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Genetics: Linking Health and the Environment

By Glen Andersen

Each year, researchers discover a greater number of genes that influence an individual’s susceptibility to disease. Investigations have also shown that in many cases, it is not genes alone that determine whether a person gets a disease; environmental factors are a key part of the equation. These discoveries will enable toxicologists and risk specialists to more accurately predict the levels at which pollutants will harm people, which will in turn help policymakers develop better public health protection strategies.

INTRODUCTION

Why do some two-pack-a-day smokers live to be octogenarians while others die of lung cancer at age 50? How is it that common household allergens that pose little concern to most people cause life-threatening asthma attacks in others? The answer lies in our genes—an individual’s susceptibility to toxic substances is not merely determined by the amount and duration of exposure, but is inextricably linked to their genetic makeup as well. Genetic variations can increase or decrease an individual’s resistance to illnesses.

Given the vast genetic variation between individuals, choosing allowable levels of exposure can be difficult. The problem lies not just in the complex science and the multitude of uncertainties that are related to setting health standards, but also the fact that pollutant levels that harm one individual will have no effect on another. Because of these uncertainties, standards are often set with wide safety margins to protect sensitive individuals. Genetic research may soon help take much of this uncertainty out of environmental standard setting by helping to

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identify susceptible individuals and subgroups.

In humans, the complete genetic code is contained in the 23 pairs of chromosomes that are present in each individual cell (excluding red blood cells). These chromosomes—which are composed of tightly coiled DNA molecules—contain all the information required to create a complete individual. Within DNA are the individual genes that determine which proteins to create in order to construct the cells that form all the different body parts. It is estimated that more than 100,000 genes determine the complete makeup of an individual, providing enough variation so that no two humans are exactly alike². These genes determine traits such as hair color, eye color, height and susceptibility or resistance to environmental exposures.

Genetic variability presents opportunities and challenges to scientists and policymakers alike, who must create environmental standards to protect public health in spite of the uncertainty created by individual genetic variance. This is particularly difficult when the only available data is derived from animal experiments? To make up for uncertainty, a “safety factor” is introduced to ensure that what we don’t know about genetic susceptibility won’t hurt us. This safety factor addresses two major problems that exist in assessing the risk of exposure to the thousands of chemicals that we encounter in our environment.

The first problem is the lack of human data. Since animals—usually rodents—are normally used to assess the toxicity of chemicals, differences between human and rodent biology must be accounted for; this is accomplished by dividing by ten the maximum amount of a chemical that can be given to the test animals without adverse effects. A second difficulty arises when accounting for susceptible individuals. To address this problem, another factor of 10 is used, providing a total reduction that is 100 times lower than the level determined not to harm rodents. The size of the safety factor may shrink as more is learned about the human toxicity of a chemical.

Some argue that safety factors are overly cautious, claiming that rodent testing is not conducted in a manner that is relevant to the low levels of chemical exposure that humans are likely to encounter. Many toxicologists however, say that while significant uncertainty is inherent in many risk calculations, the current method is the best available at the present time. Wide safety factors are considered necessary unless research is available to reduce uncertainty.

PREDICTING ENVIRONMENTAL SUSCEPTIBILITY: GENETIC TESTING

Determining genetic susceptibility to environmental agents is presently in its infancy; while scientists suspect that heredity is a risk factor for a large number of cancers, very few of the genes that confer risk have been identified, although progress has been made in discovering susceptibility genes for breast and colon cancer³. These and future discoveries will likely change

the way risk is assessed and lend more accuracy to standard setting.

The Environmental Genome Project (not connected with the Human Genome Project), which is being undertaken by the National Institute of Environmental Health Sciences, is aimed at identifying genes that determine susceptibility to environmental diseases. Research in this area will reveal whether a person has sensitivities to specific chemicals and pharmaceuticals. Since current research is based on the average person, and genetics tells us there is no “average” person, gene identification and subsequent testing will help protect sensitive populations at potentially lower regulatory costs. Even with today’s most advanced technologies, it is difficult to know—given the tremendous uncertainties related to standard making—whether we are under- or over-regulating in the quest to protect public health. Genetic research will aid in calculating the number of people who benefit from regulatory action.

In the future, genetics will provide a basis for sound science and become a prominent aspect of the debate over regulating toxicants like pesticides and mercury. Currently, there is a great deal of concern by industries and some states that regulatory agencies are over-regulating by not basing decisions on good science. This fear is reflected by resolutions passed in 1998 and 1999 by seven states: Colorado, Georgia, Kansas, Missouri, Michigan, Pennsylvania and Wyoming. The resolution urges the EPA—when considering new pesticide tolerances under the Food Quality Protection Act—to use sound science, make no decisions unless adequate data is available, and avoid actions that will have adverse economic effects.

Research on gene/environment interaction will likely have an enormous impact on health care as well. Testing for genes that indicate environmental susceptibility will be particularly beneficial if known prevention techniques and therapies exist. Knowing a person’s genetic identity will aid in taking precautions to avoid risk factors for contracting disease, and may help in reducing

Gulf War Syndrome - A Genetic Connection

Recent research indicates that genetic susceptibility played an important role in Gulf War illnesses. A study conducted by the University of Texas Southwestern Medical Center in Dallas demonstrates that veterans who suffer from Gulf War-related illnesses have lower-than-normal levels of an important chemical in the area of the brain that controls movement, memory, and emotion. This chemical deficit—a likely indicator of brain damage—is thought to be caused by genetic susceptibility to chemicals that were used in the war, such as permethrin (a liquid insecticide), the insect repellent DEET, pyridostigmine bromide (a chemical in anti-nerve gas pills) and possibly nerve gas. A related study provided evidence that people with a genetic variation that caused them to produce high amounts of an enzyme that destroys toxic chemicals did not get sick after exposure to these chemicals during operation Desert Storm, while those who lacked this gene did¹.

the occurrence of diseases like breast cancer, colon cancer, and Parkinson's.

Diseases that are likely to have an environmental and genetic component:

- Cancer (lung, bladder, breast, prostate cancer)
- Pulmonary Diseases (asthma, cystic fibrosis)
- Neurodegenerative Disorders (Alzheimer's, Parkinson's)
- Developmental Disorders (reduced intelligence, attention deficit hyperactivity disorder)
- Birth Defects
- Reproductive Function (fertility, fibroids, precocious puberty, endometriosis)
- Autoimmune Diseases (lupus, multiple sclerosis)

GENETIC INFORMATION AND INSURANCE

Most state genetics legislation supports the view that genetic information is clearly different from traditional medical information. Genetic information has the potential to identify disease risk to a degree that reaches far beyond previous methods of acquiring medical information and is more comprehensive than information that is not derived through genetic testing. There is concern that the availability of gene markers for hundreds of diseases may provide employers and insurance companies with an immense amount of potentially discriminatory information.

STATE ACTIVITY

Although states have seen a flurry of legislative activity relating to genetic discrimination and genetic privacy in 1999 and 2000 (more than 100 bills were introduced each year), none specifically mention environment as an integral component of the many diseases for which genetics is a risk factor. Some of the legislation does however mention genetic information as it relates to "genetic or chromosomal damage due to environmental factors," within the definition of "genetic test."

Typical statutes are similar to Oregon's §746.135, which prohibits policies that cover hospital or medical expenses from rejecting, canceling, limiting coverage or raising rates based on the results of genetic testing. Others such as New York's Civil Rights 79-1 focus on confidentiality, requiring that all results be confidential and that authorization be required for disclosure.

FEDERAL ACTIVITY

Genetic non-discrimination legislation has been introduced at the federal level. This includes H.R. 293, the Genetic Information Health Insurance Nondiscrimination Act of 1999, which would amend the Public Health Service and the Employee Retirement Income Security Act of 1974. This bill prohibits health insurers and group health plans from discriminating against

individuals based on genetic information. The department of Health and Human has posted proposed rules governing genetics information privacy, and should release the final rules by the year 2001.

NOTES

1. Robert W. Haley, Scott Billecke, and Bert N. La Du, "Association of low PON1 type Q (Type A) arylesterase activity with neurologic symptom complexes in gulf war veterans." *Toxicology and Applied Pharmacology* 157, no. 3 (June 15, 1999): 227-33.
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3. Y. Miki, J. Swensen, et al., "A strong candidate for the breast and ovarian cancer susceptibility gene BRCA1" *Science* 266 (1994): 66-71