Human beings vary in their responses to the drugs prescribed to them. These variations can be due to differences in their age, gender, weight, health status, diet, concurrent therapy and genetic make-up. Recently, it has been estimated that 85% of a patient’s response to drugs is due to the genetic make-up of the individual (Snedden, 1999). Genetic science is giving us many insights into why our drug responses vary, and this increased understanding is opening up opportunities to tailor drugs to common genetic profiles.

The objective of this article is to inform educators about pharmacogenomics, its applications, as well as its associated ethical and social issues. The article also suggests a suitable teaching strategy for presenting the ethics of pharmacogenomics in the classroom.

Theoretical Basis of Pharmacogenomics

Adverse Drug Reactions

An adverse drug reaction is a negative and unintended response to a medicine given in its normal dose (Merck & Co., 2003). Adverse reactions range in seriousness from headaches and nausea to life-threatening complications and sometimes death. Studies have shown that 2.2 million Americans suffered from adverse drug reactions in 1994. Of this number, 106,000 died before they could be discharged from hospitals (Lazarou et al., 1998). This makes adverse drug reactions the fifth leading cause of death in the U.S.A. (Mancinelli et al., 2000).

From Pharmacogenetics to Pharmacogenomics

The discipline of pharmacogenomics evolves from the discipline of pharmacogenetics. Both disciplines deal with the genetic basis underlying variable drug responses among individuals. The term “pharmacogenetics” was coined in 1959 to refer to the study of the variations in individual genes suspected of affecting drug responses. The term “pharmacogenomics” was coined recently to refer to the study of the variations in all the genes in the genome that determine variable drug responses among individuals (Mancinelli et al., 2000; Roden & George Jr., 2002).

Another difference between them is that pharmacogenetics is not concerned with the development of drugs whereas pharmacogenomics is.

Alcohol sensitivity is a pharmacogenetic trait that has been recognized for a long time (Snedden, 1999). In ethanol metabolism, ethanol is first oxidized to acetaldehyde by the enzyme alcohol dehydrogenase, and acetaldehyde is further oxidized to acetate by the enzyme aldehyde dehydrogenase. Many of the adverse effects of ethanol on the body system are attributed to acetaldehyde. Asians are more susceptible to alcohol poisoning than Caucasians because genetic variations make most Asians exhibit higher alcohol dehydrogenase activities but much lower aldehyde dehydrogenase activities as compared to Caucasians.

Another well-known pharmacogenetic trait is the hereditary variation of glucose-6-phosphate dehydrogenase in red blood cells (Mancinelli et al., 2000). It was observed during World War II that drug-induced hemolysis was much more prevalent among African-American soldiers than Caucasian-American soldiers who had been given the anti-malarial drug primaquine. It was later found that this effect was due to a genetic deficiency of glucose-6-phosphate dehydrogenase in the African-American males.

Goal of Pharmacogenomics

The goal of pharmacogenomics is to customize drugs for defined sub-populations of patients such that the “right medicine” is administered at the “right dose” to the “right person”. This means that the current “one-size-fits-all” approach of developing a medicine for a particular disease and the “trial-and-error” method of prescribing medicines will be replaced by the development of highly specific drugs and a “tailor-made” prescription based on the genetic make-up (pharmacogenomic profile) of individuals, respectively.

Pharmacogenomics research includes studying the genes and proteins that play essential roles in drug responses. Each drug, when administered into the human body, interacts with various types of proteins such as transport