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ISSN: 2161-1459
Journal of Clinical & Experimental Pharmacology

Perspective

Mechanism of Various Drug Actions and their Interactions

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DESCRIPTION

A "drug-drug interaction" is defined as a pharmacological or clinical response due to the administration of a combination of drugs that differs from the expected effects of the two drugs administered alone which cause harm to the patient. Over 100,000 potential drug interactions have been documented, but most of the drugs does not cause actually adverse reactions.

Drugs used to numb a specific area are called as local anesthetics. anesthetics) often contain a combination of lidocaine and epinephrine, causing blood vessels to constrict, thereby Prolongs the action of lidocaine in the injection area. Antidotes such as naloxone (Narcan) are given after surgery to stop the narcotic's effects. In cancer therapy, multiple drugs are administered simultaneously to target multiple sites of cancer cell growth. Beneficial drug interactions can enhance treatment. Knowledge of potential drug interactions is an important part of pharmaceutical care. The term "drug interactions" includes drug-disease interactions, drug-food interactions.

In pharmacology, the term Mechanism of Action (MOA) describes the specific biochemical interaction by which a drug exerts its pharmacological effect. Mechanisms of action usually involve specific molecular targets to which the drug binds. Receptor sites have specific affinities for drugs based on the drug's chemical structure and the specific effects that occur at recognition site. Agonist is a drug that binds to a receptor, thereby altering (stabilizing) the proportion of the receptor in its active conformation and producing a biological response. Full agonists produce the greatest response by occupying all or part of the receptor. Partial agonists result in less than maximal responses even when the drug occupies all receptors.

There are four types of drug antagonism. Chemical antagonism involves a chemical interaction between a drug and a chemical or another drug, resulting in diminished or there is no response. Physiological antagonism occurs when two drugs that act on different receptors and pathways have opposite effects on the same physiological system. Pharmacokinetic antagonism is the result of one drug inhibiting the action of another by decreasing absorption, altering distribution, or increasing the rate of excretion. Pharmacological antagonism occurs when an antagonist inhibits the action of a full or partial agonist by acting on the same pathway, but not necessarily the same receptor. Pharmacological antagonists consist of three subcategories. Reversible competitive antagonists produce inhibition that can be overcome by increasing concentrations of agonist. The presence of a reversible competitive antagonist causes a rightward parallel shift of the agonist's logarithmic concentration-response curve.

Irreversible competitive antagonists also involve competition between agonist and antagonist for the same receptor, but stronger avidity prevents complete reversal of antagonist effects, even at high agonist concentrations. The presence of an irreversible competitive antagonist causes a rightward shift in the agonist's logarithmic concentration-effect curve, generally exhibiting a decreased slope and reduced maximal effect. Non-competitive antagonists inhibit agonist activity by blocking one of the subsequent responses between receptor activation and pharmacological response. Non-competitive antagonism is usually reversible, but not reversible. Non-competitive and irreversible competitive antagonists cause similar perturbations in the logarithmic concentration-response curves of agonists. Since non-competitive antagonists are generally reversible, isolated tissue experiments are used to distinguish between the two subcategories.

Agonists can be effective even if they bind to the same site on the same receptor as antagonists. Explained by both structural and functional studies, receptors exist in at least two conformations, active and inactive, suggesting that these are in equilibrium. Because agonists have a higher affinity for the active conformation of the receptor, they drive the equilibrium toward the active state, thereby activating the receptor. Conversely, antagonists have a higher affinity for the inactive structure of the receptor, shifting the equilibrium to the inactive state and producing no effect.

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Received: 25-Oct-2022, Manuscript No. CPECR-22-18806; **Editor assigned:** 28-Oct-2022, Pre QC No. CPECR-22-18806 (PQ); **Reviewed:** 11-Nov-2022, QC No CPECR-22-18806; **Revised:** 18-Nov-2022, Manuscript No. CPECR-22-18806 (R); **Published:** 25-Nov-2022, DOI: 10.35248/2161-1459.22.12.339.

Citation: Derebe D (2022) Mechanism of Various Drug Actions and their Interactions. J Clin Exp Pharmacol. 12:343.