

Editorial Note on Pharmacogenomics

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EDITORIAL

The study of the function of the genome in drug response is known as pharmacogenomics. The combination of pharmacology and genomics is reflected in the name (pharmaco- + genomics). Pharmacogenomics studies how a person's genetic makeup influences their drug response. It examines the impact of acquired and inherited genetic variation on drug response in patients by linking gene expression or single-nucleotide polymorphisms to pharmacokinetics (drug absorption, delivery, metabolism, and elimination) and pharmacodynamics (effects mediated by a drug's biological targets).

The terms pharmacogenomics and pharmacogenetics are often used interchangeably. While both words refer to drug response influenced by genetic factors, pharmacogenetics focuses on single drug-gene interactions, while pharmacogenomics takes a more genome-wide approach, combining genomics and epigenetics when addressing the impact of multiple genes on drug response. Pharmacogenomics seeks to establish rational methods for optimizing drug therapy based on a patient's genotype in order to achieve optimum efficacy while minimizing side effects. It is hoped that by using pharmacogenomics, prescription drug therapies would be able to depart from the "one-dose-fits-all" strategy. It also aims to eradicate the trial-and-error method of prescribing by encouraging doctors to understand their patients' genes, how these genes work, and how this may influence the effectiveness of their current or potential treatments (and where applicable, provide an explanation for the failure of past treatments). Sequencing offers a wealth of additional information, including the identification of mutations that cause the synthesized protein to be terminated prematurely.

Several genes have been identified as being primarily responsible for differences in drug metabolism and reaction. For the sake of brevity, this article will concentrate on the genes Cytochrome P450s, VKORC1, and TPMT, which are more generally known and used clinically. The Cytochrome P450 (CYP) enzymes are the most common drug-metabolizing enzymes (DME). These enzymes modify xenobiotics, such as medications, by adding reactive or polar groups. Omura and Sato coined the term Cytochrome P450 in 1962 to describe a membrane-bound, heme-containing protein with a 450 nm spectral peak when complexed with carbon monoxide.

There are 57 genes in the human CYP family, divided into 18 families and 44 subfamilies. On the basis of amino acid sequence similarity, CYP proteins are easily classified into these families and subfamilies. Enzymes that share 35-40% identity are assigned to the same family by an Arabic numeral, whereas those that share 55-70% identity are assigned to a specific subfamily by a letter. Family 2, subfamily D, and gene number 6 are all referred to as CYP2D6. CYP2D6, CYP2C19, CYP2C9, CYP3A4, and CYP3A5 are the most widely tested CYPs in clinical settings. About 70-90 percent of commercially available prescription medications are metabolized by these genes. A list of some of the drugs that use these pathways can be found in the table below. One possible function for pharmacogenomics is to reduce the incidence of polypharmacy. Patients would not need to take several drugs to treat the same disease if they receive personalized drug therapies, according to the theory. They may be able to reduce the incidence of ADRs, increase treatment outcomes, and save money by avoiding the purchase of extraneous medications.

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