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Common Drug Interactions that Affect Therapeutic Efficacy

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Background

Ever wondered why certain medications prescribed by medical experts fail to deliver therapeutically? With billions of drug prescriptions filled in each year, it is imperative for drug users to know about drug interactions that can result in loss of efficacy and adverse drug reactions in order to avoid problems. This short commentary is to respond to the educative needs of drug users on the implications of common food-drug combinations. This article will address the appropriateness of food-drug combinations with respect to dose and timing. Literature abound to support claims for the interactions between bioactive constituents of food and drugs. These interactions may occurs in at least one of the following phases namely: absorption, metabolism, pharmacokinetic and pharmacodynamic stage [1]. Food-drug interaction results from the unique physical, chemical and physiological relationship between a drug and food consumed for the sole aim of harnessing nutrients. Dietary components can increase or decrease drug exposure increasing the risk of toxicity (adverse drug reactions) of loss of drug efficacy respectively. This happens through mechanisms which have physiologic, physicochemical and biochemical undertones [2].

Now we look at common, specific cases 'close-to-home' of food-drug interactions, drug action and how they are affected by the factors [1,2] named above.

Efficacy of antibiotics is affected by Ca^{2+} rich food (s): Such as tetracyclins and flouroquinolines can bind to divalent cations such as Ca^{2+} present in dairy products leading to reduced drug absorption and loss of drug efficacy. Several mechanisms have been suggested for this outcome however, the most plausible is competitive antagonism between Ca^{2+} and the antibiotics. This results from the affinity of the two for same carrier and binding site.

Fatty meals increase absorption of lipophilic drugs: As opposed to '1' above, fatty meals increase the absorption of lipophilic drugs leading to increased drug exposure and toxicity. In many parts of the 'third world' where animal fat is used for frying as an alternative to plant oils may be at risk not only of cardiovascular diseases but also fat-lipophilic drug-interaction toxicity.

Carbonated soft drinks enhance absorption of painkillers: Some individuals swallow drugs with carbonated drinks in order mask the unappealing taste of drugs. Analogous to '2' above, addition of CO_2 to soft drinks under pressure results to the formation of carbonic acid hence making the solution acidic. Carbonic acid though a weak acid increases the bioavailability of painkillers in the blood above therapeutic thresholds which leads to toxicity [3]. Caffeine present in some soft drinks and caffeine also enhance the absorption of drugs especially painkillers and antipsychotics. More recently, drug abusers consume tramadol an opoid pain medication in solution of soft drinks. This combination is capable of rapid absorption hence reaching a very high serum concentration in short time.

K^+ rich leafy vegetables interferes with anti-coagulant drugs: Leafy vegetables are rich in Fe^{2+} and cofactors such as K^+ important for the blood clotting biochemical pathway. By implication consumption of leafy vegetables equips the body for quick recoveries from open wounds. However, in certain disease states such as heart diseases were blot clot is a risk factor, anti-coagulant drugs e.g aspirin and warfarin are administered. K^+rich leafy vegetables e.g. spinach interferes with these blood thinning anti-coagulants hence leading to increased formation of clots. Note however, that consumption of moderate amounts of leafy vegetables may not be detrimental in individual receiving warfarin treatment.

Alcohol enhances drug toxicity: It is common knowledge that poisons act faster when consumed with alcohol. In fact, this may be the motivation to 'spike' alcoholic drinks with date rape drugs. The reason for this is not farfetched. Alcohol is metabolized by the Cytochrome P450 which is incidentally responsible for general drug metabolism [4]. It has been demonstrated through experiments that alcohol increases expression and activity of many isozymes of Cytochrome P450. This drug metabolism is required for activation of drug compounds into their active forms which could be either detrimental or therapeutic. Administration of a potentially toxic drug with alcohol enhance the rate of formation of the active species and severity of toxicity.

Vitamin C diminishes efficacy of anti-malarial drugs: Malaria parasite requires Iron present in blood for survival. Vitamin C (present in orange, grape, other common fruits and as tablets) often co-administered with anti-malarial drugs such as artemisinin and its derivatives is both an anti-oxidant and enhancer of Iron uptake by the liver. Paradoxically, artemisinin based anti-malarial drugs elicit their therapeutic effects via a pathway that involves production of anti-plasmodial oxidative derivatives by a Fenton's reaction [5]. These derivatives are oxidative radicals that can be scavenged by Vitamin C hence neutralising the effect of the artemisinin intervention.

The knowledge of how foods may interact with drugs are of great benefit to drug users. Tremendous amount of effort has gone into understanding the mechanisms underlying the food-drug interactions however, since they are no standard models for predicting food drug interactions our understanding of the matter is experiential. It is therefore necessary for drug users to bear in mind the following questions:

- What foods should I avoid when at risk or suffering certain medical conditions?
- Can I retain my feeding routines while taking certain drugs?
- What are the possible foods that may negatively affect my drug prescription?
- Can I alleviate food-drug interaction by working on the timing of drug/food administration?
- Is the doctor aware of my feeding habits?

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