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Stepmania 2.3.5 Build 1802 + Full Crack type "Log in". were found in S1P/RXF5 and RXF7 (3/4). S1P/RXF5 was very sensitive to the effect of exogenous fatty acids, showing a more than 12-fold increase in potency following supplementation with a mix of saturated and unsaturated fatty acids (1 mM) for 24 h. This observation led to our initial suggestion that the S1P/RXF5 site is an allosteric site that may be allosterically regulated by fatty acids. However, RXF7 showed only a small and nonsignificant increase in potency following exogenous fatty acids and S1P, suggesting that RXF7 may represent a second PAM site that requires the presence of a specific agonist for efficacy. Given that RXF7 showed little change in efficacy following modification with exogenous fatty acids, we hypothesized that the RXF7 site may represent a highly selective allosteric binding site. Exogenous fatty acids only modestly increase efficacy of a known RXF-family ligand, the well-described RXFP3 (also known as GPR23) site (Δ [@R40]; Δ [@R30]). Thus, the RXF7 site is an effector of a novel mechanism of GPCR allostery that is likely distinct from activation of allosteric sites known to be critical for GPCR activation. In agreement with this hypothesis, RXFP3 agonists are structurally distinct from other known RXF ligands ([Fig. S3](#SD1){ref-type="supplementary-material"}). Although RXFP3 is activated by full-length S1P ([@R31]; [@R50]), RXF7 shows little activation by full-length S1P. On the other hand, RXFP3 and RXF7 both show strong activation by the core domain of S1P and RXF5, suggesting a contribution of the N-terminal domain of S1P to the allosteric action of RXFP3 and RXF7. Given the lack of contribution of the N-terminal domain to the activation of RXFP3 and RXF5, it is likely that RXF7