LY37 (glu agonist) increases 2AR affinity for its agonists (2008). But it decreases efficacy of 5HT at Gq (2011).
Potency changes? Where are the dose response curves?

Simply the presence of the mGluR receptor decreases efficacy of 5HT (2011).
Is this the case for the hallucinogens (super agonists) too?
They are reported to have different "ligand biases" for PLC vs. PLA2 signaling.
(and differences with regards to cfos and egr-2 activation).

What does the inverse agonist do to affinity (it increases efficacy of 5HT)?

DOI (2AR agonist) decreases Glu2/3R affinity for its agonists (2008), and also decreases efficacy of the agonists (2011).

What does the inverse agonist do to affinity (it increases efficacy of Glutamate).

Cfos activation by 2AR was not affected by LY37.
Egr-2 induction by 2AR was blocked by LY37 (as was the head twitch).
Homomer vs. heteromer signaling?
How does endogenous tone play in here?

What about changes in coupling.
Might the heteromer bound to 5HT now signal to Gi?
Might the heteromer bound to Glu now signal to Gq?
There are hints of this in Fig. S4.
What experiments are missing that would test for this?

What experiments did they do to demonstrate heterodimerization rather than cross talk was responsible for the changes in efficacy?