The authors report that the SDF-1-EGFP reporter mouse had cells in the dentate gyrus filled with EGFP (Fig 1b) while SDF-1-mRFP1 mice had a more localized subcellular expression of mRFP1 (Fig 2a, 2a' and 2a''). Why?

a. The SDF-1-mRFP1 mice did not express the β and γ isoforms of SDF-1, allowing the mRFP to localize with the α-isoform.

b. The authors used a different BAC to insert the mRFP1 than the EGFP.

c. The SDF-1-mRFP1 construct is inserted with in the C-terminus of the SDF-1 gene where there are no cleavage sites, so the endogenous structure of SDF-1 is not disrupted by the fluorescent reporter gene.

d. The mRFP1 reporter gene has a selective promoter region for neural progenitor cells.

Why did the application of TTX (tetrodatoxin) *not* alter the effects of SDF-1 even when it was applied before SDF-1?

a. TTX binds to GABA receptors and SDF-1 enhances inward currents from GABA.

b. TTX binds to Na^{+} channels, which are not altered by GABA signaling.

c. TTX is quickly taken up by pre-synaptic reuptake enzymes and cannot have an effect on SDF-1 administration.

d. TTX is an agonist for Ca^{+2} channels is not altered by SDF-1 input.

Following their work with single gene transgenic mice, the authors developed SDF-1-mRFP/CXCR4-EGFP transgenic mice. As their microscopy images demonstrate in Figure 3, VGAT-positive nerve terminals were often near neural progenitor cells in the dentate gyrus. While this result is compelling (and difficult to obtain!), how could the authors have improved the specificity of their findings?

a. By labeling with a marker for a glutamate transporter in addition to a GABA transporter.

b. By labeling the slices with BrdU antibody to show neural progenitors.

c. By altering the gene construct for CXCR4-EGFP, such that the EGFP+ receptors would be properly trafficked to the membrane rather than filling each cell.

d. By labeling with Ki67 in addition to the other labels to distinguish neural progenitors.

In Supplemental Figure 3a, the authors demonstrate that the addition of SDF-1 enhanced the inward current elicited by GABA administration. Considering what you know about chemokine receptors from your readings and the lecture, which of the following is a potential mechanism for this enhancement?

a. CXCR4 changes its conformation when bound to SDF-1 to enhance GABA binding at its receptor, leaving it open for longer periods of time.

b. The α subunit of the CXCR4 receptor elicits phosphorylation of the GABA receptor, allowing it to stay open longer.

c. The signaling pathway from the CXCR4 receptor increases intracellular Ca^{+2} concentration, which alters the opening and closing of Cl^- channels in the membrane.

d. The signaling pathway from the CXCR4 receptor reduces intracellular Ca^{+2}, such that Na^+ flows into the cell at greater rates.