



## Beneficial Drug Interactions in the Field of Dentistry

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### Pharmacodynamics Interactions

Pharmacokinetic interactions are those that modify the pharmacological impact of a drug without changing its concentration within the tissue fluid. This implies that the impact of one drug is changed by the presence of another drug at the same molecular site, driving to a summation, or opposition, of the effects of the drugs. These interactions may be outlined into antagonistic, additive or enhanced impacts.

#### Antagonistic effects

This is an interaction between two drugs that have restricting pharmacological effects, driving to a diminished effectiveness of one or both of the drugs administered. A typical case would be for drugs that tend to increase blood pressure, such as non-steroidal anti-inflammatory drugs (NSAIDs); these may repress the antihypertensive impact of drugs such as angiotensin-converting chemical (Pro) inhibitors or diuretics.

#### Additive and enhanced synergistic effects

When two or more drugs are given with the same pharmacodynamics profiles, the additive impacts of the two together may result in an excessive reaction of the target tissue and hence toxicity. An 'additive synergy' is when the ultimate impact is equal to the whole of the impacts of both drugs. When the ultimate impact is greater than additive, it is known as 'enhanced synergy' or potentiation. A case of a synergistic impact would be the synchronous arrangement of a benzodiazepine and opioid medication. Both drugs are pharmacodynamically comparative and if given together can lead to an increment within the sedative impact on the central nervous system, which may cause respiratory depression.

### Pharmacokinetic Interactions

Pharmacokinetic interactions are those that modify the concentration of a medicate that comes to its location of action. Therefore, one medicate changes the concentration of another drug within the system (either an increase or diminish). This modification may happen at any phase of absorption, distribution, metabolism or excretion of the drug.

#### Absorption

Drugs collaboration with the gut lumen or those that impact gastric motility or emptying can alter the rate of absorption of a

second drug. For illustration, certain antibiotics, such as penicillin-based antimicrobials, can modify the ordinary gut flora; a few of which are vital for the generation of vitamin K, driving to a decrease in vitamin K synthesis. Vitamin K is basic for the generation of a few clotting components and any deficiency will lead to an increment within the patient's international normalised ratio (INR) and bleeding tendency. Other absorption pharmacokinetic interactions may be related with a change of the pH inside the stomach. Specific drugs, such as ketoconazole (an antifungal agent), can be absorbed in acidic conditions. In this manner any drugs that adjust the gastric pH, such as antacids (e.g. calcium carbonate), proton-pump inhibitors (e.g. omeprazole) or H<sub>2</sub>-receptor blockers (e.g. ranitidine), subsequently diminish the absorption of ketoconazole.

#### Distribution

Drugs circulating inside the bloodstream are distributed in two forms; either bound to circulating plasma proteins (pharmacologically inert or dormant), or the drug isn't protein bound and in this way free to associated with the target receptor official sites. The number of protein binding sites inside the body is limited and when another drug competes for the same site it'll result in an increase within the unbound and active drug.

An example includes warfarin and its interaction with aspirin and other NSAIDs. All these are transcendently plasma protein-bound in circulation and in this way compete for the same plasma protein-binding site. This leads to more anticoagulant being displaced from the binding site, which in turn leads to an by and large increment within the free, and thus pharmacologically active, warfarin. As warfarin is 98–99% plasma protein bound, this regularly takes off 1% free to exert its pharmacological impact. Hence, in case an NSAID or aspirin is given and diminishes the plasma protein-bound warfarin from 98% to 97%, in spite of the fact that that's as it were an increase in 1% unbound warfarin, it is successfully doubling the concentration of the active drug within the blood, hence raising the INR.

#### Metabolism

This is where there's a decrease in drug efficacy (the desired effect of the drug) due to enzymatic induction by a secondary drug in circulation. One essential location for drug digestion system is the liver, which renders the drug either inert or changes the structure of the drug to empower it to be excreted by means of the kidneys. Alternately, the liver or other metabolically active organs such as the brain may increase the efficacy or activate a drug by changing over a pro-drug to a more, or fully, dynamic shape. This can be seen within the case of levodopa, a sedate used within the treatment of Parkinson's disease. Levodopa is a pro-drug, and is as it were converted into active dopamine within the central nervous system after crossing the blood-brain barrier (which is invulnerable to dopamine).

Various drug-metabolising iso enzymes are involved with drug biotransformation, the foremost common being cytochrome CYP450. These proteins can either be repressed or initiated by certain drugs (rifampicin, which induces CYP450, and leads to increased digestion system of the oral contraceptive pill driving to its failure).

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Received: November 4, 2021 Accepted: November 19, 2021 Published: November 26, 2021

### **Excretion**

Drugs or metabolites are transcendentally excreted via the kidneys. This can be too a potential location for drug intelligent when two drugs share the same excretion site, reducing the rate of excretion of one or both of the drugs. This means that the dynamic form of the

sedate is kept in the circulation for longer, successfully drawing out its restorative impact with possibly hurtful consequences. Aspirin and other NSAIDs restrain discharge of methotrexate into the pee. This could potentially lead to harmful methotrexate poisonous quality and care ought to be taken when prompting patients to take analgesics such as NSAIDs when they are on these drugs.

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