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Biased Agonism in GPCRs: An Opportunity for Drug Discovery

Guest Editor:

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Message from the Guest Editor

G protein-coupled receptors are membrane proteins responsible for signal transduction from the outside of the cell to the inside. GPCRs, either free or bound by their endogenous agonists, may recognize G proteins and β-arrestin transducer proteins with differential affinity, which leads to distinct signaling propensities. Alteration of the normal signaling propensity of GPCRs may have pathological consequences. Moreover, because signaling pathways may have their own therapeutic or pathophysiological effect, the classic drug discovery paradigm has recently changed to signaling-pathway specific. These pharmacological and medicinal chemistry aspects of GPCR signaling encompass the concept of functional selectivity or biased agonism.

Biased agonism is at the center of current pharmacological research. Because of contradictory results between pharmacological assays and between laboratories, further work, with integrative and complementary approaches at both experimental and computational levels, is needed to mechanistically explain the functional connection between the pieces of this biological puzzle. This Special Issue is aimed at providing a step forward towards this goal.

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Message from the Editor-in-Chief

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