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GENOMICS & ETHNICITY: USING A TOOL IN THE U.S. ENVIRONMENTAL PROTECTION AGENCY'S ENVIRONMENTAL JUSTICE TOOLKIT

DAVID L. MCMURRAY, JR.*

INTRODUCTION

While a relatively new issue, environmental justice has had a major impact on environmental law. The environmental justice movement encourages the development of environmental laws that fairly treat persons regardless of race or income.[1] The movement is the result of disproportionate treatment of minority groups, both real and perceived.[2] Results of the movement include Environmental Protection Agency (EPA) investigations, Executive Orders, environmental justice infrastructure within the EPA, and most recently, the EPA's development of an Environmental Justice Toolkit.[3] This Toolkit relies on environmental justice assessors to consider Environmental Indicators, Health Indicators, Social Indicators, and Economic Indicators in applying a four-phase framework to determine whether an environmental injustice exists and, if so, its extent.[4]

New developments in genomics may help environmental justice assessors apply this Toolkit. For many decades, scientists have found that a majority of persons of different ethnicities may show a greater or lesser reaction to chemicals or drugs than other persons. These findings came to a major head when chemical trials of the drug BiDil encouraged the Food and Drug Administration to approve it specifically for African American patients in June 2005.[5] With the ability to link

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1. OFFICE OF ENVTL. JUSTICE, U.S. ENVTL. PROT. AGENCY, TOOLKIT FOR ASSESSING POTENTIAL ALLEGATIONS OF ENVIRONMENTAL INJUSTICE i (2004), *available at* http://www.epa.gov/compliance/resources/policies/ej/ej-toolkit.pdf [hereinafter TOOLKIT].

2. *Id.*

3. *Id.* at ii; 4 FRANK P. GRAD, TREATISE ON ENVIRONMENTAL LAW § 9.10 (2006).

4. TOOLKIT, *supra* note 1, at 18-20, 26.

5. Michelle Meadows, *FDA Approves Heart Drug for Black Patients,* FDA CONSUMER, Sept.-Oct. 2005, at 8, 8.

ethnicity to drug susceptibility comes the possibility of linking ethnicity to toxin and xenobiotic susceptibility.

This article postulates that advances in the genomics fields of pharmacogenomics and toxicogenomics concerning ethnicity will provide the EPA with a new and powerful tool to protect minority populations from environmental injustices. Part I explores the history of the environmental justice movement, the EPA's stake in this movement, and the EPA's Environmental Justice Toolkit. Part II introduces the reader to the field of genomics, specifically to pharmacogenomics and toxicogenomics. This part also discusses the new work being done in these fields. Part III discusses how pharmacogenomics provides an analogy for the impact that ethnic advances in toxicogenomics can have. This part also applies those advances to the Environmental Justice Toolkit and discusses ethical issues that may arise. Finally, Part IV concludes by noting that although the potential for ethnicity-related toxicogenomics developments to impact the EPA's assessment of environmental justice claims may be small, this victory could signal a new alliance between toxicogenomic scientists and those populations disproportionately affected by environmental injustices.

I. ENVIRONMENTAL JUSTICE AND THE EPA'S ENVIRONMENTAL JUSTICE TOOLKIT

In the early days, environmentalists ignored or did not perceive the existence of environmental justice issues.[6] However, as the environmental movement has gained steam and environmental injustices have come to light, the environmental justice movement has moved forward as an issue of concern to most, if not all environmentalists.[7] This development is reflected in lawsuits brought by those affected by environmental injustice, the EPA's research into the issue and decision to set up an Office of Environmental Equity, President Clinton's decision to issue an Executive Order dealing with the subject, and the subject of this article, the EPA's "Toolkit for Assessing Potential Allegations of Environmental Injustice." In this part, these developments are discussed, with particular focus on the Environmental Justice Toolkit.

A. Environmental Justice: A Brief History and Discussion

The United States Environmental Protection Agency (EPA) defines "environmental justice" as "the fair treatment of people of all races, cultures, incomes, and educational levels with respect to the development, implementation, and enforcement of environmental laws, regulations, and policies."[8] According to the EPA, "[f]air treatment implies that no population of people should be forced to

6. EDWARDO LAO RHODES, ENVIRONMENTAL JUSTICE IN AMERICA: A NEW PARADIGM 30 (2003).

7. *Id.* at 43-44, 53-63.

8. GRAD, *supra* note 3, § 9.10[1][a] (citing U.S. ENVTL. PROT. AGENCY, THE ENVIRONMENTAL PROTECTION AGENCY'S ENVIRONMENTAL JUSTICE STRATEGY (1995), *available at* http://www.epa.gov/compliance/resources/policies/ej/ej_strategy_1995.pdf).

shoulder a disproportionate share of the negative environmental impacts of pollution or environmental hazards due to lack of political or economic strength."[9]

Until recently, the unequal distribution of environmental hazards and benefits of environmental regulation had no place in the agendas of mainstream environmental organizations.[10] Instead, the environmental movement originally focused on the objects of the environment—animals or land—without considering the varying impact of environmental law and policy upon humankind.[11] In addition, mainstream environmentalists in environmental groups and the government, consisted largely of middle- and upper-class Caucasians, while minorities and the poor initially showed little interest in environmental issues.[12] Thus, it comes as no surprise that issues of race, ethnicity, and class were not among the original concerns of mainstream environmentalists.

Today, however, environmental justice has surfaced as an important theme of environmental policy discussion.[13] This surfacing traces its history back as far as 1967, a year that saw "one of the earliest public outcries for environmental justice."[14] When an eight-year-old African American girl drowned in a garbage dump located near an elementary school in a predominately African American neighborhood, an escalated campus protest took place at Southern Texas University.[15] Other incidents in Houston, Texas followed, eventually leading to one of the first class action lawsuits challenging a siting decision as a violation of the Civil Rights Act, *Bean v. Southwestern Waste Management Corp.*,[16] which set the stage for other lawsuits challenging siting decisions.[17] In the early 1980s, protests became more unified and eventually established environmental justice as an issue of national concern, perpetuating studies on the issue and focusing national attention.[18]

B. The EPA's Role in Environmental Injustice Assessment: A Toolkit for Assessing Potential Allegations of Environmental Injustice

Although history has shown environmental justice being championed by some community organizations and individuals, both the legitimacy of environmental justice as a problem and the community's role in its resolution have been called

9. RHODES, *supra* note 6, at 19.

10. *Id.* at 30.

11. *Id.* at 30-31.

12. *Id.* at 31.

13. *Id.* at 43-44.

14. GRAD, *supra* note 3, § 9.10[1][b].

15. *Id.*

16. 482 F. Supp. 673 (S.D. Tex. 1979).

17. GRAD, *supra* note 3, § 9.10[1][b].

18. *Id.* (citing Robert Bullard, *Race and Environmental Justice in the United States*, 18 YALE J. INT'L L. 319 (1993)).

into question.[19] Yet, social, economic, health, and technological factors pushed environmental justice into the limelight as an important policy issue.[20] As a result, EPA Administrator William K. Reilly established the Environmental Equity Workshop in 1990 to study evidence showing that minority and low-income communities were disproportionately affected by environmental hazards.[21] The Workshop's report, published in 1992, concluded that minorities experience disproportionately greater exposure to environmental pollutants.[22] In the same year, the EPA created the Office of Environmental Equity to oversee environmental justice at the EPA.[23] Shortly thereafter, in 1994, the EPA created a three-tiered environmental justice infrastructure to (1) work with the Office of Environmental Equity in ensuring that environmental justice issues are considered; (2) cross media policy development and multi-media environmental justice project coordination; and (3) provide education and outreach on environmental justice information.[24]

Today, many groups, both within and outside of the EPA, consider and attempt to neutralize environmental justice issues.[25] While the aforementioned Office of Environmental Equity came first, in February of 1994 President Clinton issued the first general federal initiative to address concerns for environmental justice.[26] Executive Order No. 12898, entitled "Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations," requires all federal agencies to make environmental justice part of their mission by identifying and addressing, as appropriate, "disproportionately high and adverse human health or environmental effects of its programs, policies, and activities on minority populations and low-income populations."[27] According to a Presidential Memorandum released on the same day, the Executive Order is "intended to promote nondiscrimination in Federal programs substantially affecting human health and the environment" through public participation and access to information, to modify existing laws, such as the National Environmental Policy Act of 1969 (NEPA), to consider environmental justice issues and effects, to provide opportunities for minority and low-income communities to be heard on the issues, and to address mitigation measures.[28] In response, the EPA established an Inter-

19. RHODES, *supra* note 6, at 43-44.

20. *Id.* at 51-54.

21. GRAD, *supra* note 3, § 9.10[1][b]; *see* Julie A. Roque, *Environmental Equity: Reducing Risk for All Communities*, ENV'T, June 1993, at 25, 25-26.

22. U.S. ENVTL. PROT. AGENCY, ENVIRONMENTAL EQUITY: REDUCING RISK FOR ALL COMMUNITIES 12 (1992).

23. GRAD, *supra* note 3, § 9.10[1][b]; *see* Roque, *supra* note 21, at 27.

24. GRAD, *supra* note 3, § 9.10[1][b].

25. The Interagency Working Group on Environmental Justice, as well as each federal agency, was required to address environmental justice issues by the President. Exec. Order No. 12,898, 59 Fed. Reg. 7,629 (Feb. 16, 1994).

26. GRAD, *supra* note 3, § 9.10[4][c].

27. Exec. Order No. 12,898, 59 Fed. Reg. 7,629 (Feb. 16, 1994).

28. Memorandum on Environmental Justice, 30 WEEKLY COMP. PRES. DOC. 279 (Feb. 11, 1994).

Agency Working Group to implement the Order among federal agencies, developed an Environmental Justice Strategy and a plan to implement its goals, and set up a diverse Federal Advisory Committee to make recommendations on environmental justice issues.[29]

More recently, the EPA took steps toward fighting environmental injustice by developing a "Toolkit for Assessing Potential Allegations of Environmental Injustice."[30] The EPA developed the Toolkit to help its Environmental Justice Coordinators assess the environmental and human health concerns raised by communities and other stakeholders, as well as to promote national consistency in the way environmental justice concepts are "understood and addressed" by the Agency.[31] Using the Toolkit, Agency officials can conduct preliminary assessments of environmental justice allegations.[32] EPA Assessment Teams consider the following:

> Whether individuals, certain neighborhoods, or federally recognized tribes suffer disproportionately adverse health or environmental effects from pollution or other environmental hazards; [w]hether individuals, certain neighborhoods, or federally recognized tribes suffer disproportionate risks or exposure to environmental hazards, or suffer disproportionately from the effects of past under-enforcement of state or federal health or environmental laws; [w]hether individuals, certain neighborhoods, or federally recognized tribes have been denied an opportunity for meaningful involvement, as provided by law, in governmental decisionmaking relating to the distribution of environmental benefits or burdens. Such decisionmaking might involve permit processing and compliance activities.[33]

In accordance with these goals, the Toolkit presents a framework for an overall environmental justice assessment methodology to be applied on a case by case basis.[34] The framework contains four phases: Phase 1 – Problem Formulation; Phase 2 – Data Collection; Phase 3 – Assessment of the Potential for "Adverse"[35] Environmental and Human Health Effects; and Phase 4 – Assessment of the

29. GRAD, *supra* note 3, § 9.10[4][c][iii].

30. TOOLKIT, *supra* note 1, at ii. The Toolkit was created in 2004 and intended as a "living document," which may be revised periodically. *Id.* While it is not legally binding on the EPA, states, Indian tribes, or the regulated community, the Toolkit does provide a framework for understanding national policy on the subject of environmental justice. *Id.*

31. *Id.* at 2, 4.

32. *Id.* at 2. It is important to recognize that the Toolkit is not designed to assess allegations under Title VI of the Civil Rights Act of 1964. *Id.*

33. *Id.* at 4 (citing U.S. DEP'T OF JUSTICE, GUIDANCE CONCERNING ENVIRONMENTAL JUSTICE (1995), *available at* http://www.usdoj.gov/enrd/79648environmentaljusticestrategy.pdf).

34. *Id.* at 15.

35. An "adverse" effect or impact is defined as including "the entire compendium of 'significant' (as defined under the National Environmental Policy Act) individual or cumulative human health or environmental effects or impacts which may result from a proposed project or action." *Id.* at 16.

Potential for "Disproportionately High and Adverse"[36] Effects or Impacts.[37] These phases may be considered in the order that each situation demands.[38] However, the key consideration is "whether a particular community is likely to suffer from disproportionately greater environmental effects or impacts, regardless of its demographics."[39]

Noting that the concept of environmental justice means that all people should receive fair treatment, have equal protection, and have opportunities for involvement in decisions affecting the environment and community health, the Toolkit sets out a number of indicators that should be examined together to "provide a comprehensive picture of a community's economic, social, environmental, and health level status or well-being."[40] These "Environmental Justice Indicators" are divided into four categories that represent areas in which conditions occur that cause or intensify environmental justice issues: (1) Environmental Indicators; (2) Health Indicators; (3) Social Indicators; and (4) Economic Indicators.[41] Environmental Indicators help EPA Assessment Teams understand the potential sources of environmental stressors and the community's proximity thereto, the relative levels of stressors to which a community is being exposed and the ways exposure occurs, and the general physical environment in which exposure occurs.[42] Health Indicators help EPA Assessment Teams understand the general health of community residents and their ability to cope with environmental stressors.[43] These include existing health conditions, such as morbidity and health problems, which may indicate an exposure as well as resistance or sensitivity to pathogens and chemicals.[44] Health Indicators may also show whether a subset of a population has health sensitivities such as a greater susceptibility to toxic substances, or whether a subset of a population has had a higher cumulative exposure.[45] Social Indicators help EPA Assessment Teams understand the socio-demographic aspects of the community.[46] Some examples are

36. A "disproportionately high and adverse effect or impact" is defined as:

> [A]n adverse effect or impact that: (1) is predominately borne by any segment of the population, including, for example, a minority population and/or a low-income population; or (2) will be suffered by a minority population and/or low-income population and is appreciably more severe or greater in magnitude than the adverse effect or impact that will be suffered by a non-minority population and/or non-low-income population.

Id. at 16.

37. *Id.* at 18.

38. *Id.* at 19.

39. *Id.*

40. *Id.* at 25.

41. *Id.* at 27. The Toolkit notes that "a community's level of public participation and access to environmental information can diminish or augment environmental injustices." *Id.* at 28.

42. *Id.* at 28.

43. *Id.* at 29.

44. *Id.*

45. *Id.*

46. *Id.*

vulnerability indicators—indicators that show whether a certain subpopulation is more vulnerable to exposure because of limited access to certain amenities, such as hospitals, potable water, proper sewage, and public transportation, or because of personal behavior, such as smoking.[47] Other Social Indicators include community education levels and government encouragement thereof, as well as community participation levels.[48] Finally, EPA Assessment Teams consider Economic Indicators, which may reveal trends in a community's wealth and the community's dependence on the operation of a facility alleged to cause environmental injustice.[49] These indicators are incorporated into a four-phase assessment of the potential impacts of proposed actions and existing impacts, to make an overall determination of whether environmental injustice exists.[50]

As noted previously, environmental justice assessors use four phases to assess potential environmental injustice.[51] In phase one, the problem formulation phase, EPA Assessment Teams must consider the myriad of questions and issues that started the assessment.[52] Environmental justice indicators are important in this phase for evaluating who, what, where, when, and in what context the EPA is assessing the possible injustice.[53] For example, Environmental Indicators help determine the size and breadth of the problem and determine environmental vulnerability.[54] Social Indicators are important for assessing the reference community's demographics, knowledge, political awareness and power, and potential vulnerability to environmental stresses.[55] Finally, Economic Indicators are important for assessing the potential impact of environmental injustice based on reliance placed on the polluting industry for jobs and a community's overall economic well-being.[56] In phase two, the data collection phase, EPA Assessment Teams collect data on "(1) the environmental actions or entities (e.g., a facility) that create the environmental and health effects; and (2) the community of concern where these impacts will be manifested."[57] In part one of the data collection phase, EPA Assessment Teams collect source of stress data about Environmental Indicators, including the number of facilities and facility proximity.[58] Social Indicators, also collected at this stage, are useful to determine behavioral factors

47. *Id.* at 29-30.
48. *Id.* at 30.
49. *Id.*
50. *Id.* at 58.
51. *See supra* notes 34-39 and accompanying text.
52. TOOLKIT, *supra* note 1, at 58.
53. *Id.* at 20.
54. *Id.* at 31, 38-39.
55. *Id.* at 43-53.
56. *Id.* at 30, 53-57.
57. *Id.* at 63.
58. *Id.*

that affect community exposure.[59] In part two of the data collection phase, EPA Assessment Teams focus more on data collection concerning the Health, Social, and Economic Indicators of the community of concern and a reference community, which is used to compare the adverse effects to determine if there is a disproportionate effect on the community at issue.[60] The Toolkit encourages data collection that includes information on the existing health conditions of the communities, such as mortality and morbidity, and qualitative information such as diet and smoking.[61]

Phase three of the environmental assessment, "Assessment of the Potential for 'Adverse' Environmental and Human Health Effects or Impacts," uses the data collected during phase two of the assessment to determine "whether the stresses are likely to cause adverse environmental and human health/welfare impacts."[62] Environmental Indicators are important in this phase to determine whether the environment will suffer or has suffered an adverse impact from the proposed actions or existing situation.[63] Health Indicators are important in this phase of the analysis to determine whether the environmental stress exposure will be of a sufficient magnitude to cause adverse effects on a community's health or welfare.[64] By examining the potential effects of stressors, as well as a population's health status and its potential vulnerability to such stressors, EPA Assessment Teams can create a risk assessment of the existing or prospective situation.[65] Existing health conditions indicate whether a community might be more sensitive to stresses than other communities.[66] "People with poor or compromised health status, whether from exposure to environmental contaminants, genetics . . . , poor nutrition, obesity, smoking, or abuse of alcohol or drugs, can be less resistant to infections . . . and less capable of detoxifying contaminants absorbed into their systems . . . than people in better health."[67] A study of potential effects or impacts from stressors under consideration represents the prospective health risk assessment.[68] It would compare "likely contaminant exposure concentrations and intake with information on the toxicity of the contaminants."[69] Specifically, this assessment would,

> [C]ompare the available indicators of exposure to readily available information on the toxicity of the contaminants (e.g., from IRIS) . . . , identif[y] toxicity-weighted emissions that may be associated with

59. *Id.*
60. *Id.* at 64.
61. *Id.* at 64-65.
62. *Id.* at 66.
63. *Id.* at 66-67.
64. *Id.* at 68.
65. *Id.*
66. *Id.*
67. *Id.*
68. *Id.*
69. *Id.*

> significant risks . . . , [and compare] measures or estimates of the concentrations of some contaminants in environmental media . . . with chemical-specific environmental quality benchmarks . . . [or] risks to the community of concern . . . with risks to the reference community by comparing the indicators of exposure to the same contaminant for the two communities.[70]

Alternatively, when a stressor already exists, environmental justice indicators are less useful because these indicators do not reveal cause-and-effect relationships.[71] Thus, further examination would be needed to show that pollution-generating facilities are contributing to environmental stressors causing health effects.[72] Initially, retrospective analyses of possible environmental injustices can be based on whether or not the potential sources could have caused the effects.[73] This assessment requires the postulation of two questions: (1) "Could the population be exposed at high levels or levels exceeding toxicity benchmarks?" and (2) "Are the observed health effects in the community consistent with the effects that are known to be caused by the contaminants at issue?"[74] If both of these questions are answered affirmatively, the EPA Assessment Team will additionally assess evidence that particular sources of contaminants are causing adverse health effects in the community, including whether other sources are not causing or contributing to the effects, whether the effects began to occur when the facility began operation or began dumping toxins in the environment, and whether a stressor-response relationship can be demonstrated.[75]

The final phase, "Assessment of the Potential for 'Disproportionately High and Adverse' Effects or Impacts," is reached when the EPA Assessment Team ascertains that it cannot be certain that there are negligible risks of adverse effects on the community.[76] This final phase of analysis determines whether a "segment of the population, including a minority population" inequitably bears an adverse effect or impact, or whether a minority or low income population suffers more severely or in greater magnitude than a non-minority or low-income population.[77] To make this determination, the EPA Assessment Team collects data about environmental justice indicators and compares the community of concern with the reference community.[78] Finally, the EPA Assessment Team may conduct a quantitative

70. *Id.* at 68-69.
71. *Id.* at 69.
72. *Id.* at 70.
73. *Id.*
74. *Id.*
75. *Id.*
76. *Id.* at 70-71.
77. *Id.* at 71.
78. *Id.*

comparison to see if there are statistically significant differences between the two groups in one or more measures of risk.[79]

II. GENOMICS: A BRIEF INTRODUCTION AND DISCUSSION OF PHARMACOGENOMICS AND TOXICOGENOMICS

For thousands of years, humans have known that heredity and relative health are intertwined.[80] More recently, however, humans discovered the connection between genetics and phenotype[81] and we began to apply this knowledge less than 150 years ago.[82] Today, genetics, the study of single genes and their effects, and genomics, the study of the functions and interactions of the entire genome, have become critical to modern healthcare.[83] Two relatively new areas of study are having an increasingly large effect on our knowledge of human health: pharmacogenomics and toxicogenomics.[84]

A. Pharmacogenomics

Pharmacogenetics is the methodical investigation of the influence of variation in genes (polymorphisms) on interindividual variability in biological response to drugs.[85] The assemblage of pharmacogenetics and human genomics has resulted in pharmacogenomics, which means "the influence of DNA-sequence variation on the effect of a drug."[86] Today, the terms "pharmacogenomics" and "pharmacogenetics" are synonymous for all practical purposes.[87]

Inherited differences in response to a chemical were observed and documented as long ago as 1932.[88] However, pharmacogenetics and pharmacogenomics became viable fields of study in the 1950s when genetically

79. *Id.*

80. Alan E. Guttmacher & Francis S. Collins, *Genomic Medicine—A Primer*, *in* GENOMIC MEDICINE 3, 3 (Alan E. Guttmacher et al. eds., 2004) (citing 2 THE GENUINE WORKS OF HIPPOCRATES 338 (Francis Adams trans., 1886)).

81. Phenotype is defined as "[t]he clinical presentation or expression of a specific gene or genes, environmental factors, or both." Alan E. Guttmacher et al., *Glossary*, *in* GENOMIC MEDICINE, *supra* note 80, at xix, xxi.

82. Guttmacher & Collins, *supra* note 80, at 3.

83. *Id.* (citing Victor A. McKusick & Frank H. Ruddle, Editorial, *A New Discipline, A New Name, A New Journal*, 1 GENOMICS 1-2 (1987)).

84. For an exhaustive discussion of genetics, including applications of toxicogenomics to environmental law generally, see Jamie A. Grodsky, *Genetics and Environmental Law: Redefining Public Health*, 93 CAL. L. REV. 171 (2005).

85. Kurt Kesseler, *Pharmacogenetics: The Effect of Inherited Genetic Variation on Drug Disposition and Drug Response*, *in* 21 MOLECULAR BIOLOGY IN MEDICINAL CHEMISTRY 325, 325 (Theodor Dingermann et al. eds., 2004).

86. Richard Weinshilboum, *Inheritance and Drug Response*, *in* GENOMIC MEDICINE, *supra* note 80, at 41, 50.

87. William E. Evans & Howard L. McLeod, *Pharmacogenomics—Drug Disposition, Drug Targets, and Side Effects*, *in* GENOMIC MEDICINE, *supra* note 80, at 54, 54.

88. Kesseler, *supra* note 85, at 325.

determined variations were considered to be the cause of adverse drug effects.[89] In the early 1980s, researchers clarified the role of the molecular genetic basis of inherited differences in drug response when they began to clone and characterize genes.[90] Since then, researchers have identified more and more genetic variations that are associated with varying drug responses.[91] In fact, it is now estimated that genetic variability can explain between twenty and ninety-five percent of variability in drug disposition and effects.[92]

Of particular interest for purposes of this article, pharmacogenetics has shown that discrete populations such as minority groups may react differently to various drugs based on their genetic makeup.[93] Initially, studies showed that primaquine sensitivity, a drug-induced hemolytic anemia, occurred more frequently in African Americans than among Caucasian Americans.[94] In another early study, almost all persons of African and Native American descent were able to taste phenylthiourea, while almost a third of European descendants could not.[95] In both of these cases, the differences in these populations consisted of differences in the relative frequency of one gene, most likely as a result of the balance between advantages and disadvantages conveyed by that gene.[96]

Although differences in populations were noted as early as 1962,[97] "[s]tudies of the pharmacokinetics [the process by which a drug is absorbed, distributed, metabolized, and eliminated from the body], pharmacodynamics (concentration-response relationship), efficacy, and toxicity of drugs have traditionally been conducted in primarily Caucasian populations."[98] In response to this problem, in 1988, the National Institutes of Health (NIH) issued a policy statement that NIH-supported biomedical and bio-behavioral research projects using human subjects

89. Evans & McLeod, *supra* note 87, at 54 (citing Werner Kalow, *Familial Incidence of Low Pseudocholinesterase Level*, 2 LANCET 576 (1956); Paul E. Carson et al., *Enzymatic Deficiency in Primaquine-Sensitive Erythrocytes*, 124 SCI. 484 (1956); Hettie B. Hughes et al., *Metabolism of Isoniazid in Man as Related to the Occurrence of Peripheral Neuritis*, 70 AM. REV. TUBERCULOSIS 266 (1954); David A. Price Evans et al., *Genetic Control of Isoniazid Metabolism in Man*, 2 BRIT. MED. J. 485 (1960)).

90. Kesseler, *supra* note 85, at 325-26.

91. *Id.* at 326.

92. Evans & McLeod, *supra* note 87, at 54 (citing Werner Kalow et al., *Hypothesis: Comparisons of Inter- and Intra-Individual Variations Can Substitute for Twin Studies in Drug Research*, 8 PHARMACOGENETICS 283 (1998)).

93. *See generally* WERNER KALOW, PHARMACOGENETICS: HEREDITY AND THE RESPONSE TO DRUGS 206-22 (1962) (describing racial differences in the response to drugs).

94. *Id.* at 206.

95. *Id.* at 120, 206.

96. *Id.* at 206.

97. *See id.* at 206-22.

98. Julie A. Johnson, *Influence of Race or Ethnicity on Pharmacokinetics of Drugs*, 86 J. PHARMACEUTICAL. SCI. 1328, 1328 (1997).

should, in the absence of a compelling excuse, include women and minorities.[99] Inclusion of minorities has increased human understanding of the racial differences in the pharmacokinetics of drugs. A review of the literature on the subject reveals that "pharmacokinetic processes that are biologically or biochemically mediated have the potential to exhibit differences between racial or ethnic groups."[100] Unfortunately, as this review notes, information on differences in pharmacokinetics amongst racial and ethnic groups is only available for relatively few drugs.[101] The review postulates that the problem may be due to the low inclusion rate of ethnic minorities in clinical drug studies or, alternatively, because ethnic minorities are not evaluated as a separate group.[102] It does not help that "[t]he FDA does not require evaluation of kinetic, dynamic, efficacy, or toxicity data by racial or ethnic group."[103] Thus, while many, like the NIH, recognize the importance of kinetic and response differences amongst racial groups, others, such as the FDA, fail to respond to or capitalize on the potential of these data.

Work is being done in pharmacogenomics to remedy this problem. For example, a recent pharmacogenomic study conducted by Yee-How Say, a faculty member in the Department of Human Growth and Development at the University Putra Malaysia, sought to determine the genetic effect of a gene called angiotensinogen (AGT) on hypertension.[104] According to the study, genes account for about thirty percent of variation in blood pressure.[105] Three versions out of fifteen identified variants of the AGT gene have been reported to have a genetic association to hypertension.[106] Association studies concerning linkages for the variant studied in this case, M235T, have produced conflicting results—specifically, that there may or may not be linkage to various ethnic populations.[107] Noting this variation, the scientists conducting this study sought to determine effects in Malaysians, recognizing that "genetic diversity exists among different ethnic populations and . . . the association in one population could not be extrapolated to another population."[108] The study found an association between the

99. *Id.* (citing *Inclusion of Women in Study Populations*, NIH GUIDE FOR GRANTS AND CONT., Jan. 15, 1988, at 2, 2; *Inclusion of Minorities in Study Populations*, NIH GUIDE FOR GRANTS AND CONT., *supra*, at 2, 2-3). Such a rationale would only be compelling if it "shows that inclusion is inappropriate with respect to the health of subjects or the purpose of the research." *Id.*

100. *Id.* at 1332.

101. *Id.*

102. *Id.* at 1328.

103. *Id.*

104. Yee-How Say et al., *Angiotensinogen M235T Gene Variants and Its Association with Essential Hypertension and Plasma Renin Activity in Malaysian Subjects: A Case Control Study*, 5 BMC CARDIOVASCULAR DISORDERS (2005), *available at* http://bmc.ub.uni-potsdam.de/1471-2261-5-7/1471-2261-5-7.pdf.

105. *Id.*

106. *Id.*

107. *Id.*

108. *Id.*

M235T polymorphism of the AGT gene and hypertension.[109] Recognizing that the population studied was not homogenous, because the Malaysian population consists of different ethnic groups, the authors of the study concluded that "[t]he T235 variant is a risk factor or possibly a potential genetics marker for hypertension."[110]

Another study, which focused on the effects of the drug BiDil, linked the association between race and drug effect so closely that the FDA, for the first time ever, approved the drug for use specifically for African American patients in June 2005.[111] After two previous studies suggested a benefit of BiDil when taken with other heart medications for self-identified African American patients, but no evidence of benefits for Caucasian patients, Dr. Anne Taylor led a study known as the African American Heart Failure Trial, which examined 1,050 self-identified African American patients with severe heart failure who were already being treated with the best available therapy.[112] Dr. Taylor noted that genetics alone did not necessarily cause the effect, suggesting that different responses to the drug may also be associated with environmental, social, or lifestyle factors, or interactions among all those factors.[113] However, the study was conducted at more than 160 sites throughout the United States.[114] The BiDil treatment resulted in a forty-three percent reduction in death, a thirty-nine percent decrease in hospitalization due to heart failure, and a decrease in heart failure symptoms for African American patients.[115] One hypothesis as to why BiDil works is that heart failure among African Americans is associated with a deficit of nitric oxide, and BiDil may work by replenishing nitric oxide in the vascular tissue.[116] However, more research is necessary to determine precisely why the drug works.[117] Furthermore, a study of the drug's effect on other populations is warranted.[118] Regardless of the exact mechanism, the impact of this breakthrough is enormous, considering that African Americans between ages forty-five and sixty-four are 2.5 times more likely to die from heart failure than Caucasian Americans in the same age range.[119]

The BiDil study has raised turmoil in the medical community.[120] "Some experts see the approval of BiDil as a steppingstone to the goal of personalized

109. *Id.*

110. *Id.*

111. Meadows, *supra* note 5, at 8-9.

112. *Id.* at 9.

113. *Id.*

114. *Id.*

115. *Id.*

116. *Id.*

117. *Id.*

118. *Id.*

119. *Id.*

120. *Gray Area for New Heart Failure Drug: Although the FDA Approved BiDil for Blacks with Heart Failure, It May Work in Anyone*, HARV. HEART LETTER (Harvard Med. Sch., Boston, Mass.), Nov. 2005, at 1, 1 [hereinafter *Gray Area*].

medicine, with race serving as a stand-in for genetic variations that may point the way to safer, more effective medication use."[121] But, while BiDil may be the first sanctioned race-based medicine, it likely will not be the last because, although lots of drugs are extra effective for some people and not as effective for others, drugs like BiDil represent a potentially profitable niche.[122] However, this does not mean that BiDil is ineffective for any other patients, and it will likely be prescribed "off-label" to patients regardless of their ethnicity. Despite the fact that BiDil is recommended for African American patients, individuals who lack access to au courant drug and lifestyle therapies may be prescribed the components of BiDil.[123] And, according to Dr. Christopher Newton-Cheh, BiDil just might work as effectively for these individuals:

> Humans across the globe share most of their genes. The American "melting pot" has added to this commingling. "It is more likely that the genetic variation that modifies the response to a particular drug in one race or ethnic group is also present in others," says Dr. Christopher Newton-Cheh . . . . "If a drug does have a different average effect in individuals of a certain ancestry, that effect could just as well stem from differences in nutrition, environment, and access to health care as from genetics. We just don't know yet." . . . NitroMed[124] and the FDA used race as a stand-in for nitric oxide deficiency. That's partly because asking an individual his or her race is easy. And there aren't yet simple, inexpensive tests for nitric oxide production or genetic tests that might reveal one's response to BiDil.[125]

Thus, the BiDil controversy is not a simple black and white issue, in terms of race or clarity, but may represent a real breakthrough in personalized medication based on genomics. As one group of researchers aptly wrote,

> Ideally, the specific genes that determine a pharmacogenetic response should be tested without regard to genetic ancestry since the relevant traits usually exist in all populations . . . at different frequencies. In the absence of a specific test, choice of optimal drug treatment based on 'racial' assignment therefore may be justified.[126]

A lucrative new technology associated with pharmacogenomics—microarray chips, which allow medical personnel to scan the entire human genome for polymorphisms relevant to drug response and susceptibility—is being developed to help determine what genes are involved in drug response and whether those genes

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121. *Id.*

122. *Id.*

123. *Id.* at 2.

124. NitroMed is the patent holder for BiDil. *Id.* at 1.

125. *Id.* at 2.

126. Arno G. Motulsky & Ming Qi, *Pharmacogenetics, Pharmacogenomics and Ecogenetics*, 7 J. ZHEIJIANG U. SCI. B 169, 170 (2006).

are present.[127] Furthermore, as this technology develops, it may serve to determine the expression of genes in a target tissue to help researchers understand the mechanisms of drug action in a genomic context.[128] Chip technology will be useful in clarifying interindividual differences in drug response.[129] This technology will create profound changes in treatment, including, in the near future, genotyping to "help avert severe drug toxicity that is genetically determined but occurs only rarely."[130] In the alternative, the advancement of microarray chip technology and pharmacogenomics promises to help researchers design drugs "so that they are not subject to extreme variations that result from a few well defined polymorphisms."[131] For example, drug structures are being developed so that they do not interact with cytochrome-450 subtype CYP2D6, a gene subtype that plays a critical role in determining the response to several drugs, to prevent toxicity problems for poor metabolizers.[132] The study emphasizes that there are limitations to the promise of pharmacogenomics that must be overcome for the furtherance of science.[133] For example, "[t]he dynamic complexity of the human genome, involvement of multiple genes in drug responses, and racial differences in the prevalence of gene variants impede effective genome-wide scanning and progress towards practical clinical applications."[134] It is also notable that, while the importance of gene chips is unquestionable, broad use of gene chips in clinical practice will not occur overnight.[135] However, the value of pharmacogenomics is the knowledge of the principles underlying genetic variability and the ability to provide more individualized treatment.[136]

B. Toxicogenomics

The term toxicogenomics describes "the application of new genomics information and methods to toxicology studies, with the goal of advancing [human] understanding of the mechanism of action of compounds, the response of organisms to these exposures, and the ultimate application of contributing to the development of more accurate risk assessment models."[137] Records show that toxicology, as a discipline, dates back as far as the eleventh or twelfth century,

127. Wolfgang Sadée, *Pharmacogenetics*, 171 W. J. MED. 328, 331 (1999) (citing Robert F. Service, *Microchip Arrays Put DNA on the Spot*, 282 SCI. 396 (1998); Bob Sinclair, *Everything's Great When it Sits on a Chip: A Bright Future for DNA Arrays*, 13 SCIENTIST 18 (1999)).

128. *Id.*

129. *Id.*

130. *Id.*

131. *Id.*

132. *Id.* at 330, 331.

133. *Id.* at 332.

134. *Id.*

135. *Id.*

136. *Id.*

137. Hisham K. Hamadeh & Cynthia A. Afshari, *Preface, in* TOXICOGENOMICS: PRINCIPLES AND APPLICATIONS, at xix, xix (Hisham K. Hamadeh & Cynthia A. Afshari eds., 2004).

beginning with concern about the toxic effects of metals, such as lead and arsenic, among other things.[138] However, over the past several decades, toxicology has advanced to the point of using molecular methods to determine cellular responses to compound exposures.[139] With this advance and the recent completion of the mapping of the human and other genomes, the field of toxicology is ready to embrace the new discipline of toxicogenomics.[140] Toxicogenomics involves the application of "omic" technologies to toxicology and the replacement of burdensome testing methodology with technology that will improve understanding of the biological basis of toxicity of drugs and other environmental factors.[141] Toxicogenomics offers the promise that risk assessments will no longer need to rest on experimental studies performed at high doses in rodents, and that scientists will be able to gain knowledge currently lacking concerning the intrinsic toxicity for the majority of the high production chemicals introduced into the environment during the last half of the twentieth century.[142] This promise is one of a better understanding of the risk of toxicity of chemicals and environmental xenobiotics through combining recent advances in genomics and other "omics" such as proteomics and metabolomics to evaluate "(i) the diverse structure and properties of various chemicals; (ii) the relationship between the time of exposure, dose, and health outcomes; (iii) the influence of genetics and behavioral factors; and (iv) interactions between multiple components of biological systems in development of the toxic response."[143]

Although toxicogenomics is still in its fledgling stage, work is being done to rapidly advance it.[144] For example, the National Institute of Environmental Health Sciences (NIEHS) created the National Center for Toxicogenomics in 2000, and Center investigators are currently surveying the human genome and organ systems for toxic responses to drugs and environmental xenobiotics with the goal of determining "whether gene, protein or metabolite expression profiles or 'signatures' can serve as markers to predict toxicity."[145] Furthermore, investigators are in the process of developing a database of chemical effects on biological systems to predict toxicity and to better understand the underlying mechanisms.[146] This work combined with the work of others seeks to fulfill toxicogenomics's potential "(i) to identify sources of interindividual variability in response to drugs and environmental xenobiotics . . . ; (ii) to provide a database for the development

138. Kenneth Olden, *Foreword*, *in* TOXICOGENOMICS: PRINCIPLES AND APPLICATIONS, *supra* note 137, at xv, xv.

139. Hamadeh & Afshari, *supra* note 137, at xix.

140. *Id.*

141. Olden, *supra* note 138, at xv.

142. *Id.* at xv-xvi.

143. *Id.* at xvi.

144. *Id.* at xvii.

145. *Id.* at xvi.

146. *Id.* at xvi-xvii.

of high-throughput and low-cost platforms for screening substances for toxicity; and (iii) to improve the process of discovering new targets for drug action."[147]

Studies researching links between race and toxic susceptibility in the field of toxicogenomics are rare, if existent at all. However, researchers working through an NIEHS-created Toxicogenomics Research Consortium at a member university, the Massachusetts Institute of Technology (MIT), are beginning to understand some of these connections.[148] One of these researchers, John Essigmann, who was recently awarded Thailand's Princess Chulabhorn Gold Medal[149] for his "commitment to and sustained support for the advancement of science in developing countries, as well as for his selfless dedication to teaching and research," is working to understand differential gene expression in response to Aflatoxin.[150] Aflatoxin is a DNA damaging agent pervasive in the environment, particularly in developing countries.[151] Exposure may cause liver toxicity and cancer.[152] However, through continuous research, Essigmann hopes to understand "why some people are sensitive and some are resistant, how race and gender may affect why some people respond to therapeutic drugs and others do not, why infants and children are more susceptible than adults, and why hepatitis confers heightened sensitivity to aflatoxin effects."[153] While Essigmann may currently be alone in his research, development of ethnicity-specific drugs, a toxicogenomics database, and microarray chips are likely to spur other studies searching for the genetic effects of ethnicity on susceptibility to toxins and xenobiotics. Thus, although toxicogenomics is a young science, new developments such as Essigmann's are paving the way toward a developed field that will provide insights into the effects of ethnicity on toxic response.

III. DISCUSSION

As previously noted, the EPA's environmental justice program seeks the fair treatment of people of diverse ethnicities.[154] As part of that program, the EPA seeks to investigate environmental justice issues using its Environmental Justice Toolkit.[155] By analogy to the field of pharmacogenomics, toxicogenomics research

147. *Id.* at xvii.

148. *See* Mary Eubanks, *MIT Toxicogenomics Research Program,* 113 ENVTL. HEALTH PERSP. A 234 (2005).

149. The Princess Chulabhorn Gold Medal is given every five years to honor world-renowned individuals or organizations that have provided exceptional support for the activities of the Chulabhorn Research Institute, as well as support for the advancement of science in developing countries. Elizabeth A. Thomson, *Essigmann Receives Royal Honor*, MIT TECH TALK, Sept. 15, 2004, at 5, *available at* http://web.mit.edu/newsoffice/2004/techtalk49-2.pdf.

150. Eubanks, *supra* note 148, at A 234.

151. *See id.*

152. *Id.*

153. *Id.* at A 235.

154. RHODES, *supra* note 6, at 19.

155. TOOLKIT, *supra* note 1, at ii.

will likely offer major breakthroughs in human understanding of the impact of ethnicity on susceptibility to toxins and xenobiotics within the near future. These breakthroughs may potentially provide an advantage to those suffering from environmental injustice by adding or modifying tools in the EPA's Environmental Justice Toolkit. This part will first address how pharmacogenomics research and breakthroughs are applicable to toxicogenomics and genetic differences concerning ethnicity, before moving on to consider how these advances are applicable to the Environmental Justice Toolkit.

A. Applying Pharmacogenomics Breakthroughs to Toxicogenomics

Pharmacogenetics and pharmacogenomics provide, by analogy, a descriptive analysis of how and why genomics can influence our understanding of discrete population susceptibility to environmental toxins. Based on this analogy, within a short time research could show that toxins and xenobiotics have differing effects based on ethnicity. Breakthroughs in pharmacogenomics and ethnicity are applicable to toxicogenomics and ethnicity in three ways.

First, pharmacogenomics has already shown that genetically determined variations in response to stimuli exist based on ethnicity.[156] While there is no indication that a vast number of drugs will have differing effects based on ethnicity, there is evidence that some drugs will be shown to be extra effective for certain ethnicities.[157] The same must be true for toxicogenomics. Like pharmaceuticals, the effects of toxins and xenobiotics are chemical and based on metabolism.[158] At the root of the use of genomics in either of these fields is the study of genomic sources of metabolic differences.[159] Thus, if one field found differences in response to chemicals based on race, it is very likely that the other should as well. Studies seeking to determine these differences can help toxicogenomic scientists understand what genetic variations will lead to increased or decreased susceptibility and the process by which environmental toxins work. As previously noted, at least one scientist is already examining the xenobiotic aflatoxin.[160]

Second, pharmacogenomics can contribute to the general knowledge of how genomics affects chemical disposition by humans.[161] Toxicogenomics, as compared

156. *See supra* text accompanying notes 93-126.

157. *See supra* text accompanying notes 111-126.

158. *See* Olden, *supra* note 138, at xvi.

159. *See generally id.* at xvii; Evans & McLeod, *supra* note 87, at 54 (discussing the importance of considering genetics in drug response).

160. *See supra* text accompanying notes 148-150.

161. *See, e.g.*, Tilo Mandry, *Legal Implications of Pharmacogenomics Regarding Drug Trials, Drug Labeling, and Genetic Testing for Drug Prescription: An International Approach*, 59 FOOD & DRUG L.J. 519, 519 (2004).

to pharmacogenomics, is in its relative infancy.[162] Furthermore, toxicogenomics does not have the kind of financial backing that pharmacogenomics has.[163] Thus, it stands to reason that pharmacogenomics will develop more rapidly, and that toxicogenomics will benefit because both fields use "omic" technologies.[164] Furthermore, since pharmacogenomic scientists must study the effects of genetics on toxicity for purposes of understanding drug disposition, toxicogenomic scientists stand to benefit vicariously from advances in pharmacogenomics.[165]

Third, toxicogenomic scientists and studies on toxicity related to ethnicity stand to benefit from pharmacogenomic advances in microarray chip technology.[166] As previously noted, these chips allow medical personnel to scan a person's genome for polymorphisms relevant to drug response and susceptibility.[167] In addition, this technology could help scientists to understand gene expression in target tissues.[168] As Sadée's study, discussed *supra*, further notes, this technology will help scientists avert severe drug toxicity that is genetically determined.[169] Finally, microarray chips will help scientists to design drugs to avoid toxic sensitivity.[170] All of the above is equally applicable to toxicogenomics.[171] As previously mentioned, these two fields work on the same mechanisms, but vary with subject matter.[172] Accordingly, microarray chips should allow toxicogenomic scientists to scan genomes to search for ethnic differences in toxic susceptibility.[173] Microarray chips help toxicogenomic scientists to understand how gene expression affects chemical disposition in affected tissues.[174] Just as with pharmacogenomics, these chips could help toxicogenomic scientists avert severe toxic reactions that are

162. As previously noted, pharmacogenetics and pharmacogenomics began developing in the 1950s. Evans & McLeod, *supra* note 87, at 54. By contrast, toxicogenomics only began to gain speed over the past several decades. Hamadeh & Afshari, *supra* note 137, at xix.

163. Race-based prescription drugs like BiDil represent, "a very profitable niche." *Gray Area, supra* note 120, at 1. Thus, drug companies are likely to continue to invest money in pharmacogenomics to further develop this niche. In contrast, toxicogenomics seeks to identify the harmful effects of pollutants and environmental xenobiotics. *See* Olden, *supra* note 138, at xvii. Thus, while toxicogenomics research may receive some government funding, there is no impetus for polluting industries to invest in toxicogenomics in a similar way that there is an impetus for drug companies to invest in pharmacogenomics.

164. *See* Sadée, *supra* note 127, at 332; Olden, *supra* note 138, at xv.

165. *See* Sadée, *supra* note 127, at 330-32.

166. *Id.* at 331.

167. *Id.*

168. *Id.*

169. *Id.*

170. *Id.*

171. *See supra* text accompanying notes 156-165.

172. *See supra* text accompanying notes 156-160.

173. Toxicogenomists already regularly use microarray chips to determine toxic response generally. *See* Edward K. Lobenhofer et al., *Progress in the Application of DNA Microarrays,* 109 ENVTL. HEALTH PERSP. 881, 886 fig.2 (2001) (illustrating the microarray image and data analysis processes for toxicogenomic studies).

174. *Id.* at 885.

ethnically determined, and could help chemical manufacturers design chemicals that will not more severely effect different populations of persons.

Thus, pharmacogenomic advances are applicable to the toxicogenomic study of ethnically associated risks of toxicity in at least three ways: by showing that ethnicity may be a factor in susceptibility, by helping scientists to develop a general understanding of the effects of ethnicity on chemical disposition, and by developing applicable technology.[175] As pharmacogenomics and toxicogenomics advance further, this information will be supplemented with an even better understanding of the potential impact of these new sciences. Therefore, toxicogenomics will likely be an important tool for the EPA in assessing environmental justice issues.

B. Using Toxicogenomics as a Tool for Environmental Justice Assessments

When considering an environmental justice issue, the EPA must consider "[w]hether individuals, certain neighborhoods, or federally recognized tribes suffer disproportionately adverse health or environmental effects from pollution or other environmental hazards."[176] To do so, the EPA has created a framework to conduct environmental justice assessments.[177] This framework uses indicators to assess environmental justice problems in four phases.[178] As will be discussed in this part, advances in toxicogenomics concerning ethnic susceptibility to toxins can have an impact on several of the indicators in this assessment, and on the overall implementation of the four phase framework.

1. Effects of Toxicogenomics Advances with Regard to Ethnicity on Environmental Justice Indicators

Environmental justice indicators are intended to help Environmental Justice Assessment Teams get the "big picture" of a community's economic, social, environmental, and health level status, or well-being.[179] Indicators are divided into four categories: Environmental, Health, Social, and Economic.[180] Toxicogenomics advances pertaining to ethnic susceptibility to toxins and xenobiotics will affect how an Environmental Justice Assessment Team deals with several of these indicators.

First, these advances will help EPA Assessment Teams understand Environmental Indicators. As previously noted, Environmental Indicators include potential sources of environmental stressors and proximity thereto, the relative levels of stressors to which a community is being exposed and the ways exposure

175. *See supra* text accompanying notes 156, 161, and 166.

176. *See* TOOLKIT, *supra* note 1, at 4.

177. *Id.* at 15.

178. *Id.* at 18.

179. *Id.* at 25.

180. *Id.* at 27-30.

occurs, and the physical environment in which exposure occurs.[181] These potential sources can be hard to identify because the effects and interactions of various chemicals are little understood.[182] Furthermore, the severity of a toxic exposure varies with proximity, leading to prolonged exposure before any outward signs of toxicity become available.[183] However, with ethnicity-related advances in toxicogenomics, such as the availability of information on whether a particular ethnicity is more greatly affected by a toxin or xenobiotic, or information on how chemicals are metabolized based on genomics, EPA Assessment Teams will be able to better identify the chemicals that are at work, and how far away those chemicals are.[184] Thus, advances in toxicogenomics concerning ethnicity should be useful to Assessment Teams in their effort to identify environmental indicators.

Second, toxicogenomics advances related to ethnic susceptibility will help EPA Assessment Teams assess Health Indicators. As previously noted, Health Indicators allow Assessment Teams to understand the general health of a community and their ability to cope with stressors.[185] These indicators are also used to determine whether a subset of a population has certain health sensitivities, such as greater susceptibility to toxic substances.[186] Because toxicogenomics assessments of ethnic susceptibility to toxins would be the primary reason for such research, EPA Assessment Teams would stand to greatly benefit from toxicogenomics advances of this kind. Knowledge of ethnic susceptibility could be used to determine whether a population was suffering from disproportionate harm or even whether a population had a higher potential to be at risk. Because of this ability, an EPA Assessment Team could proactively prevent the production of toxins and xenobiotics around susceptible communities. Thus, Assessment Team's use of Health Indicators would greatly benefit from advances in toxicogenomics concerning ethnicity.

Finally, toxicogenomics advances concerning ethnicity will impact Environmental Justice Assessment Teams' understanding of Social Indicators. If Assessment Teams know about ethnic susceptibility to a particular toxin, it will better help them understand the impact of vulnerability indicators such as proximity

181. *Id.* at 28.

182. *Id.* at 33-34.

183. *Id.* at 63-64, 68.

184. *See* COMM. ON HOW TOXICOGENOMICS COULD INFORM CRITICAL ISSUES IN CARCINOGENIC RISK ASSESSMENT OF ENVTL. CHEMICALS, NAT'L RESEARCH COUNCIL OF THE NAT'L ACADS., TOXICOGENOMIC TECHNOLOGIES AND RISK ASSESSMENT OF ENVIRONMENTAL CARCINOGENS: A WORKSHOP SUMMARY 28-29 (2005) (noting that microarray patterns as a result of toxic exposure may change depending on whether the exposure was low, medium, or high); Mark Toraason et al., *Applying New Biotechnologies to the Study of Occupational Cancer—A Workshop Summary*, 112 ENVTL. HEALTH PERSP. 413, 414 (2004) (noting that toxicogenomics can produce biomarkers to show that a toxic exposure has occurred).

185. *See* TOOLKIT, *supra* note 1, at 29.

186. *Id.*

and access to hospital care.[187] With increased susceptibility and decreased access to medical care, a population would be more vulnerable to environmental toxins than in the absence of either.[188] Other Social Indicators will also be impacted.[189] If a particular ethnic community is more susceptible to a particular toxin, with time, they are likely to be educated about that susceptibility.[190] Thus, when a manufacturer that produces a particular toxin is sited close by, these communities are more likely to participate in the political process to fight that siting.[191] Thus, social factors will be impacted by a better understanding of toxicogenomics related to ethnicity.

2. Effects of Toxicogenomics Advances Concerning Ethnicity on Environmental Justice Indicators as Applied Through the Environmental Justice Toolkit's Four Phase Framework

Environmental justice indicators create the backbone of consideration that an Environmental Justice Assessment Team uses to work through each of the four phases of the Environmental Justice Framework.[192] Thus, toxicogenomics advances concerning ethnicity will also be important. As noted, four phases are used to assess potential environmental injustice: problem formulation, data collection, assessment of the potential adverse environmental and human health effects or impacts, and assessment of the potential for disproportionately high and adverse effects or impacts.[193]

187. A vulnerability indicator such as access to hospital care is an important demographic of a susceptible population because it indicates that the population is more vulnerable to exposure. *Id.* Access to hospital care is an important consideration because it indicates a population's ability to offset impacts of exposure. *Id.*

188. *See* Paul J. Nietert et al., *Demographic and Biologic Influences on Survival in Whites and Blacks: 40 Years of Follow-Up in the Charleston Heart Study,* 5 INT'L J. FOR EQUITY HEALTH 1, 2 (2006) (citing INSTITUTE OF MEDICINE, UNEQUAL TREATMENT: CONFRONTING RACIAL AND ETHNIC DISPARITIES IN HEALTH CARE (2002); Janice E. Williams et al., *Racial Disparities in CHD Mortality From 1968-1992 in the State Economic Areas Surrounding the ARIC Study Communities*, 9 ANN. EPIDEMIOLOGY 472 (1999); Donna L. Armstrong et al., *United States Coronary Mortality Trends and Community Services Associated with Occupational Structure, Among Blacks and Whites, 1984-1998*, 58 SOC. SCI. & MED. 2349 (2004); David R. Williams, *Race, Socioeconomic Status, and Health: The Added Effects of Racism and Discrimination*, 896 ANN. N.Y. ACAD. SCI. 173 (1999); Jeff Whittle et al., *Racial Differences in the Use of Invasive Cardiovascular Procedures in the Department of Veterans Affairs Medical System*, 329 NEW ENG. J. MED. 621 (1993); A. Marshall McBean & Marian Gornick, *Differences by Race in the Rates of Procedures Performed in Hospitals for Medicare Beneficiaries*, 15 HEALTH CARE FIN. REV. 77 (1994)).

189. *Id.* (citing Williams, *supra* note 188; Whittle et al., *supra* note 188; McBean & Gornick, *supra* note 188).

190. *See* GRAD, *supra* note 3, § 9.10[3][b].

191. *Id.*

192. *See supra* text accompanying notes 30-79.

193. *See supra* text accompanying notes 34-39.

In the first phase, problem formulation, toxicogenomics advances concerning ethnicity will help EPA Assessment Teams consider what started an assessment.[194] The problem formulation phase involves evaluating the population at potential risk, what kind of toxin might cause that risk, the location of the risk and population, and in what context the EPA is assessing the possible injustice.[195] As noted, the EPA assesses the Environmental and Social Indicators involved with this problem.[196] Thus, ethnic associations between toxins and the harms that impact those indicators will be important for the proper characterization of the environmental justice problem. In the second phase, the EPA collects data on the environmental actions or entities that cause the environmental or health effects, and the community of concern where impacts will be manifested.[197] Obviously, proper data collection would involve a search of Health Indicators involving ethnic susceptibility to toxins.[198] In phase three, the EPA Assessment Team considers the potential for adverse environmental and human health effects or impacts.[199] Because an EPA Assessment Team would have collected data about proximity, type of toxin, and ethnic susceptibility as well as other indicators,[200] it will have a fuller picture to assess, prospectively or retrospectively, the type of harm and relative seriousness of the environmental justice issue. Finally, knowledge of different susceptibility levels is powerfully useful in the final phase of the environmental justice assessment, since that phase involves determining differential impacts on a segment of the population.[201] Obviously, if one ethnic group has a higher overall susceptibility to a toxin or xenobiotic, an EPA Assessment Team must consider this in making a determination of whether there is an environmental injustice.

A full airing of the issues at stake in considering the potential for ethnic advances in toxicogenomics on environmental justice assessments requires that ethical issues be considered. As Sandra Soo-Jin Lee notes in her article, *Paradoxes of Difference*, "if we are all the same, why do we continue to search for the ways in which we differ from one another?"[202] Lee notes that although we are 99.9 percent the same, "there is increasing interest in identifying the relatively small percentage difference that distinguishes individuals."[203] As Lee notes, "[a]t stake are issues of power and trust, and the question of whether new genetic technologies will close

194. TOOLKIT, *supra* note 1, at 58.

195. *Id.*

196. *Id.* at 28, 29-30.

197. *Id.* at 63.

198. *See id.* at 65 (recommending that data be collected on factors that increase community vulnerability to stress).

199. *Id.* at 66.

200. *Id.* at 63-68.

201. *Id.* at 71.

202. Sandra Soo-Jin Lee, *Paradoxes of Difference*, 2 PUB. LIBR. SCI. BIOLOGY 1263, 1263 (2004).

203. *Id.*

the gaps between groups or make them wider."[204] Others argue that ethnicity is merely being used as a stand in for individual genetic variability in pharmacogenomics advances.[205] While this may be true, from the point of view of an environmental justice advocate it should not matter. Since environmental justice advocates are concerned that *populations* rather than individuals may suffer disproportionate harms based on their ethnicity,[206] it is actually preferable to make generalizations about adverse exposures. Rather than opening gaps between races, toxicogenomics as applied to environmental justice assessments stands to help close gaps between races. The reason is simple: it provides a small tool for those fighting environmental injustice.

IV. A REAL WORLD HYPOTHETICAL APPLICATION

A. The Background

The question then becomes in what situations is the public health of one ethnic group so disproportionately threatened that environmental justice advocates can call upon toxicogenomics in the context of the Environmental Justice Toolkit to make positive changes for a threatened community? An obvious example is the siting of any major pollution emitter.[207] Within that example lies a less obvious paradigm—the production of hot spots through local, state, or federal emissions trading programs.

Emissions trading programs have found a broad following among economists, industry, and some environmental groups as an alternative means of regulating industry that is market based and does not rely on command and control techniques.[208] The main features of such programs are an emissions cap, "the total amount of pollution that sources can emit," and allowances or credits that provide for the holder to discharge a certain amount of pollution.[209] These allowances may be freely traded within an industry.[210] Over time, the emissions cap is reduced, thus lessening the overall number of allowances.[211] The idea is that free trading of

204. *Id.* at 1264.

205. *See supra* text accompanying notes 120-126.

206. TOOLKIT, *supra* note 1, at i.

207. *See, e.g.*, Rena I. Steinzor, *Toward Better Bubbles and Future Lives: A Progressive Response to the Conservative Agenda for Reforming Environmental Law*, 32 ENVTL. L. REP. 11421, 11430 (2002); *see also* ROBERT V. PERCIVAL ET AL., ENVIRONMENTAL REGULATION: LAW, SCIENCE, AND POLICY 15-19 (5th ed. 2006) (discussing generally whether environmental justice victims come to areas where major polluters are sited or whether major polluters tend to site near victims).

208. *See* U.S. Envtl. Prot. Agency, Clear Skies Cap and Trade Basics, http://epa.gov/air/clearskies/captrade.html (last visited Dec. 5, 2006) [hereinafter Clear Skies Cap and Trade]; Rena Steinzor, Ctr. for Progressive Reform, Emissions Trading (2005), http://www.progressiveregulation.org/perspectives/emissions.cfm.

209. Clear Skies Cap and Trade Basics, *supra* note 208.

210. *Id.*

211. *Id.*

emissions "rewards companies that discover better ways to reduce emissions by allowing them to sell unneeded allowances in the market," allows companies that cannot reduce emissions to buy additional allowances, and puts businesses in the position of determining the cheapest way to comply with regulations.[212] Critics raise many concerns about the use of emissions trading programs.[213] Of great importance here, "[t]rading schemes have proved so vulnerable to abuse that they have resulted in absolutely unacceptable concentrations of life-threatening pollutants in areas where large numbers of people of color live."[214] These concentrations, known as "hot spots," are particularly dangerous when trading schemes are applied to toxics.[215] In addition, they may represent a major environmental justice issue.

One such trading program raised environmental justice issues in the jurisdiction of the South Coast Air Quality Management District (SCAQMD), a sophisticated regulatory agency in the heart of a major pollution area—Los Angeles, California.[216] The trading program, known as Rule 1610, or the "car scrapping program," "allow[ed] stationary source polluters (such as factories and refineries) to avoid installing expensive pollution control equipment if they purchase[d] pollution credits generated by destroying old, high-polluting cars."[217] The major environmental justice issue occurred because four oil companies began purchasing most of the emission credits.[218] These companies sought to avoid installing equipment to avoid the release of toxic gases, such as benzene, that escape during oil tanker loading at their marine terminals.[219] In the process, an

212. *Id.*

213. Aside from environmental justice issues, critics also raise ethical problems with the idea that a value can be placed on the "public interest in natural resources." Steinzor, *supra* note 207, at 11426. This problem centers around the distaste associated with turning the right to pollute into a commodity, thus removing the social stigma normally associated with it. Richard Toshiyuki Drury et al., *Pollution Trading and Environmental Injustice: Los Angeles' Failed Experiment in Air Quality Policy*, 9 DUKE ENVTL. L. & POL'Y. F. 231, 270-71 (1999).

214. Steinzor, *supra* note 207, at 11426.

215. *Id.* at 11427.

216. Drury et al., *supra* note 213, at 242 n.44.

217. *Id.* at 246. Because of the collectively high level of pollution from old cars and mobile sources generally and the cheapness of procuring and destroying such cars, the program was planned around the idea that high polluting stationary sources could buy and destroy the cars, reducing pollution in equal proportions to that emitted by the source and reducing overall pollution at a lower cost than that of installing pollution control equipment. *Id.* at 246-47, 247 n.60. Once destroyed, the procurer of the car received emission credits based on projected emissions of the car had it not been destroyed. *Id.* at 247.

218. *Id.* at 252 (citing Letter from Richard Toshiyuki Drury et al., Communities for a Better Environment, to Anne Goode, U.S. Envtl. Prot. Agency (Nov. 23, 1998) (on file with Richard Toshiyuki Drury et al.)).

219. *Id.* at 252-53 (citing Shipra Bansal & Scott Kuhn, *Stopping an Unfair Trade: Environmental Justice, Pollution Trading, and Cumulative Impacts in Los Angeles*, ENVTL. L. NEWS, Spring 1998, at 16, 17-18). The cancer risk associated with emissions from exposure to these toxic gases is "greater than 150 in 1 million for the maximum exposed individual." *Id.* at 253 (citing LOS ANGELES COUNTY BLDG & CONSTR. TRADES COUNCIL & THE STEAMFITTERS & PIPEFITTERS LOCAL 250, *Comments on the Draft Environmental Impact Report for the Renewal of Unocal's Lease for Berths 148-151, in* FINAL ENVTL

overwhelmingly Latino local population was being exposed to high levels of dangerous pollutants, while the general public in Los Angeles received a slight decrease in their exposure to these toxics.[220]

B. The Hypothetical Application

Applying toxicogenomics to a hypothetical environmental justice assessment of the SCAQMD trading program provides a useful context for understanding how the science can benefit the health of a disproportionately affected Latino population. Specifically, toxicogenomics studies could identify biomarkers of susceptibility, biomarkers of exposure, and biomarkers of effect that could then be used to identify the effects of the toxin on the population.[221] Biomarkers of susceptibility represent those biomarkers related to "variations affecting absorption, metabolism, or response to environmental agents."[222] Biomarkers of exposure help toxicogenomists see how much of a substance the body has absorbed.[223] Finally, "[b]iomarkers of effect reflect changes in cells or tissues triggered by chemical exposure or changes that are qualitatively or quantitatively predictive of health impairment or potential impairment due to toxic exposure."[224] Each of these types of biomarkers may assist environmental justice assessors in determining whether an environmental injustice has occurred.[225] Accordingly, each is discussed below in the context of the SCAQMD trading program.

Discovery of biomarkers of toxin susceptibility related to ethnicity will be particularly useful because these polymorphisms affect sensitivity to a toxin and can be discovered before exposure even occurs.[226] In the context of the Latino population suffering from high emissions of toxic fumes, these studies could show that the population suffered a higher susceptibility to cancer than the general,

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IMPACT REPORT FOR BERTHS 148-151 PORT OF LOS ANGELES 17, 23 (1996)). On the other hand, the Clean Air Act establishes that a cancer risk to a maximally exposed person of greater than one hundred in one million is significant and warrants EPA Administrator action to protect human health. *Id.* at 253 n.91 (citing 42 U.S.C. § 7412(f)(2)(A) (2000)).

220. Drury et al., *supra* note 213, at 254-55 (citing S. COAST AIR QUALITY MGMT. DIST./CALIFORNIA STATE UNIV. FULLERTON FOUND., THE DISTRIBUTION OF CURRENT AND FUTURE EXPOSURE TO OZONE, FINE PARTICULATE MATTER, CARBON MONOXIDE, AND NITROGEN DIOXIDE AMONG DEMOGRAPHIC GROUPS IN THE SOUTH COAST AIR BASIN, FINAL REPORT 5 (1993)).

221. Grodsky, *supra* note 84, at 183-87, 196-98. Grodsky notes that discovery of susceptibility biomarkers is usually associated with toxicogenetics, while discovery of biomarkers of exposure and effect are usually associated with toxicogenomics. *Id.* at 191-94. For purposes of this paper, toxicogenetics is enveloped in the discussion of toxicogenomics.

222. *Id.* at 183.

223. *Id.* at 185.

224. *Id.* at 186 (citations omitted).

225. *See supra* text accompanying notes 180-184.

226. Grodsky, *supra* note 84, at 183-84. It is important to note that "[s]usceptibility genes are 'neither necessary nor sufficient to cause disease. They modify risk.'" *Id.* at 184 (quoting Kenneth Olden & Janet Guthrie, *Genomics: Implications for Toxicology*, 473 MUTATION RES. 3, 5 (2001)).

mixed population.[227] Environmental justice assessors would consider these studies as a viable health indicator, identifying both a potential weakness in the population and how this weakness relates to that population's susceptibility.[228] As a result, assessors could have made a very early determination that an environmental justice problem existed. This could have resulted in specific guidelines for industry, including caps on the amount of emissions units that industry could purchase in a specific area.

Toxicogenomists' discovery of biomarkers of toxin exposure and effect will be useful because these biomarkers will give environmental justice assessors a direct method to measure human exposure to toxic substances.[229] Using gene chips, toxicogenomists would measure genes and the gene products of the disproportionately affected and genetically more susceptible population.[230] Assessors will know from these biomarkers how much exposure is in fact occurring, and can use that understanding as an environmental indicator to determine what action needs to be taken.[231] Thus, prior to any symptom manifestation, assessors will have the capability to determine whether higher exposure rates are affecting the Latino populations and may take appropriate steps to stop the environmental injustice, including altering the program or ending it all together.

This example, though hypothetical in the application of toxicogenomics, shows that toxicogenomics has real life applications in environmental justice assessments. By using toxicogenomics to identify and measure biomarkers of susceptibility, exposure, and effect, toxicogenomists can assist assessors in identifying both health and environmental indicators. Thus, in cases where an environmental justice issue may exist, such as the SCAQMD trading program, toxicogenomics represents a new tool that can be applied to assess and remedy environmental injustice.

CONCLUSION

As demonstrated throughout this article, new advances in genomics and ethnicity may provide the EPA with a new and powerful tool to protect minority populations from environmental injustice. As discussed, toxicogenomics can use advances in pharmacogenomics, and make advances of its own that will

227. For example, the risk of cancer for maximally exposed Latinos might be 300 persons in 1 million rather than the general population's "greater than 150 in 1 million for the maximum exposed individual." Drury et al., *supra* note 213, at 253 (citing LOS ANGELES COUNTY BLDG & CONSTR. TRADES COUNCIL & THE STEAMFITTERS & PIPEFITTERS LOCAL 250, *Comments on the Draft Environmental Impact Report for the Renewal of Unocal's Lease for Berths 148-151, in* FINAL ENVIRONMENTAL IMPACT REPORT FOR BERTHS 148-151 PORT OF LOS ANGELES 17, 23 (1996)).

228. TOOLKIT, *supra* note 1, at 29.

229. Grodsky, *supra* note 84, at 185-87.

230. *Id.* at 185-86, 195-96.

231. *See* TOOLKIT, *supra* note 1, at 28.

fundamentally change how Environmental Justice Assessment Teams use indicators in the four phase assessment framework and determine whether an environmental injustice does, could, or will exist.[232] Admittedly, genomics is unlikely to significantly change the outcome of EPA's assessment of environmental justice issues, but environmental justice advocates can use genomics to make incremental improvements to the way that injustice problems are addressed. As Karl Weick, a psychologist from Cornell University, once noted,

> Once a small win has been accomplished, forces are set in motion that favor another small win. When a solution is put in place, the next solvable problem often becomes more visible. This occurs because new allies bring new solutions with them and old opponents change their habits. Additional resources also flow toward winners, which means that slightly larger wins can be attempted.[233]

With new allies in the fields of toxicogenomics and pharmacogenomics, perhaps those who are disproportionately affected by environmental harm can gain new wins.

232. *Supra* text accompanying notes 156-206.

233. Karl E. Weick, *Small Wins: Redefining the Scale of Social Problems*, 39 AM. PSYCHOLOGIST 40, 43 (1984).