With the increasing accessibility and affordability of sequencing our genomes, it becomes very relevant to ask questions about how this information can be applied to improve human health. Some might even say we have an ethical duty to invest in exploring this possibility. One emerging application of this research is in the area of pharmacogenomics, which Dr. Amalia M. Issa, Assistant Professor and Clinical Ethicist at Southern Illinois University School of Medicine, defines as the process of “identifying candidate genes and polymorphisms, correlating these polymorphisms with possible therapies, predicting drug response and clinical outcomes, reducing adverse events and selection and selecting dosing of therapeutic drugs on the basis of genotype” (Issa, 2002, p. 1). While this appears to be a novel and practical concept, it is important to think critically about the rhetoric surrounding the prospect of “saving the world” that the scientists seeking funding can purport. There are drawbacks and concerns surrounding pharmacogenomics, including a possible threat to the equal access to pharmaceuticals due to market supply and demand as well as insurance coverage. Some biological anthropologists, like Jonathan Marks, fear it is a means of reinstitutionalizing racism. Nonetheless, pharmacogenomics is an emerging field that pushes us to ask urgent questions regarding how, and in what way, we can use genetics to help improve pharmaceutical treatments.

Pharmacogenomics concerns the variations in the genes that produce enzymes, and how these differences affect drug metabolism in the body. Pharmacogenomics is commonly used interchangeably with another term: “pharmacogenetics” which applies more narrowly to the connection between drug response and single-nucleotide polymorphisms (SNP’s). SNP’s are variations in DNA at a single base that are found in at least 1% of the population (Debnath, 2009). Mousumi Debnath continues to explain that pharmacogenomics is “the whole genome application of pharmacogenetics, which examines the single gene interactions with drugs” (Debnath, 2009, p. 3). The overall goal of both disciplines is to “develop rational means to optimize drug therapy, with respect to the patients’ genotype, to ensure maximum efficacy with minimal adverse effects” (Debnath, 2009, p. 3). One diagnostic tool that is being used to sequence genomes is called a DNA microarray. Debnath provides a detailed description of this process:

“In a typical application, high-density nucleic acid samples, usually cDNAs or oligonucleotides, are delivered (or printed) by a robotic system onto very small, discrete areas of coated substrates (or chips) usually microscopic glass slides or membrane filters, and then immobilized to the substrate. The resulting microarray is then hybridized with a complex mixture of fluorescently labeled nucleic acids (probe) derived from a desired source. Following hybridization, the fluorescent markers are detected using high-resolution laser scanner. A pattern of gene expression is obtained by analyzing the signal emitted from each spot with digital imaging software.

In 2009, one microarray could screen 100,000 single nucleotide polymorphisms in a patient’s genome in a matter of hours (Debnath, 2009, p. 4) and this technology is only increasing in speed and efficacy. Therefore the possibility of...
having this technology in your doctor’s office is becoming more and more likely. Already, DNA microarrays have been responsible for revealing numerous associations between specific gene loci and complex diseases, such as breast cancer, type II diabetes, coronary artery disease, asthma, and bipolar disorder (Wiseman, 2009). This is why there is a sense of urgency in addressing the regulation and application of pharmacogenomic under- standings.

Figure 1. Scientists and scholars today are debating the medical benefits and the potential social risks of pharmacogenomics.

In addition to predicting a patient’s response to drugs, there are many more theorized benefits to pharmacogenomic research, such as: the ability to develop “customized” prescriptions, to screen and monitor certain diseases, to develop the ability to develop “customized” prescriptions, pharmacogenomics. In this case, this is accomplished by extension, genetics) that ground a wide array of pharmaceuticals to patients. This is due to the theory that during the process of profiling, some groups might be excluded or face unfair representation. In one Alzheimer’s study, research subjects were selected based on their apolipoprotein E genotype because they might be less likely to respond to the treatment drug, tacrine. For those who were able to participate, there was a reduced risk of nursing-home placement (Issa, 2002). This example serves to show that as soon as genotyping becomes an inclusion or exclusion criteria, the opportunity to benefit for clinical trials might be allocated in a more concentrated manner.

Historically, sub-groups such as women, the elderly, and children have been underrepresented in clinical trials (Issa, 2002). A similar concern applies to trials using pharmacogenomic-