



Transporter Genomics: Genetic Control of Molecular Traffic and Medication Disposition

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DESCRIPTION

Transporter genomics examines how genetic differences in membrane transporter proteins influence the movement of medicines and endogenous molecules throughout the body. Transporters are proteins located on cellular membranes that control the entry and exit of numerous compounds. They participate in intestinal absorption, tissue distribution, hepatic uptake, biliary secretion, renal elimination and movement across specialized barriers. Differences in transporter genes can alter protein activity or expression and consequently affect drug concentrations, therapeutic effects and unwanted reactions. The human genome contains many genes that encode membrane transporters. These proteins belong to several families, including solute carrier transporters and Adenosine Triphosphate (ATP) binding cassette transporters. Solute carrier proteins generally facilitate movement of molecules across membranes using concentration gradients or ion-coupled processes. ATP-binding cassette proteins use cellular energy to move selected substances across membranes. Both groups have important functions in pharmacology.

Transporter activity begins when a pharmaceutical compound reaches a cell membrane. Depending on the transporter, the protein may facilitate movement into the cell or remove the compound from the cellular environment. The location of the transporter is also important. Transport proteins in intestinal cells can affect the amount of a medicine entering the bloodstream, while hepatic and renal transporters can influence elimination. The ATP-Binding Cassette Subfamily B Member 1 (ABCB1) gene, which encodes P-glycoprotein, is one of the best-known transporter genes in pharmacology. P-glycoprotein acts as an efflux transporter and can move various compounds out of cells. It is expressed in tissues such as the intestine, liver, kidneys and blood-brain barrier. Genetic differences in ABCB1 may influence transporter expression or activity, although the clinical significance can differ according to the medicine and genetic variant involved.

ABCG2 is another important transporter gene. Its protein product, breast cancer resistance protein, participates in the transport of several pharmaceutical compounds and endogenous molecules. It is present in tissues involved in absorption and elimination. Variants in ABCG2 can alter transporter function and may influence drug exposure. Certain genetic variants have been associated with differences in the handling of medicines used for cardiovascular, metabolic and other conditions. Organic anion transporting polypeptides are encoded by members of the Solute Carrier Organic Anion Transporter (SLCO) gene family. These transporters facilitate the uptake of selected compounds into cells, particularly in the liver. SLCO1B1 is an important example because its protein product transports several medicines from blood into hepatocytes. Genetic differences in SLCO1B1 can reduce transporter activity and alter the concentration of certain medicines in circulation.

The relationship between transporter genotype and medication response can be particularly relevant for drugs with a narrow therapeutic range. If reduced transporter activity increases circulating drug concentration, the possibility of dose-related adverse effects may increase. Conversely, increased transport into a tissue may alter local drug exposure and influence pharmacological activity. The final effect depends on the drug, transporter, tissue and other pathways involved in its disposition. Transporter genomics also has relevance to the blood-brain barrier. Specialized endothelial cells contain transport proteins that restrict the entry of many compounds into the central nervous system. Efflux transporters can remove substances from brain tissue and return them to the circulation. Genetic differences in these proteins may influence the amount of a medicine reaching neural tissues, although several physiological and biochemical factors contribute to this process.

Renal transporters play an important role in drug elimination. After a medicine reaches the kidneys, transporter proteins in renal tubular cells can facilitate secretion or reabsorption. Variants affecting these proteins may change the rate at which certain compounds leave the bloodstream. Kidney function itself

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also has a major influence, so transporter genotype should be considered alongside clinical measures of renal status. Transporter-mediated drug interactions can occur when one compound blocks or modifies the activity of a transporter used by another medicine. Such interactions may increase or decrease drug exposure. Genetic variation can further modify the magnitude of these effects. A person carrying a transporter variant with reduced activity may respond differently to an interacting medicine than someone with normal transporter function.

Transporter genomics uses methods such as targeted genotyping, polymerase chain reaction assays, microarray analysis and

Deoxyribonucleic Acid (DNA) sequencing. These approaches can identify single-nucleotide variants, insertions, deletions and other genetic differences. Computational tools can then relate detected variants to known functional information and pharmacogenomic recommendations. Gene expression adds another dimension to transporter activity. A genetic variant may affect how much transporter Ribonucleic Acid (RNA) is produced rather than changing the protein sequence. Hormones, inflammatory signals, diet, disease conditions and other medicines can also alter transporter expression. Consequently, transporter genotype is one component of a larger network that determines drug disposition.