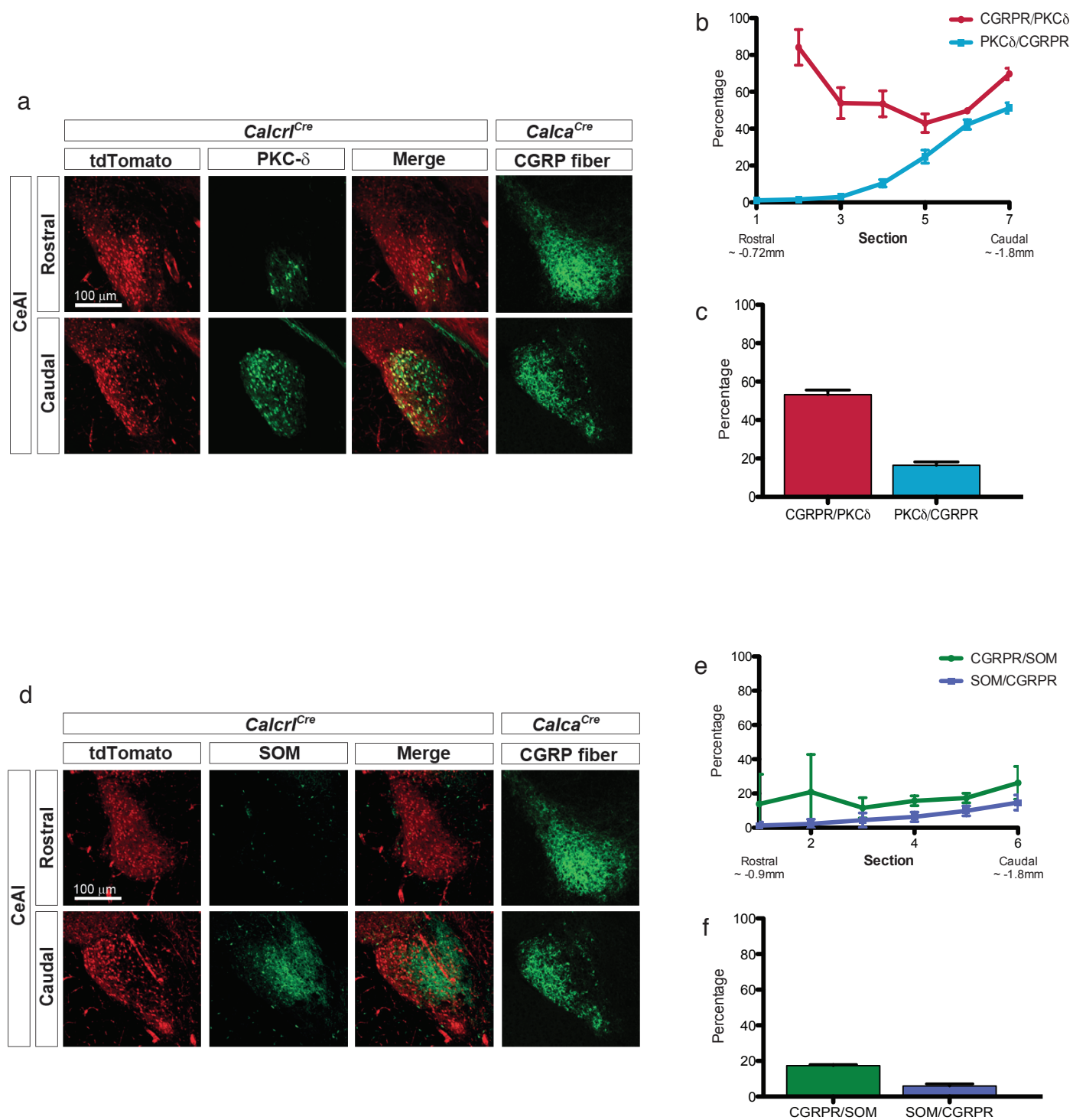
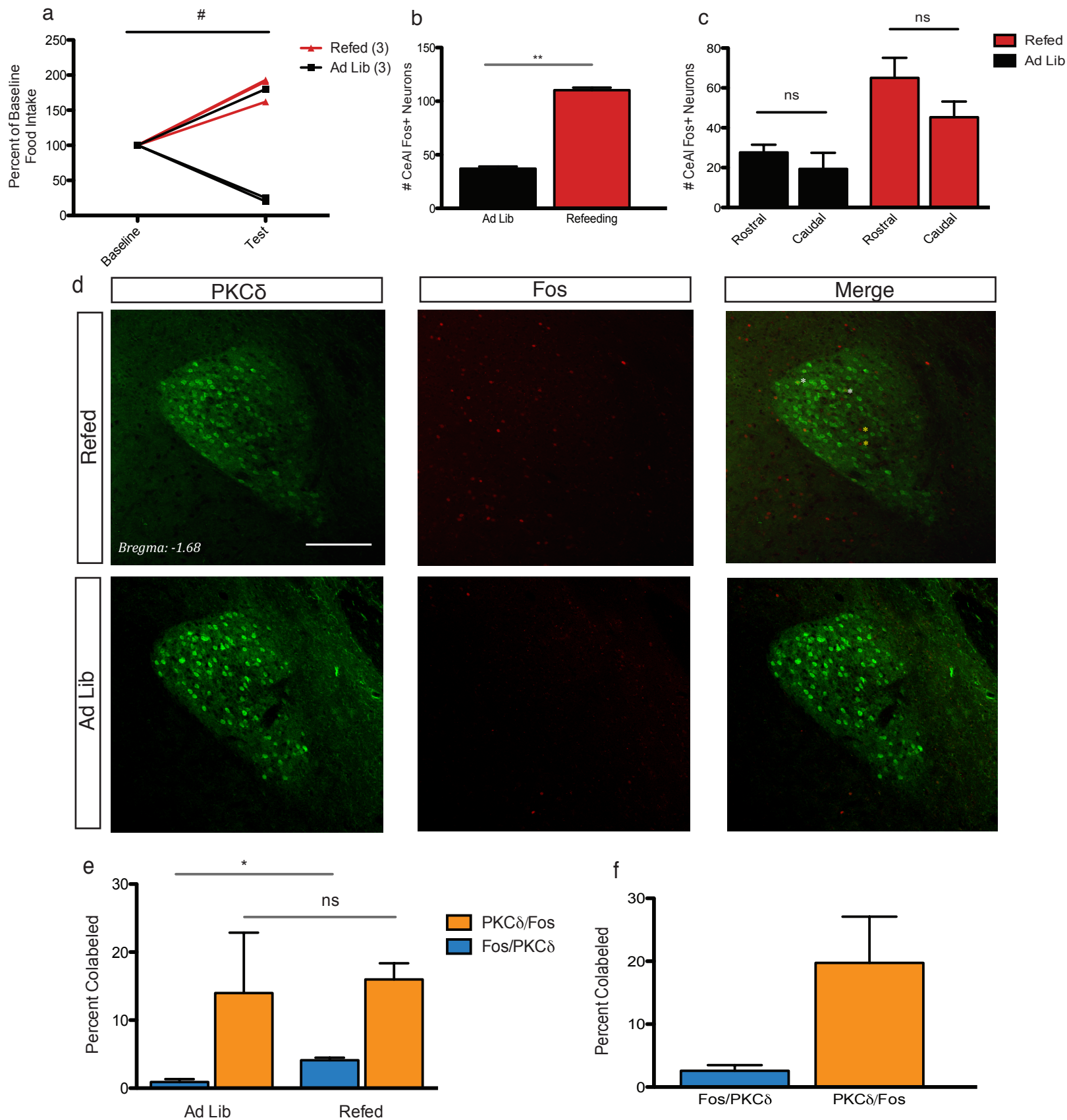


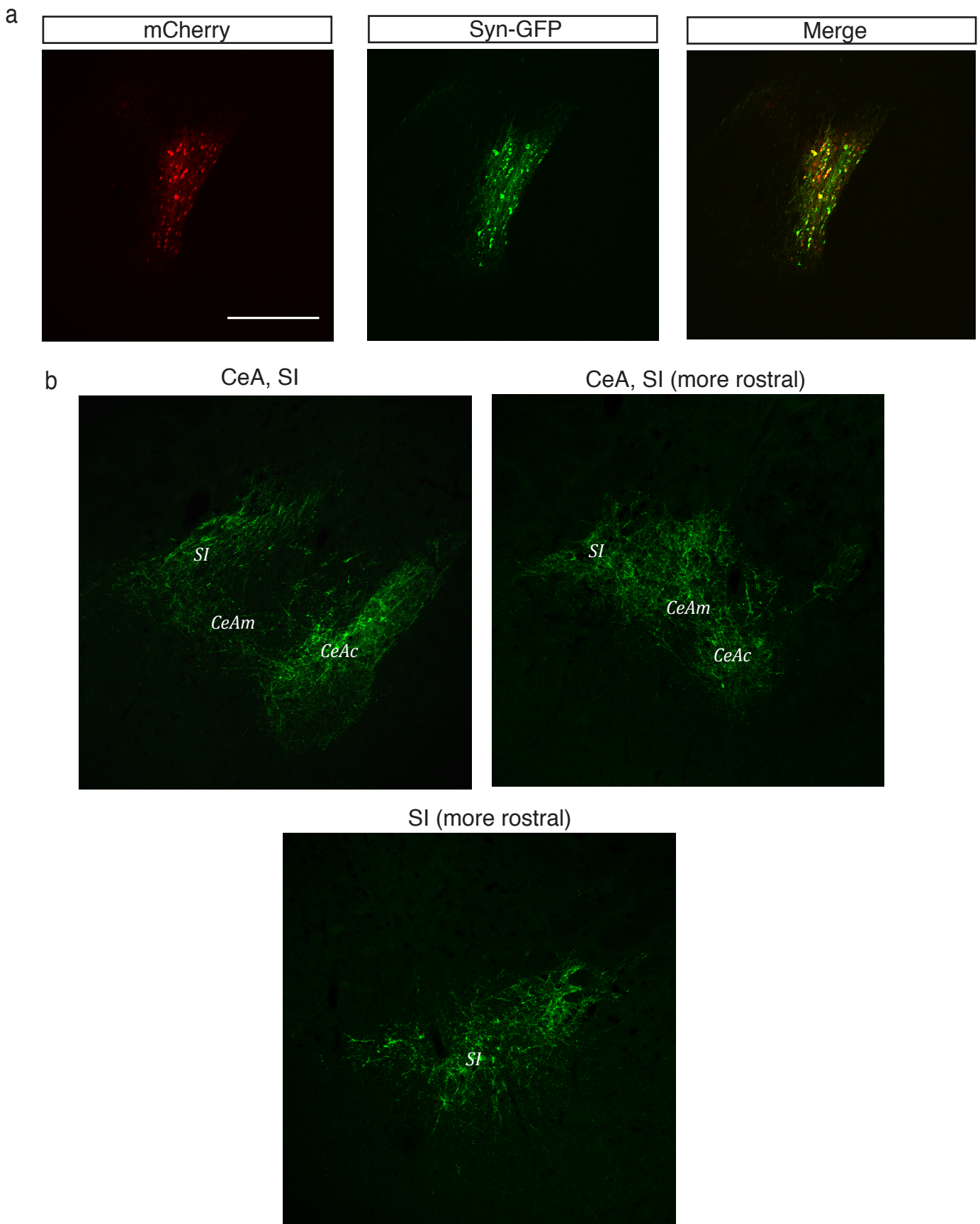
**Figure 1. Genetic gradients of the central amygdala.** **a.** Coronal image from the Allen Mouse Brain Reference Atlas highlighting the amygdala nuclei and the subdivisions of the central amygdala, CEAc - lateral capsular; CEAl - lateral; CeAm - medial **b.** Circuit model of the central amygdala, note that CeAl includes both the lateral capsular and lateral subdivisions, and that collaterals are used for simplicity without necessarily reflecting actual wiring, VR - vasopressin receptor; LHA - lateral hypothalamus; PVH - paraventricular nucleus of the hypothalamus **c.** Representative coronal image of the CeA and surrounding regions in *Calcr1;tdt* mice. Scale bar = 100 microns **d.** Representative coronal images of the rostral versus caudal CeAl across different markers. **e.** Quantitative analysis of genetic gradients across seven sections spanning the CeAl rostrocaudal axis.  $n = 3$  per group **f.** Summed neuron counts for each respective marker shows CGRPR<sup>+</sup> neurons significantly outnumber other markers. One-way ANOVA,  $p < .0001$ , Tukey's post-hoc tests: all  $p < .01$ , except PKC $\delta$  vs Tac2.  $n = 3$  per group.



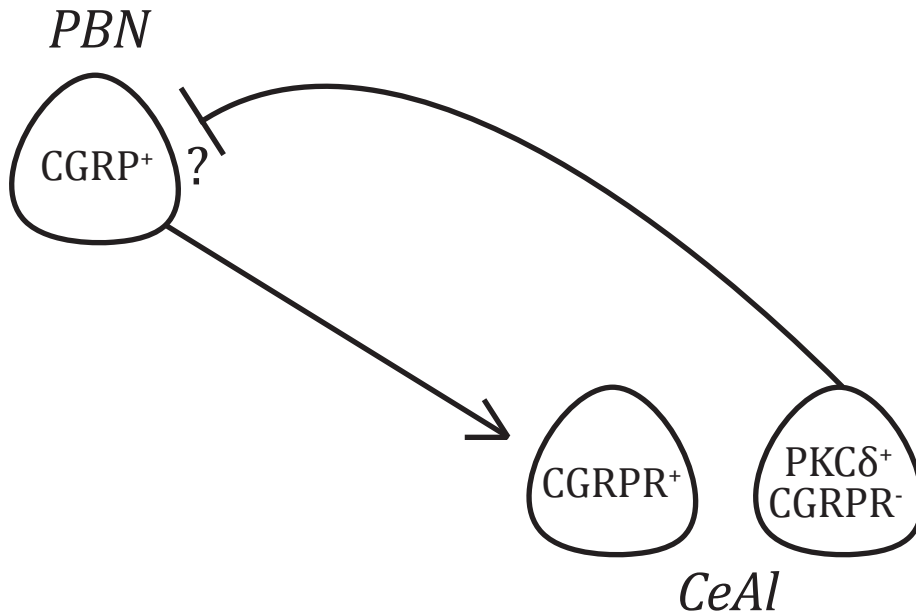
**Figure 2. Proportions of CGRPR<sup>+</sup> neurons coexpress PKC $\delta$  and SOM.** **a.** Representative coronal images of CGRPR and PKC $\delta$  colabeling in the rostral and caudal CeAl. PBN CGRP innervation parallels CGRPR expression. **b.** Quantitative analysis of the percentage of CGRPR<sup>+</sup> neurons coexpressing PKC $\delta$  and *vice versa* as a function of bregma. **c.** The two percentages collapsed across bregma. **d.** Representative coronal images of CGRPR and SOM colabeling in the rostral and caudal CeAl. CGRP innervation borrowed from **a.** **e.** Quantitative analysis of the percentage of CGRPR<sup>+</sup> neurons coexpressing SOM and *vice versa* as a function of bregma. **f.** The two percentages collapsed across bregma.  $n = 3$  for **a-c** and **d-e**.



**Figure 3. Satiety activates neurons across the CeAl.** **a.** Refed mice ate  $<2\times$  than those fed ad-lib. One-tailed t-test,  $\#p = .058$ . **b.** Refed mice had significantly more Fos $^+$  neurons than those fed ad-lib. Two-tailed t-test,  $**p < .01$ . **c.** The number of Fos $^+$  neurons did not significantly differ for the rostral versus caudal CeAl. Two-tailed t-tests. **d.** Representative coronal images showing that PKC $\delta^+$  and PKC $\delta^-$  neurons were Fos $^+$  in refed mice. White asterisks PKC $\delta^+$ , yellow asterisks PKC $\delta^-$ . Scale bar = 100 microns. **e.** Percentages of PKC $^+$  neurons that were Fos $^+$  and *vice versa* collapsing across bregma. Only the percentage of PKC $\delta^+$  neurons that were Fos $^+$  significantly differed between groups. Two-way ANOVA, post-hoc two-tailed t-test,  $*p < .05$ . **f.** The same percentages as in e, but only for the caudal CeAl of refed mice.



**Figure 4. CeAI CGRPR<sup>+</sup> neurons project locally and distally to the SI.** **a.** Cotransduction of CeAI CGRPR<sup>+</sup> neurons with mCherry and Syn-GFP to visualize somata and putative synapses respectively.  $n = 3$   
**b.** Representative coronal images of projections locally to the capsular and medial subdivisions of the CeA, as well as more rostrally to the substantia innominata (SI).



**Figure 5. Model for circuitry connecting the parabrachial nucleus (PBN) and central amygdala (CeA).** As the direct recipients of input from PBN CGRP<sup>+</sup> neurons, CeA CGRPR<sup>+</sup> neurons do not send reciprocal projections to the PBN. Rather, CGRPR<sup>-</sup>,PKCδ<sup>+</sup> project to the PBN, although monosynaptic input specifically to CGRP<sup>+</sup> neurons is still unclear. Arrowheads represent excitation, flatheads inhibition. Note the CeA includes the lateral capsular and lateral subdivisions.