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Short Communication

Paradoxical Pharmacology of Modern Pharmacodynamics

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DESCRIPTION

Pharmacodynamics (PD) is the study of the biochemical and physiological effects of drugs, especially pharmaceuticals. Effects may include those appearing in animals (including humans), microorganisms, or combinations of organisms (such as infections) [1].

Pharmacodynamics and pharmacokinetics are major areas of pharmacology, themselves topics of biology that are of interest in the study of interactions between organisms and endogenous and exogenous chemicals.

Specifically, pharmacodynamics is the study of how drugs affect organisms, affecting dosage, benefits, and side effects. Recently, the concept of pharmacodynamics has been expanded to include Multicellular Pharmacodynamics (MCPD). The MCPD concept helps researchers understand the dynamic and static relationships between drugs and the multicellular, four-dimensional organization of organisms. In this way, the effects of drugs on minimal multicellular systems can be investigated both *in vivo* and *in silico*. Networked multicellular pharmacodynamics extends the concept of MCPD to include precise modeling of regulatory genomic networks combined with signaling pathways. These concepts allow us to more effectively study the complex interacting components and drug effects in cells [2-4].

Current pharmacodynamics modeling problems are the scalability of data from preclinical animal studies to human studies and how to accurately predict drug behavior in the human body.

By integrating information from in vitro HE bioassays and preclinical animal pharmacology studies, scientists can predict clinical and adverse drug effects. PK/PD models have several important drug- and system-specific factors, such as the time course and intensity of a drug's pharmacological effect [5].

Pharmacodynamics is the study of subatomic, biochemical, and physiological effects of drugs or activities. All drugs exert their effects by interacting with organic structures or by focusing below the atomic level, which helps modify the influence of particles on intermolecular interactions. These associations include receptor restriction, post-receptor effects, and performance interactions. Conditions for this type of interaction include drugs that inhibit the active chemical milieu, tranquilizers that bind to cell signaling proteins and destroy downstream flags, and drugs that deplete atoms such as: Oncogenic growth factors [6].

Antipsychotics interact with many transmitters, target receptors throughout the body as well as the brain, and cause a number of known side effects that make schizophrenia treatment less effective. Due to differences in pharmacokinetics and pharmacodynamics, clinically relevant side effects differ between men and women. Changes in sex steroid levels throughout life, especially at critical times such as puberty, pregnancy, menopause, and aging, affect drug uptake, distribution, metabolism, and excretion, as well as binding to neurotransmitter receptors. There are also important effects of exogenous hormones, hormonal contraceptives, menopausal hormone replacement, transgender therapy, and performance-enhancing steroids play an equally important role [7].

The above determine not only the effectiveness of the drug, but also its side effects. In a cross-sectional study, Iversen et al found that over 75% of those taking AP reported side effects, extrapyramidal effects, sexual symptoms, sedation and weight gain. Twice as many women as men say these effects are severe.

The relationship between drug concentration and observed pharmacological response depends on the mechanism by which the drug exerts its effect. Responses may be the result of direct reversible effects mediated by binding to specific receptors (e.g., β-adrenergic receptor blockers). For these drugs, there is a relatively simple and direct relationship between drug concentration and pharmacological effect. Responses to other drugs are due to indirect effects. The best example is warfarin, which blocks the synthesis of vitamin K-dependent clotting factors but does not affect the breakdown of these same factors. In this case, drug concentration may be related to clotting factor synthesis, but only indirectly to the observed anticoagulant

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effect. Examples of drugs with irreversible effects are acetylsalicylic acid Against Platelet Aggregation (APA), omeprazole, bactericidal antibiotics, and some antineoplastic agents.

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