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Commentary

Efflux and Influx Transporters of the Cytochrome Enzyme Drug Interaction

Ajay Dubey*

Department of Pharmaceutical Science, UOS College of Pharmacy, Sharjah, United Arab Emirates

ABOUT THE STUDY

Drug transport proteins have turned into a significant new topic in the last decade that is critical to understanding response to therapy. Many drug interactions that were previously unfathomable due to their impact on the cytochrome P450 (CYP) enzyme system can now be explained by their impact on influx and efflux cell membrane transporters. The cytochrome P450 enzymes are required for the production of a variety of substances, including cholesterol and steroids. They are also required for the detoxification of foreign substances as well as medication metabolism. Drugs that cause CYP450 drug interactions are referred to as either inhibitors or inducers. Regarding drug clearance, these cell membrane transporters frequently collaborate with drug-metabolizing enzymes, such as the CYP enzyme system. The enzyme CYP plays a significant role in drug metabolism. Since different pharmaceuticals inhibit, induce, and compete for common enzymatic pathways, it is the cause of numerous drug interactions. Unpredictable pharmacological effects can also be attributed to CYP genetic variations. Influx cell membrane transporters are required not only for the intracellular movement of nutrients, but also for the intracellular movement of prescription drugs, where they will come into contact with enzymes responsible for their metabolism and preparation for abolition, or potentially to exert their pharmacological effects. As a result, everything which blocks or inhibits a medication from entering target cells for metabolism or performing its therapeutic value could result in a high plasma concentration of the drug, causing unwanted side effects. A number of drugs can inhibit or induce influx cell membrane transporters in addition to inhibiting or inducing Cytochrome enzymes involved in drug metabolism. When evaluating medication interactions, practitioners must be aware of the role and degree of influence that each of these systems plays.

The much more frequent transporters known to date are divided into two categories which are super families, including ATP Binding Cassette (ABC) transporters and Solute Associated Carriers/Transporters (SLC). The genes that code for each type of transporter are identified in part by the superfamily out of which they derive. The multiple ABC transporters, for example, have at least 49 genes. There are at least 43 families of SLC transporters, encompassing over 300 distinct transporters. As a result, every transporter gene has its unique name. Furthermore, each gene may use a distinct transcription factor to activate gene transcription and produce additional transporters. Clinicians would be able to find drugs that can inhibit or promote the function of a transporter if they recognize that pharmaceuticals can influence gene transcription by modulating transcription factors. Medication can alter transporter function in the same way that it can influence active Cytochrome enzymes or their synthesis to induce a change in another medication's concentration. To make matters even more complicated, several drugs affect both metabolic enzymes and transporters. The majority of influx transporters belong to the SLC superfamily, whereas efflux transporters belong to the ABC superfamily. They are both found in the brain, gastrointestinal tract, liver, and kidney, regardless of the kind of transporter. Understanding the role of influx transporters in the transit of drugs throughout the body is critical for determining a drug's efficacy, metabolism, excretion, and potential for creating or being engaged in drug interactions.

For instance, the Blood–Brain Barrier (BBB) inhibits most pharmaceuticals from reaching the brain. This characteristic is due to epithelial-like tight junctions within the brain capillary endothelium. The BBB is physically and physiologically separate from the choroid plexus' blood–cerebrospinal fluid barrier. Some specific molecular pharmaceuticals that have a molecular weight of 450 Da and make less than 8 hydrogen bonds can penetrate the BBB *via* lipid-mediated free diffusion. The most of small molecule pharmaceuticals and all large molecule pharmaceuticals lack these chemical features. Nonetheless, using understanding of the BBB's endogenous transport systems, medicines can be reengineered for BBB transport. The detailed functions of the described ABC and SLC transporters in brain are still poorly understood. The research data of Knock-out mice of the ABC and some SLC transporters are available, but have been mainly used to study the role of these transporters for brain penetration of certain drugs.

Correspondence to: Ajay Dubey, Department of Pharmaceutical Science, UOS College of Pharmacy, Sharjah, United Arab Emirates, E-mail: dubeyA@yahoo.com

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