In toxic tort litigation, a person is often claiming to have developed cancer or some other serious disease due to having been exposed to a particular substance. Determining if the exposure to the substance was the cause of the disease is often a central focus of the litigation. New technical abilities to determine genetic sequences combined with traditional epidemiological methods offers the promise of improved tools to determine whether or not a person’s disease was caused by or aggravated by exposure to a particular chemical or other substance. Toxicogenomics is the area of research that combines evaluation of gene expression and toxicology to investigate the interaction between genes and environmental stressor as it relates to disease causation. As the field advances, toxicogenomics may play an increasing role in toxic tort litigation.

The core of toxicogenomics is the ability to examine the human genome. A genome is the full complement of genes from an organism determined at the time of conception by the combination of maternal and paternal DNA. The human genome consists of approximately 3 billion base pairs of deoxyribonucleic acid. There is an approximately 0.1 percent variability in the DNA sequences between individuals. The identification of the variability of gene expression between individuals after exposure to various toxins serves as the basis of toxicogenomics.

One tool used in toxicogenomics is the use of microarrays to determine which genes are activated (expressed) after exposure to various chemicals. In this technology, thousands of gene copies can be placed on small glass chips to see if, after a subject is exposed to a substance, the products of gene expression from any of the tested genes are made. Patterns of gene expression can be compiled and compared in order to identify possible toxins. Another toxicogenomic tool is the seeking of genetic abnormalities after toxin exposures as possible biomarkers demonstrating exposure to a specific toxin.

The third tool of toxicogenomics is the linkage of genetic information with epidemiological techniques in the field of molecular epidemiology to seek genetic classes of increased susceptibility.

In toxic tort litigation, the plaintiff usually attempts to prove causation of a disease due to a toxin exposure while the defense counters by introducing evidence of alternative causes for the disease other than the alleged toxin exposure. The plaintiff must show both general and specific causation. General causation is a showing that exposure to the substance can cause the disease from which the plaintiff is suffering. After general causation has been proved, the plaintiff must prove specific causation by demonstrating that the particular exposure experienced by the plaintiff caused the plaintiff’s disease. Currently the issue of general causation is elucidated by epidemiological studies showing elevated rates...
of disease in exposed versus non-exposed groups. Proof of specific causation is usually made through expert testimony concerning the exposure and the likelihood that that exposure caused the disease rather than some other exposure or circumstance.

Attempts to establish general causation may be made by introducing microarray findings that certain gene expression patterns demonstrate that exposure to a particular substance can cause a particular disease. The toxic tort litigator must remember that gene expression does not necessarily indicate that the expressed gene or genes causes a particular disease. However, the combination of toxicogenomics with molecular epidemiology may advance scientific consensus concerning general disease causation.

The presence of biomarkers may be introduced in an attempt to prove specific causation. Again, the presence of a biomarkers alone without conventional methodologies used by qualified medical and exposure experts may not be sufficient to prove specific causation.

Toxicogenomics may be used in an attempt to establish exposure to a particular substance. For example, DNA damage has been detected in benzene exposed workers. However, the findings of the DNA damage measures have not found widespread acceptance as biomarkers of exposure or effect with benzene mediated disease.

A hereditary difference within a single gene which occurs in more than one percent of the population is referred to as genetic polymorphism. These genetic variations can lead to increased risks of disease in exposure groups. For example, approximately 50 percent of the Caucasian population has a gene deletion for the enzyme glutathione S-tranferase M1. This gene deletion causes an increased risk of lung cancer. Toxicogenomics combined with molecular epidemiology studies may elucidate genetic types at increased risk of disease due to various substance exposures. The ability to indentify persons at increased risk of disease after substance exposures due to their genetic makeup has implication in litigation involving claims concerning fear of cancer, medical monitoring, and failure to warn.

Toxicogenomics and the related discipline of molecular epidemiology hold great promise in identifying causes of disease in various exposure groups. These new tools may find increasing roles in toxic tort litigation. It is the responsibility of the toxic tort litigator to know when the right tool is being used for the right job.


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