Sup. Ct., Bronx Co.
Index Nos. 8576/91

COURT OF APPEALS
STATE OF NEW YORK

PATRICIA NONNON, as Executrix of the Estate of KERRI NONNON, Deceased, and PATRICIA NONNON, individually, JOAN KOHN, as Administrator of the goods, chattels and credits which were of DANIELLE MAGLIO, deceased, and JOAN KOHN, individually, CHRISTOPHER ANGELILLI, RITA SEBASTIAN, an infant, by her mother and natural guardian, THERESA SEBASTIAN, and THERESA SEBASTIAN, individually, JUSTIN ZEITLIN, an infant, by his mother and natural guardian, SUSAN ZEITLIN, and SUSAN ZEITLIN individually, ANGELA DA BENIGNO, as Executrix of the Estate of JOANNE MARIE DA BENIGNO, deceased, and ANGELA DA BENIGO, individually, ANGELA DA BENIGNO, as Executrix of the Estate of PATRICIA ANN DA BENIGNO, individually,

Plaintiffs-Respondents,

-against-

THE CITY OF NEW YORK,

Defendant-Appellant.

BRIEF *AMICUS CURIAE* OF MARCIA ANGELL, PATRICIA A. BUFFLER, RONALD E. GOTS, PHILIP GUZELIAN, LEONARD D. HAMILTON, LAWRENCE S. LESSIN, SALLY L. SATEL AND RICHARD WILSON IN SUPPORT OF DEFENDANT-APPELLANT

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April 20, 2007

TABLE OF CONTENTS

page

TABLE OF CONTENTS (cont'd)

page

TABLE OF AUTHORITIES

page

Cases

TABLE OF AUTHORITIES (cont'd)

page

Cases (cont'd)

TABLE OF AUTHORITIES (cont'd)

page

Treatises and Other Secondary Materials

TABLE OF AUTHORITIES (cont'd)

page

Treatises and Other Secondary Materials (cont'd)

TABLE OF AUTHORITIES (cont'd)

page

Treatises and Other Secondary Materials (cont'd)

TABLE OF AUTHORITIES (cont'd)

page

Treatises and Other Secondary Materials (cont'd)

TABLE OF AUTHORITIES (cont'd)

page

Treatises and Other Secondary Materials (cont'd)

Internet Websites

TABLE OF AUTHORITIES (cont'd)

page

Internet Websites (cont'd)

INTEREST OF *AMICI*

Amici are scientists who have studied the role that scientific issues play in public affairs and in particular the way in which they can illuminate disputes between different persons or elements of society in courts of law. *Amici* include epidemiologists, physicians with expertise in epidemiology and oncology, a prominent physicist with broad expertise in toxicology, epidemiology, public health and safety, environmental pollution, chemical carcinogens, air pollution, ground water pollution, and cancer risk and toxicologists with expertise in toxicology, epidemiology, oncology, public health and public policy on medical issues.[1]

Amici believe, and contend here, that whether a court applies the "*Daubert*" approach or the more "traditional" test first articulated in *Frye v. United States*, 293 Fed. 1013 (D.C. Cir. 1923) and by this Court in *People v. Wesley*, 83 N.Y.2d 417 (1994), the central issue any court must decide when expert evidence is proffered is whether that evidence is "reliable."

Reliability is a criterion common to law and to science. *Amici* advocate the principle that the particular criteria established by scientists in the scientific field relevant to a dispute should be applied at the interface between science and law in both federal and state litigation because it reflects the way scientists approach

[1] The credentials of *amici* are set forth in the biographical addendum to this brief.

questions of causation and is the accurate way to determine whether there is causation.

Sympathy for a claimant is not a substitute for evidence, and should not eliminate the requirement in tort law that the plaintiff prove causation -- that is, that the alleged cause was more likely than not to be the actual cause of the injury. If the legal system is not to become merely a "lottery," where determinations are random, or a social mechanism to redistribute wealth, it should ensure that claims are based on the best science the various relevant scientific disciplines can muster.

Amici were disappointed that the trial court and the closely divided Appellate Division panel did not properly assess the unreliable causation evidence submitted by plaintiff and that the thoughtful dissenting opinion of Justice Andreas was not followed.

PRELIMINARY STATEMENT

This consolidated appeal concerns nine separate toxic tort actions in which the plaintiffs allege that they, or their family members, who live or lived in the vicinity of the Pelham Bay Landfill in the Bronx ("the Landfill") were exposed to hazardous substances in the Landfill and contracted either acute lymphoid leukemia ("ALL") or Hodgkin's disease, which they claim were caused by that exposure. The total damages claimed is approximately $5 billion.

Defendant-appellant, the City of New York ("the City"), has appealed from the order and decision of the Appellate Division, First Department, dated June 6, 2006 (1153-1214, *see Nonnon v. City of New York*, 32 A.D.3d 91 (1st Dept. 2006), *app. granted*, __A.D.3d__, 2006 N.Y. App. Div. LEXIS 10431 (1st Dept. 2006)) in which, by a three to two majority, the Appellate Division upheld the denial of the City's motion for summary judgment dismissing these actions by the Supreme Court, Bronx County (Hunter, J.) (*Nonnon v. City of New York*, 1 Misc.3d 897 (Sup. Ct., Bronx Co., 2003) (21-26).[2] The Appellate Division granted the City leave to appeal on a certified question as to whether the order of the Supreme Court, as modified by the Appellate Division, was properly made (1151-52).

The principal issue is whether the Appellate Division erred in failing to grant the City's motion based upon plaintiffs' failure to establish causation by submission of admissible expert opinions which rested on a proper foundation and which were based upon scientifically reliable methodologies. The City seeks reversal based on *Parker v. Mobil Oil Corp.*, 7 N.Y.3d 434 (2006), *rearg. den.*, __ N.Y.3d __, 2007 N.Y. LEXIS 3 (2007) ("*Parker*").

Amici agree that *Parke*r supports reversal, and submit that the well-reasoned dissenting opinion in the Appellate Division by Justice Andreas correctly assessed

[2] Unless otherwise stated, numbers in parentheses refer to pages in the Record on Appeal.

the scientific evidence, and recognized that these actions should be dismissed as a matter of law. In contrast, the majority opinion in the Appellate Division appears motivated by sympathy for claimants who suffered or are suffering serious, often fatal, illnesses. But the majority decision is not supported by the record or sound science, and fails to assess the reliability of the evidence proffered by plaintiffs.

QUESTION PRESENTED

Amici will address only the first question presented: Should the City's motion for summary judgment have been granted where plaintiffs failed to meet their burden to show causation, both general and specific, in that their belated experts' submissions were conclusory, lacked foundation and were based on scientifically-invalid and infirm methodologies not generally-accepted in the scientific community?

Amici believe that the plaintiffs' experts did not offer proper foundation for their ultimate conclusions that the Landfill caused plaintiffs to contract ALL and Hodgkin's disease, that their expert opinions were not based on accepted scientific methodology, that they were unreliable and inadmissible, and should have been excluded as a matter of law. It follows that the City's motions for summary judgment dismissing the complaints should have been granted.

STATEMENT OF FACTS

Amici rely on the Statement of Facts in Appellant's Brief. To the extent relevant and necessary or appropriate, we cite to and describe the record evidence in our Argument.

ARGUMENT

I.

THE EXPERT AFFIDAVITS PROFFERED BY PLAINTIFFS SHOW THAT PLAINTIFFS' EXPERTS DID NOT FOLLOW GENERALLY ACCEPTED METHODS, AND THEY THUS LACK FOUNDATION AND ARE NOT RELIABLE

Introduction

This case is not about precautionary actions to be taken to ensure that landfills are properly designed or maintained, nor is it about precautions to be taken by or for those living near land fills. Statutory and regulatory authorities each have legal procedures to ensure that proper precautions are being taken. This case is about a claim for injury allegedly caused by a particular action or omission by a particular actor. Both in ordinary common sense and in law, these are very different, and different standards of proof apply.

This case involves the claim that several children living close to the Pelham Bay Landfill in The Bronx, New York developed one of two disorders – Acute Lymphocytic Leukemia (ALL) and Hodgkin's Disease -- as a result of their residential proximity to the Landfill. (42-44, 627, 751).

A. Plaintiffs' Epidemiological Evidence

Epidemiological evidence is indispensable in toxic and carcinogenic tort actions where direct proof of causation is lacking. *See Brock v. Merrell Dow Pharmaceuticals, Inc.*, 874 F.2d 307 (5th Cir.), *modified on reh'g*, 884 F.2d 166 (5th Cir.1989), *cert. denied*, 494 U.S. 1046 (1990).

Plaintiffs rely principally on one retrospective study by Richard Neugebauer (hereafter called the "Neugebauer study") purporting to show a higher than expected incidence of ALL[3] among persons living close to the Landfill. However, two other studies, with equal or greater statistical power performed by the New York City Department of Health, found no such relationship. There are several independent, fundamental and profound flaws in the claimants' experts' reasoning, any one of which would invalidate the conclusion. To reach their causal conclusions, Plaintiffs' experts have violated all of the critical principles of generally-recognized and generally accepted causation methodology.

Epidemiologist Richard Neugebauer, Ph.D., M.P.H. (602-10) reviewed the 1988 and 1994 NYCDOH Studies and conducted his own study (which was not

[3] Neugebauer apparently did not study the incidence of Hodgkin's Disease in the vicinity of the Landfill.

provided to defendant or the Court)[4], and based on this he concluded that "to a reasonable degree of epidemiological certainty that the dump was a substantial factor in causing an excess number of cases of [ALL] among children in the surrounding neighborhoods." (602)

Neugebauer's affidavit is also unreliable because it presented insufficient information to allow a reader to understand and replicate his methods.[5]

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[4] The failure of plaintiffs' experts to provide their studies and underlying data is itself fatal from a scientific perspective. Experiments or analyses that are the foundation of scientific "findings" must be replicable and thus falsifiable. "Scientific methodology today is based on generating hypotheses and testing them to see if they can be falsified; indeed, this methodology is what distinguishes science from other fields of human inquiry." *See* C. Hempel, PHILOSOPHY OF NATURAL SCIENCE 49 (1966) ("[T]he statements constituting a scientific explanation must be capable of empirical test."); K. Popper, CONJECTURES AND REFUTATIONS: THE GROWTH OF SCIENTIFIC KNOWLEDGE 37 (5th ed. 1989) ("[T]he criterion of the scientific status of a theory is its falsifiability, or refutability, or testability"). Likewise, submission to the scrutiny of the scientific community is a component of "good science," in part because it increases the likelihood that substantive flaws in methodology will be detected. *See* J. Ziman, RELIABLE KNOWLEDGE: AN EXPLORATION OF THE GROUNDS FOR BELIEF IN SCIENCE 130- 133 (1978); A.S. Relman and M. Angell, "How Good Is Peer Review?," 321 *New Eng.J.Med*. 827 (1989). *See Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 593-94 (1993).

[5] Respondent' assertion that Dr. Neugebauer disclosed that he used "regression analysis" (Respondents' Brief at 42) is meaningless, because without knowing all the inputs the expert used in his regression analysis, this statement does not permit another researcher to replicate the calculation and test it for accuracy and completeness. A regression model attempts to combine the values of certain variables (the independent variables) in order to get expected values for another variable (the dependent variable). *See* David H. Kaye & David A. Freedman, Reference Guide on Statistics, § V.B.3, in the Federal Judicial Center, REFERENCE MANUAL ON SCIENTIFIC EVIDENCE (2d ed. 2000) (hereafter "Reference Manual.") [We refer to the Reference Manual on Scientific Evidence not because it is in any sense "binding" on this Court, but because its description of the methodologies underlying the several disciplines described therein are accurate and instructive. Indeed, the Reference Manual is not "binding" on federal courts; it is designed to facilitate the identification of issues concerning scientific evidence and to educate judges by outlining the fundamental principles in the areas of science that are often critical to issues in dispute.] Reference (continued...)

It is a well known principle of statistics that in assessing the statistical probability of an event or experimental result, decisions about what data to include and exclude, the size of the cohort and the boundaries of the bins in which the data are collected must not be modified to maximize the result. After purportedly limiting his study to a proximity analysis utilizing central circle and concentric bands around the census tracts, Neugebauer concluded that the Landfill was the cause of ALL in each of these plaintiffs because the incidence rate in children living closest to the Landfill was greater than in children living further away. In contrast, the NYCDOH

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[5](...continued)
Manual at 144. "Failure to develop the proper theory, failure to choose the appropriate variables, or failure to choose the correct form of the model can bias substantially the statistical results, that is, create a systematic tendency for an estimate of a model parameter to be too high or too low." (Reference Manual at 186). Moreover, in interpreting the results of a multiple regression analysis, it is important to distinguish between correlation and causality. Two variables are correlated when the events associated with the variables occur more frequently together than one would expect by chance. A correlation between two variables does not imply that one event causes the second. Therefore, in making causal inferences, it is important to avoid spurious correlation. Even when an appropriate theory has been identified, causality can never be inferred directly. One must also look for empirical evidence that there is a causal relationship. Conversely, the fact that two variables are correlated does not guarantee the existence of a relationship; it could be that the model—a characterization of the underlying causal theory—does not reflect the correct interplay among the explanatory variables (Reference Manual at 183-184). Failure to include a major explanatory variable that is correlated with the variable of interest in a regression model may cause an included variable to be credited with an effect that actually is caused by the excluded variable. In general, omitted variables that are correlated with the dependent variable reduce the probative value of the regression analysis. (Reference Manual at 188), and bias caused by the omission of an important variable that is related to the included variables of interest can be a serious problem. (Reference Manual at 189). This may lead to inferences made from regression analyses that do not assist the trier of fact. (Reference Manual at 188).

studies had also performed a proximity analysis, but had reached an opposite conclusion.[6]

In the case of the Neugebauer study, the decision to look at Acute Lymphocytic Leukemia and Hodgkin's Disease (only two) instead of one of twenty or so other cancers that might have had increased rates of occurrence, was made in full knowledge of the result (he knew there were 12 cases), and that invalidates ordinary

[6] Dr. Neugebauer admittedly initially did not perform any adjustments for race and ethnicity in assessing cancer incidence rates for the New York City population (605), as had the NYCDOH studies, and it was extended through 1994 and specifically considered only ALL, not all types of leukemia. (603-605). As a result Neugebauer admittedly failed to take into account random error and known risk factors associated with ALL such as race and ethnicity, which rendered his findings invalid. This failure to recognize and control for confounding factors biased Neugebauer's findings did not comport with sound epidemiologic methods. Reference Manual at 371-73) An epidemiological study should look for and address sources of bias before concluding the validity of a study's findings (Reference Manual at 363). Neugebauer's purported correction of his error in his reply affidavit, in which he claimed that adjustment for race made no difference in his statistical findings, again failed to meet generally accepted scientific standards because Neugebauer provided insufficient information about his methods and incomplete information about his analysis.

statistical criteria.[7] This alters the statistical validity and epidemiologists[8] often call a study such as that of Neugebauer a "hypothesis generating" study, useful for generating a hypothesis (that landfills cause ALL), but not for proof of the hypothesis. In this case, the hypothesis is negated by the other two studies of the Landfill and other information concerning chemical causation of ALL.

What Neugebauer observed was a geographical association with a certain form of leukemia. An association does not establish causation.[9] This principle is so well

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[7] The late Nobel Laureate Richard Feynman had a dramatic way of expressing this to his freshman physics class. Coming into class he said, "You know, the most amazing thing happened to me tonight. I was coming here, on the way to the lecture, and I came in through the parking lot. And you won't believe what happened. I saw a car with the license plate ARW 357! Can you imagine? Of all the millions of license plates in the state, what was the chance that I would see that particular one tonight?" (*See* D.L. Goodstein, "Richard P. Feynman, Teacher," *Phys. Today*. 70-75 (February 1989)). We can easily work it out: 3 is one out of 10 numbers, 5 is one out of 10 numbers, 7 is one of 10 numbers, A is one of 26 letters, R is one out of 26 letters, and W is one out of 26 letters. If we multiply these numbers together we find a low probability of one in eighteen million. Yet Feynman saw it. This commonplace experience does not seem that improbable. What is the answer to this paradox? As presented, the answer to this paradox is obvious: Feynman asked the question about the license plate when he already knew the answer. It thus made no sense to ask the question.

[8] Epidemiology is the study of disease patterns in human populations. *See, e.g., General Electric Co. v. Joiner*, 522 U.S. 136, 143, n.2 (1997); Reference Manual at 336. It "attempts to define a relationship between a disease and a factor suspected of causing it." *Brock v. Merrell Dow Pharmaceuticals, Inc.*, 874 F.2d 307, 311 (5th Cir.), *modified on reh'g*, 884 F.2d 166 (5th Cir.1989), *cert. denied*, 494 U.S. 1046 (1990). Epidemiological evidence is indispensable in toxic and carcinogenic tort actions where direct proof of causation is lacking. *See Brock v. Merrell Dow Pharmaceuticals, Inc.*, 874 F.2d 307 (5th Cir.), *modified on reh'g*, 884 F.2d 166 (5th Cir.1989), *cert. denied*, 494 U.S. 1046 (1990).

[9] However, it should be emphasized that *an association is not equivalent to causation*. "Observational studies can establish that one factor is associated with another, but considerable analysis may be necessary to bridge the gap from association to causation." Reference Manual on

(continued...)

established in all areas of science that to state or even imply otherwise is methodologically erroneous.

Even if one were to assume that the Neugebauer study was actually performed in an epidemiologically acceptable manner (itself in dispute) and was not merely a hypothesis generating study, the study must be considered in conjunction with the other two studies. Any claim that the Neugebauer study is proof of causation would have to demonstrate that it was superior to, and negates, the two NYCDOH studies.

Case-control studies such as Neugebauer's allow the *possibility* of showing an association which might represent causation, but a single such study is seldom sufficient to do so. It is only by combining the results of numerous epidemiological

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[9](...continued)
Statistics, Reference Manual at 91. An association identified in an epidemiologic study may or may not be causal. Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study's design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge. It is important to emphasize that most studies have flaws. Some flaws are inevitable given the limits of technology and resources. In evaluating epidemiologic evidence, the key questions, then, are the extent to which a study's flaws compromise its findings and whether the effect of the flaws can be assessed and taken into account in making inferences. A final caveat is that employing the results of group-based studies of risk to make a causal determination for an individual plaintiff is beyond the limits of epidemiology. (Reference Manual at 337). A well known example that a simple correlation between two variables does not establish causation comes from an article by H. Sies in 332 *Nature* 495 (1988) where he plots the number of brooding storks in Germany between 1965 and 1980 and relates them to the number of newborn babies. (Available at http://physics.harvard.edu/~wilson/soundscience/storks1.gif). The "association' is far stronger than the claimed association in this case. It would obviously be unwise to accept that association as "proof" of the hypothesis that storks bring babies (or that babies bring storks). Likewise it would be unwise for the Court to accept the Neugebauer study, even if it had no other problems, as evidence of causation.

and toxicological[10] studies that it is possible to verify cause-effect relationships. The single Neugebauer study has been improperly applied to both general and specific causation in this case. However, as we explain below, this lynchpin of the plaintiffs' case does not support the ultimate conclusion of causation.

B. General Causation and Specific Causation

I. General Causation

There are two generally recognized and accepted components to causation assessment. By "causation assessment," we mean determining whether a disease in an individual was caused by exposure to a chemical substance, in this case a chemical actually found in the Landfill.

The first component is "general causation" – the determination that a chemical or group of chemicals is capable of producing the disease(s) at issue. If there is no chemical or agent known that can cause the specific disease, it becomes superfluous to ask whether an agent caused the disease in a specific person. The second component is specific causation – the determination that the agent(s), known to have such a capability, did, in fact, cause the diseases in these particular Plaintiffs. In this

[10] Toxicology is "the study of the adverse effects of chemicals on living organisms." *See* Casarett and Doull's TOXICOLOGY: THE BASIC SCIENCE OF POISONS 13 (Curtis D. Klaassen ed., 5th ed. 1996); *see also* Reference Manual at 397.

case, general causation is not proved, and even if it were, there is no evidence of a level of exposure or dose that could lead to specific causation.

The disease(s) at issue are acute lymphocytic leukemia (ALL) and Hodgkin's Disease. No chemicals are known to cause either of these diseases. Leukemia is a general term for a cancer of the blood. While it is true that ALL is a form of leukemia and benzene is known to cause a form of leukemia, benzene has been proven, and is generally accepted, to cause only acute myelogenous leukemia (AML), and not other forms of leukemia such as ALL. AML and ALL are not the same disease and can be easily distinguished by examination of blood samples. Many dozens of studies in heavily-exposed worker populations (with real measured exposures) have found a consistent relationship between benzene and AML, *but not* for either ALL or Hodgkin's Disease. [11, 12, 13, 14] Although some studies are positive for these diseases,

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[11] The famous study of Istanbul shoemakers found Acute Myelogenous Leukemia but no excess of Acute Lymphocytic Leukemias. M. Aksoy, M. and S. Erdem, 165 *Annals of New York Academy of Sciences* 13 (1969).

[12] Rinsky, R.A., Young R.J. and Smith A.B., 2 American Journal of Industrial Medicine 217 (1981 and updates). Follow-up was 98.6% complete. Again, a statistically significant excess risk of leukemia was found for the total cohort (9 observed, 2.7 expected), but only for Acute Myelogenous Leukemia, a type not seen near Pelham Bay.

[13] A Study in China found Acute Myeoloegous Leukemia but not ALL. R.B. Hayes, Y. Songnian, M. Dosemeci, M. Linet, "Malignancies in humans," 40 *American Journal of Industrial Medicine* 117-126.

[14] A similar study in Pavia and Milan (Italy) E.C. Vigliani. and A. Forni, "Leukemias Associated with Benzene Exposure," *Annals of New York Academy of Sciences* 143-151 (1976).

many others are negative and, thus, the weight of evidence is that there is no known chemical cause of ALL or Hodgkin's Disease and the requirement for establishing causation is not fulfilled. There are a number of national and international agencies that collect and sift such information and make the information generally available. One such is the U. S. Environmental Protection Agency which keeps an up to date listing on its "Integrated Risk Information System" ("IRIS") website.[15] Another is the International Agency for Research on Cancer (IARC)[16] which maintains a series of monographs, evaluated by expert committees, on "Evaluation of the Carcinogenic Risks to Humans."[17]

The majority in the Appellate Division based its holding in large part of the "fact" that:

> Diane Trainor, plaintiffs' occupational and environmental safety and health expert, based her opinion on her review of mountainous documents[18] and noted that "defendants in this lawsuit originally commissioned many of the studies I looked at." The expert stated that nine known carcinogens (benzene, carbon tetrachloride, chloroform, ethylene dichloride, methylene chloride, perchloroethylene,

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[15] Integrated Risk Information System: http://www.epa.gov/iris/subst/index.html. This lists all the chemicals believed to be important by the U.S. EPA.

[16] IARC is a United Nations agency, a part of the World Health Organization.

[17] Available at http://monographs.iarc.fr/

[18] We will discuss the significance, or rather insignificance, of the volume of literature an expert supposedly relies upon elsewhere in this brief.

> trichloroethylene, vinyl chloride, and vinylidene chloride) have been found in the landfill and its emissions and that there is an abundance of research conclusively supporting the association between low-level exposure to toxic substances and the development of diseases, including leukemia and cancer.

32 A.D.3d at 116.

"Occupational safety and health expert" Diane Trainor, Ph.D., C.I.E. (584-92, 594), stated that her study (which was never provided) sought to analyze the "possible" exposure pathways for the Landfill neighbors, and then concludes that "a variety of pathways exist by which the plaintiffs were exposed to hazardous and toxic chemicals emanating from the dump for the time period the dump was open and for many years following . . . ." (586) Based upon her review of various documents concerning the Landfill, the disposal activities and the NYCDOH studies, she states that nine known carcinogens (benzene, carbon tetrachloride, chloroform, ethylene dichloride, methylene chloride, perchloroethylene, trichloroethylene, vinyl chloride, and vinylidene chloride)[19] have been found in the Landfill and its emissions and

[19] Respondents repeatedly refer to various substances in the Landfill as "carcinogens" and assert that they are linked to "cancer" or "leukemia." For this proposition they cite such sources as J. Harr, A CIVIL ACTION (1995) about toxins in groundwater at Woburn, Massachusetts (Respondents' Brief at 46-47) and the movie "Erin Brokovich," about ingestion of hexavalent chromium in groundwater in California (Respondents' Brief at 49) and a report by the New York Public Interest Research Group (NYPIRG), an advocacy and lobbying organization, not a scientific body (Respondents' Brief at 11-12). These sources can hardly be considered scholarly or authoritative and for the most part take dramatic license with the facts. In citing these sources and (continued...)

conclusorily states that there is "an abundance of research conclusively supporting the association between low-level exposures to toxic substances and the development of diseases, including leukemia and cancer." (591) In her opinion, "the [L]andfill caused the leukemia, Hodgkin's disease *and other forms of cancer* that exist in the plaintiffs . . . ." (584).[20, 21] (emphasis supplied). The Trainor affidavit mainly addresses concerns about "*possible* exposure pathways" by which the plaintiffs might have been exposed to landfill chemicals (586, emphasis supplied). Trainor did not consider whether specific chemicals were capable of causing ALL or whether any of the individual plaintiffs were exposed to particular chemicals known to cause ALL and whether they received a dose of that chemical sufficient to cause the disease. Trainor lists chemicals without any indication of biological association with ALL, without measures or estimates of exposure or dose, and without considering whether

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[19](...continued)
others, Respondents ignore the questions of dose, pathway of exposure, the specific disease implicated, and other factors that must be taken into account when assessing specific causation.

[20] Plaintiffs submitted affidavits in which they described their contact with the Landfill or the area surrounding it (302-85, 496-517; *see* 1198-99 (dissent)). Plaintiffs also submitted an Examination Before Trial of Sandra Irizarry (386-494), and the foreword to a 1983 NYPIRG Toxic Projects Report regarding the City's six Landfills (518-21). None of these documents establish levels of exposure or dose.

[21] It may seem a minor point, but it is telling that Trainor refers generally to "leukemia," not differentiating among the various forms of leukemia (each of which is a distinct disease) and then refers to "other forms of cancer," seemingly unaware that the only diseases diagnosed and claimed to have been contracted by the plaintiffs in this case are ALL and Hodgkin's disease.

any of the exposures were sufficient to cause the diseases which plaintiffs claim to have contracted.

Let us examine each of the nine "carcinogens" Trainor identified:

First, she failed to mention that only two of these are known to cause cancer in humans at any site and none has been found to cause ALL or Hodgin's Disease. It is instructive to examine the judgement of IRIS and IARC on these materials.

Second, of the chemicals suggested by Trainor, only Benzene and Vinyl Chloride are classified by IARC as "carcinogenic to humans.[22] The entry for Ethylene Oxide is "Evidence for Carcinogenicity (limited)"and the entry for vinylidene chloride is "Evidence for Carcinogenicity (inadequate)."[23] [emphasis supplied]

Following is a synopsis of EPA's IRIS[24] listing for the chemicals Trainor identified:

Carbon tetrachloride	Carcinogenic based on animal studies although only three cases of liver cancer have been suggested.
Chloroform	Has only been suggested to be carcinogenic based on animal studies. Moreover, "chloroform is not likely to be carcinogenic to humans by any route of exposure under exposure conditions that do not cause cytotoxicity and cell

[22] IARC Monographs on Evaluation of the Carcinogenic Risks to Humans Supplement 7 at 41-42.

[23] *Id*. at 205 and 376, respectively.

[24] http://www.epa.gov/iris/

	regeneration." These would be at high doses not suggested to have occurred near the Landfill.
Ethyl chloride[25]	IRIS lists only mouse study that showed delayed fetal ossification, but there was no established a concentration-response relationship, and no data on effects on the maternal mouse.
Methyl chloride	IRIS lists no human data IRIS states that "the available data suggest that methyl chloride would be classified as an agent whose carcinogenic potential cannot be determined."
Perchloroethyelene	Not separately listed in the IRIS database.
Trichloroethylene	Carcinogenic for animals but "carcingenicity for humans is withdrawn. A more recent evaluation for trichloroethylene states "there is limited evidence in humans for the carcinogencicity of trichloroethylene,"[26] although in one small study of 25 cases of Hodgkin's disease in Sweden in 12 of the cases some exposure to solvents had been reported.
Vinyl chloride	Noted to cause "Liver angiosarcomas, angiomas, hepatomas, and neoplastic nodules" but there is no mention of Hodgkin's disease or ALL.
Vinylidene chloride	Not separately listed in the IRIS database.

The EPA lists include chemicals that are known to cause cancer at high doses in laboratory animals, but there are serious limitations and risks in extrapolating from

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[25] "Ethylene dichloride," which Trainor mentions is not separately listed in IRIS. 1, 1 Dichloroethylene is listed, but was found only to cause "minimal"changes in the fatty content of liver cells in rats at very high doses. Tests in mice showed no carcinogenic effects. *See* http://www.epa.gov/iris/subst/0039.htm

[26] IARC Monograph 63 at 75-158 (1995).

animals to humans and from high exposures to low doses.[27] Moreover, the IRIS list is to be used for precautionary purposes, not for a determination of specific causation

[27] One of the uncertainties is associated with extrapolation both from animals to humans and from high to low doses. *See* Reference Manual at 97. To get measurable effects in animal experiments, chemicals are administered at very high doses. Results are extrapolated, using mathematical models, to the very low doses of concern in humans. However, there are many dose response models to use and few grounds for choosing among them. Different models often produce radically different estimates of the "virtually safe dose" in humans. D. A. Freedman & H. Zeisel, "From Mouse to Man: The Quantitative Assessment of Cancer Risks," 3 *Stat. Sci*. 3 (1988). Many experts have concluded that evidence from animal experiments is generally insufficient by itself to establish causation. *See generally* B. N. Ames, et al., "The Causes and Prevention of Cancer," 92 *Proc. Nat'l Acad. Sci. USA* 5258 (1995); S. R. Poulter, "Science and Toxic Torts: Is There a Rational Solution to the Problem of Causation?," 7 *High Tech. L.J.* 189 (1993) (epidemiological evidence on humans is needed). *See also* Committee on Comparative Toxicity of Naturally Occurring Carcinogens, National Research Council, Carcinogens and Anticarcinogens in the Human Diet: A Comparison of Naturally Occurring and Synthetic Substances (1996); Committee on Risk Assessment of Hazardous Air Pollutants, National Research Council, Science and Judgment in Risk Assessment 59 (1994). The generally accepted methodology for determining toxicity requires positive results in tests on more than one species and supporting epidemiological evidence.

We would also point out that animal studies do *not* enable us to *predict* whether or not an agent will be toxic in humans. Animal studies may provide supportive information, they may provide information regarding mechanisms of potential toxicity, but they are only one small piece of the puzzle. They tell us that at *this* particular dose, in *this* one species under *these specially-controlled circumstances*, the agent is toxic. But in contrast with laboratory experiments, the environment of humans is not controlled, humans are not laboratory bred, and humans are not rats or mice. Because of differences between species, it is almost impossible to extrapolate animal findings to humans with any certainty. *See Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987) ("the effects of chemicals differ between humans and rats").

The dose, as it relates to body mass, is also significant. If the human exposure involves low dose, the animal tests should involve similarly low doses. Scientists, and courts, reject the applicability of high-dose animal tests because any substance can be toxic when given at sufficiently high doses. Comparable doses are necessary in order to appropriately apply animal data to humans.

Toxicologists also require that animal tests involve the same or equivalent routes of exposure as human exposure before they extrapolate animal test results to humans because the route of exposure can dramatically affect whether a substance is toxic. *See Chikovsky v. Ortho Pharm. Corp.*, 332 F. Supp. 341 (S.D. Fla. 1996).

or fault, which is the function of a lawsuit. Typically, regulatory regimes identify risks, not proven causation. So, for example, the fact that ATSDR includes a chemical does not establish causation.

An extensive search has been made by national and international agencies for human data on these subjects, but there has been no highly exposed group of people that would enable such a study to be made.

Although there are some general groupings that are listed by IARC as being carcinogenic to people such as coke production, rubber industry, furniture making, and soots, landfills and waste dumps are not among them.

The overall weight of evidence does not therefore permit a conclusion that General Causation has been established. Neither ALL nor Hodgkin's disease is "known" to be chemically induced, nor known to be induced by landfills. A full examination of IRIS and IARC[28] listings will not show <u>any</u> chemical listed as causing ALL or Hodgkin's disease in humans.[29] The burden is clearly on the plaintiffs to

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[28] The IARC list is available at: http://monographs.iarc.fr/ENG/Classification/crthgr02a.php

[29] Respondents' reliance on the Agency for Toxic Substances and Disease Registry (ATSDR) treatment of benzene as a "known risk factor" is misplaced. A classification "risk factor" is simply not the equivalent of "known to" or even "probable" cause of a disease. Moreover, the ATSDR website states that benzene can cause "acute myelogenous leukemia, often referred to as AML." ATSDR also states:

> Cohort studies of benzene-exposed workers in several industries (*e.g.*, sheet-rubber manufacturing, shoe manufacturing, and rotogravure [a special printing process]) have demonstrated significantly elevated risk of leukemia-predominantly acute myelogenous leukemia, but also erythroleukemia and acute myelomonocytic

(continued...)

suggest a chemical that caused the two specific diseases diagnosed in the plaintiffs in this case, but none of the chemicals accused by Trainor have been found to cause ALL or Hodgkin's Disease in humans, so Plaintiffs' "proof" fails to satisfy the requirement of General Causation. To conclude that the Landfill caused these cases of ALL and Hodgkin's disease flies in the face of generally accepted knowledge regarding general causation.

Plaintiffs also submitted affidavits from toxicologist Jesse H. Bidanset, Ph.D. (523-29), who was asked to conduct an investigation to determine whether "chronic long term exposure" to the toxins emanating from the Landfill plaintiffs caused the various diseases (524). He reviewed the usage history of the Landfill and the NYCDOH Studies. He stated that the conditions allowed for chemical conversion of toxic materials to even more hazardous substances, providing a list of 59 chemicals from A (acetamide) to Z (zinc) (575-7 8) (some of which were duplicates or

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[29](...continued)
leukemia. The latency period for benzene-induced leukemia is typically 5 to 15 years after first exposure. Patients with benzene-induced aplastic anemia progress to a preleukemic phase and develop acute myelogenous leukemia. However, a person exposed to benzene may develop leukemia without having aplastic anemia.

Studies addressing the risk of leukemia associated with occupational exposures to low levels of benzene (less than approximately 1 ppm) have been inconclusive. Death certificates do not reveal increased leukemia mortality among workers potentially exposed to low levels of hydrocarbons and other petroleum products.

ATSDR, "Benzene Toxicity, Physiologic Effects, Hematologic Effects, Leukemia," at http://www.atsdr.cdc.gov/HEC/CSEM/benzene/physiologic_effects.html.

erroneously listed), which he termed "a collection of chemicals the likes of which has never been simulated in a laboratory." (525-26)[30] He acknowledged that ". . . it is impossible to determine exactly what effects each chemical has had due to its reaction with the other chemicals on the list" but nevertheless believed ". . . it is to a toxicological certainty that the cumulative effects of combining all these chemicals is highly detrimental to human health." (526). He also stated: "The toxicological literature [31] clearly indicates an association between chronic exposures to the toxic substances released from landfills like the DUMP that is the subject of this lawsuit and the development of diseases *like those* present in the plaintiffs." (528) (emphasis supplied). He opined that "to a reasonable degree of toxicological certainty, I can conclude that the presence of known carcinogens emanating from the DUMP in the form of landfill gases, leachate, ground water contamination and soil contamination have been the cause of a greater than usual cancer, Leukemia and Hodgkin's disease rate among neighbors to the landfill site. Specifically, the plaintiff's [sic] in this lawsuit." (528-29). Dr. Bidanset did not submit any data, scientific literature or other documents to support his opinion. Bidanset did not discuss whether any of the

[30] Such an 'expert' opinion, essentially indicating that he believed the chemical mix to be unique and not previously subjected to study, and for which there no relevant peer-reviewed scientific data, is inherently unreliable because it cannot be objectively tested, replicated or falsified.

[31] Dr. Bidanset cited none of this literature, however, so it is impossible to know whether his statement is accurate.

chemicals he listed have a biologically plausible association with ALL (*see* 525-26, 575-78), did not provide any measurements of exposure or dose estimates for any of the chemicals, and it did not consider whether particular exposures were sufficient to cause ALL. Bidanset did not specifically consider or discuss ALL, the primary disease at issue, and instead the affidavit was generic, speaking of 'the development of diseases like those present in the plaintiffs in this lawsuit.[32]

Neither the Bidanset or Trainor affidavits provided sufficient information to support an opinion regarding specific causation because they failed to follow the well-established standard scientific approach employed by toxicologists and environmental scientists to determine reliably whether chemicals have caused a particular disease in individuals, which involves a determination of: 1) whether the individual was exposed to a particular chemical or chemicals plausibly associated with the illness at issue; 2) the quantitative 'dose' of the chemical(s) that the person absorbed; and 3) whether that dose is capable of causing the specific illness (also known as 'dose-response' and 'biological plausibility'), ascertained by reference to the published literature and other scientific authority (Reference Manual at 419). The Bidanset and Trainor affidavits contain no information concerning how any of the

[32] The only reference to the disease at issue was the incomplete sentence: "Specifically, the plaintiff's [sic] in this lawsuit."

plaintiffs were exposed to any chemicals from the Landfill, whether any of the chemicals in the Landfill have been linked to ALL or Hodgkin's disease or whether the plaintiffs were exposed to any of these chemicals at doses sufficient to cause ALL or Hodgkin's disease. These deficiencies rendered their conclusions on whether the Landfill caused plaintiffs' illnesses scientifically inadequate to support a causation opinion.[33]

If general causation is not established, it is scientifically wrong to assert that specific cases of these diseases were caused by benzene or any other chemical. Such a relationship is simply unknown. Since general causation has not been proven, that should be the end of the story.[34]

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[33] Plaintiffs also submitted an affidavit of Philip Lanzkowsky, M.D., a pediatric hematologist/oncologist (626-27). Dr. Lanzowsky merely recited plaintiffs' diagnoses ("I can conclude to a reasonable degree of medical certainty that the plaintiffs were diagnosed with [ALL] and Hodgkin's disease" (627)) and, without offering any published studies or stating any link to the Landfill or plaintiffs' specific exposures, stated: "It has been known for almost half a century that Benzene is associated with bone marrow toxicity and that it plays a causative role in Leukemia." (627). As we explain infra, benzene has been proven, and is generally accepted, to cause only acute myelogenous leukemia (AML), and not other forms of leukemia such as ALL. Thus Dr. Lanzkowsky's opinion, while approximately (but not precisely) is not relevant to the issues in this case.

[34] "[A]n agent cannot be considered to cause the illness of a specific person unless it is recognized as a cause of that disease in general." Philip Cole, "Causality in Epidemiology, Health Policy and Law," 27 *Envtl L Rep* [Envtl L Inst] 10279, 10284, n. 53 (1997), quoted in Reference Manual, at 383-384).

ii. Specific Causation

Specific Causation goes further, and asks whether the specific conditions of exposure as are known to have been present in the particular case can cause the disease. In order to satisfy the specific causation test there must, in addition, have been enough exposure to the substance (which substance is already suggested by the general causation requirement) to show that the individual exposure (the dose), was sufficient to cause the cancer at issue.

To prove specific causation one should satisfy, through rigorous methodology, five criteria: 1. General causation, 2. dose response, 3. timing, 4. alternative cause, and 5. coherence.[35]

Specific causation demands a knowledge or reliable estimate of exposure and hence dose. In the case of the Landfill the "evidence" supporting the Plaintiffs' experts' testimony is insufficient as to both general and specific causation. Even if, *arguendo*, one were to accept the Neugebauer study as proof of general causation (which as shown above it is not) it would still not establish specific causation in any particular Plaintiff. To do that, one would have to know that exposure and dose were

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[35] *See* P.S. Guzelian, M. Victoroff, N.C. Halmes, R.C.James, C.P. Guzelian, "Evidence-based Toxicology: A Comprehensive Framework for Causation," 24 *Human and Experimental Toxicology* 161-201 (2005); P.S. Guzelian and C.P. Guzelian, "Authority-Based Explanation," 303 *Science* 1468-1469 (2004).

sufficient in the particular individuals. A fundamental tenet of toxicology is that the "dose makes the poison" and that all chemical agents, including water, are harmful if consumed in large quantities, while even the most toxic substances are harmless in minute quantities. *See* Reference Manual at 403. In determining whether Plaintiffs' exposure to chemicals in the Landfill could have caused the ALL or Hodgkin's Disease, it is necessary to establish the dose/response relationship between those chemicals and those particular illnesses. It is highly likely that the dose is smaller in this study than in any study – probably an occupational study – where the dose was higher and would have been used to establish general causation. Actual exposure of any kind or amount has never been shown in this case; it is not even known to have occurred at all. There is only speculation that such exposure occurred.

The Appellate Division majority wrote: "Dr. Trainor, one of plaintiffs' experts, identified at least four pathways (leachate, groundwater, soil, and air emissions) by which plaintiffs, and other individuals in proximity to the landfill, *could have been* exposed to those carcinogens." (32 A.D. 3d at 107) (emphasis added). This is a general statement of possible pathways; indeed, it could be a statement of pathways for any person in New York City.

There are no measurements of a postulated chemical exposure. Nor is there any exposure carefully calculated from some environmental model. Until there is a

measurement, or at least a calculation that even one of these pathways produced a significant concentration in proximity to any of the plaintiffs for a significant period of time, Trainor's statement has no operational utility and should be ignored. Because there is no established exposure, there is no established dose. If there is no established dose, there can be no established specific causation.

Knowledge of exposure, preferably by measurement, but if not by reliable calculation, is essential. On this scientists and this Court in *Parker* agree.[36] This can be illustrated by two situations standing in stark contrast to the present one. In studying the late cancer effects of exposures following the atomic bombing of Hiroshima, although it was not possible to measure the exposure *ex post facto.* Nonetheless very sophisticated modeling enabled reliable estimates to be made. At both Hiroshima and Nagasaki, experts could map radiation levels at various distances from the epicenters of the blasts and follow populations at various location with different exposure levels. This enabled researchers, after numerous studies, to reach conclusions about exposure-disease relationships.[37]

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[36] Respondents argue that there is no need to know the "exact level" of exposure (Respondents' Brief at 2). That misses the point – to show specific causation, a scientist must at least proffer a reliable calculation of exposure.

[37] This has been described in a series of papers by scientists at the Radiation Effects Research Foundation (RERF) in Hiroshima, Japan, available on the web at: http://www.rerf.or.jp/eigo/titles/radtoc.htm.

The same is true of the many studies that have routinely found a connection between cigarette smoking and lung cancer.[38] In both of these examples, exposure levels were known and multiple studies were consistent and positive. There is not even a suggestion that such data are available for the Landfill.

In discussing exposure, the majority in the Appellate Division stated that "...no scientist could make an *accurate* measurement of the doses of the combined carcinogens to which these plaintiffs were exposed." (32 A.D. 3d at 105) and "It is not surprising that plaintiffs' toxicologists did not present a specific dose-response threshold of any particular carcinogen to support their opinions that plaintiffs' cancer was caused by exposure to the landfill. Neither Dr. Bidanset nor plaintiffs' other expert toxicologist purported to establish a dose-response relationship between the large number of carcinogens in the landfill for over a decade and plaintiffs' cancers. Instead, plaintiffs have proffered a combination of epidemiological and toxicological reports to support the *theory* that their extended exposure to hazardous levels of numerous carcinogens in this particular landfill caused their cancers." (*Id*. at 107) (emphasis supplied).

[38] *E.g.*, IARC Supplement 7 at 359-362.

Amici stress the word "accurate" in this expression of the Appellate Division majority opinion. The Appellate Division majority notwithstanding, none of these reports make any attempt to show that the exposure, and hence dose, was anywhere near sufficient to produce cancer. Nor do they provide a theoretical calculation, or a model, proceeding from what is known to a estimate of dose. There is no mention of the obvious general statement that when pollutants are emitted at ground level with no hot chimney stack to point plumes upwards, then pollutant concentrations would be expected to fall with distance as the inverse square ($1/r^2$). For this reason it makes sense to perform a "proximity analysis" as a substitute for a dose response analysis. Did the plaintiffs live close to or far away from the landfill? Plaintiff's "experts" failed to do so, but the New York City experts showed that they were not unusually close.

To suggest, as the Appellate Division majority did, that a plaintiff does not need to provide an estimate of exposure (or dose) nor a dose-response finding, while perhaps charitable or noble, nevertheless obviates any evidentiary burden to show that the methodology used is sound, and thus fails to adhere to principles of science and this Court's precedents. The Appellate Division majority implies that proof of

causation is unnecessary and that merely the fact that people lived near the Landfill will suffice. Were that the law, anyone could commission a "study" to "prove" cause of disease related.

C. Weight of Evidence

Many international and national regulatory agencies, and some courts, use the expression "weight of evidence" to evaluate information. The motions and affidavits in this case consist of hundreds of pages. This is a daunting data set for the court to consider. The volume of paper pages might suggest that there is a likelihood that the plaintiffs are "right" and that there are mere factual or opinion disputes among experts, to be evaluated and weighed by the trier of fact. Nothing could be farther from the truth.

The term "weight of evidence" refers not to pounds of paper[39], but to quality, in this case the probative value, of the scientific support for the claim that one can actually connect the occurrence of specific cases of ALL and Hodgkin's disease to the Landfill. This involves the review of numerous studies to assess their strength, weaknesses, and consistency of findings. The term "weight-of-evidence" is used in

[39] As noted earlier, the majority in the Appellate Division seems to have been impressed that Plaintiffs' experts reviewed "mountains" of documents. The sheer volume of material reviewed does not give the resulting opinion credibility or reliability.

the scientific community to characterize a process or method in which all scientific evidence that is relevant is taken into account.[40]

A widely-accepted framework for such a process is the one suggested by Austin Bradford Hill.[41] The Hill criteria, modified and expanded by numerous other epidemiologists are used within the scientific community to assist in a determination of whether evidence in the scientific literature is sufficiently strong to indicate a causal relationship. The preamble to the IARC monograph series states their approach and explicitly mentions "weight of evidence." Of particular relevance to this case is the statement: "The uncertainties that surround the interpretation of case

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[40] S. Krimsky, "The weight of scientific evidence in policy and law," 95 Am. J. Public Health (Suppl. 1):S129-S136 (2005).

[41] Sir Austin Bradford Hill in his Presidential Address to the Section of Occupational Medicine of the Royal Society of Medicine (U.K.) proposed a list of "attributes" of the association to be considered in evaluating causation: 1. Strength; 2. Consistency; 3. Specificity; 4. Temporality; 5. Biological gradient or dose response relationship; 6. Plausibility; 7. Coherence; 8. Experiment; and 9. Analogy. "The Environment and Disease: Association or Causation?," 58 *Proceedings of the Royal Society of Medicine* 295-300 (1965). Hill emphasized that no one principle should be governing, but all should be considered. Earlier, Koch and Henle had proposed a similar series of criteria for making an epidemiological assessment of causation, known as "Koch's postulates": 1. Strength of association; 2. Temporal relationship; 3. Consistency of association; 4. Biologic plausibility (coherence with existing knowledge); 5. Alternative explanations; 6. Specificity of the association; 7. Dose-response relationship. Any person who claims to be an "expert" on medical causation should be familiar with these principles and should be able to demonstrate how his claim to causality fits each of them. *See also*, U.S. Dep't of Health, Educ., and Welfare, Public Health Serv., Smoking and Health: Report of the Advisory Committee to the Surgeon General (1964).

reports, case series and correlation studies make them inadequate, except in rare instances, to form the sole basis for inferring a causal relationship."[42]

The results of studies that are judged to be of high quality are given more weight than those of studies that are judged to be methodologically less sound. Factors used to judge the quality of a study include attention in the study design to potential biases, control of confounding factors,[43] statistical analysis to determine the role of chance in the interpretation of the study results, the size of the study population, and objective evidence of an exposure and a dose. One study, either of high or low quality, can almost never be sufficient to establish general causation. Were that not the case, thousands of chemicals would be on IARC's known human carcinogen list;[44] instead there are about 70.

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[42] Available at http://monographs.iarc.fr/ENG/Preamble/currentb2studieshumans0706.php

[43] Confounding is a problem even in careful epidemiologic studies. For example, women with herpes are more likely to develop cervical cancer than women who have not been exposed to the virus, and it was thought that herpes caused cancer. Later research suggests that herpes is only a marker of sexual activity and women who have had multiple partners are more likely to be exposed not only to herpes but also to human papilloma virus, which seems to cause cervical cancer, while herpes does not. *See* Reference Manual at 345, 349, 369-373; *see also* Richard Peto & Harald zur Hausen, eds., VIRAL ETIOLOGY OF CERVICAL CANCER (1986); N. Muñoz et al. eds., THE EPIDEMIOLOGY OF CERVICAL CANCER AND HUMAN PAPILLOMAVIRUS (1992); D. Freedman, "From Association to Causation: Some Remarks on the History of Statistics," 14 *Stat. Sci.* 243 (1999).

[44] A thousand chemicals or more have at least one study showing at least one positive association of one form of cancer with exposure to that chemical.

Because the phrase "weight of evidence" has been used erroneously to describe little more than the "weight of the paper" containing the opinions of presumed experts,[45] scientists are beginning to use the phrase "evidence based," a phrase well understood in medicine, to replace the term.[46] This puts emphasis on a rigorous analysis rather than subjective opinion.

When applying a weight-of-evidence approach, one is looking at the totality of available studies. Studies vary widely in their method and their quality. Often, even very well-performed studies contradict one another. This why we hear the "cancer scare of the week" followed by retractions or silence when other studies are

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[45] The definition of "weight of the evidence"is unclear. Douglas L. Weed of the National Cancer Institute undertook a systematic review of the scientific literature for the years the ten years 1994 through 2004 and found that WOE was used in three different ways in the literature: (1) metaphorical, where WOE refers to a collection of studies or to an unspecified methodological approach; (2) methodological, where WOE points to established interpretative methodologies (e.g., systematic narrative review, meta-analysis, causal criteria, and/or quality criteria for toxicological studies) or where WOE means that "all" rather than some subset of the evidence is examined, or rarely, where WOE points to methods using quantitative weights for evidence; and (3) theoretical, where WOE serves as a label for a conceptual framework. Weed concluded that there were several problems with the use of the term, including frequent lack of definition of "weight of evidence," multiple uses of the term, a lack of consensus about its meaning, and the many different kinds of weights, both qualitative and quantitative, which can be used. D.L. Weed, "Weight of Evidence: A Review of Concept and Methods," 25 *Risk Analysis,* 1545-1557 (2005)

[46] *See* D.J. Friedland, J.B. Davoren, A.S Go, Evidence-Based Medicine: A Framework for Clinical Practice (1998); D.L. Sackett, S. Straus, S. Richardson, W. Rosenberg, R.B. Haynes, Evidence-Based Medicine: How to Practice and Teach EBM (2nd ed. 2000); D.L. Sackett, R.B. Haynes, P. Tugwell, "Deciding Whether Your Treatment Has Done Harm," in D.L. Sackett, R.B. Haynes, P. Tugwell, eds., CLINICAL EPIDEMIOLOGY: A BASIC SCIENCE FOR CLINICAL MEDICINE (1985).

not confirmatory. The association between coffee drinking and pancreatic cancer is illustrative: approximately a dozen studies have examined this relationship; at least ten were well-designed, competently performed, and inconsistent with one another in their findings. The first case control study was apparently flawed due to improper selection of the control subjects, but it brought a flurry of news reports warning the public about coffee consumption. After others were carried out and the results published, the connection became far less clear and, today that relationship, seen in some studies, but not others, is not generally accepted.[47] What is more, as with the Hiroshima and cigarette smoking examples, in those studies exposure and dose were relatively well ascertained. In this case the exposure and dose are not known.

Also, in this case there are two studies which contradict Neugebauer's conclusion and which provide the underlying data (or at least references to the source of that data) and the methodology. In 1988, the New York City Department of Health ("NYCDOH") Environmental Epidemiology Unit conducted an epidemiological study to determine the incidence of childhood leukemia among residents near the Landfill and whether there was a possible disease cluster in the neighborhoods surrounding the Landfill (760, 763, *see* 757-98). The study, "An Evaluation of

[47] The coffee-pancreatic cancer episode is described in detail in the Reference Manual at 370-372.

Childhood Leukemia in the Pelham Bay Area of the Bronx," analyzed the number of all types of leukemia combined, which includes ALL (a subtype of leukemia), in the Pelham Bay area as reported to the New York State Cancer Registry from 1974 until 1985 (id.). The 1988 NYCDOH study concluded that there was no evidence of an increased incidence of childhood leukemia during this period when compared to the rates of childhood leukemia in the City as a whole (761, 785).

In January 1994, NYCDOH issued a second study, "Cancer Incidence in the Pelham Bay Area of the Bronx," and issued an addendum in 1996 (751, 799-896y). This Study, based on cancer cases reported to the State's Cancer Registry from 1978 to 1987, examined the rates for all types of cancer combined and for 13 specific types of cancer in adults and three types of cancer in children (*id*.). The 1994 Study concluded that, for both adults and children, the incidence of total cancers and most specific types of cancer in the Pelham Bay area was consistent with, and similar to, rates for the City as a whole, and consistent with the earlier study (822, 805, 751). There were no sites for which there were significantly more cases of childhood cancer observed than expected for the 10 years period (805). Proximity analysis found no pattern for leukemias or all forms of cancer in children (805, 751). The 1994 study found statistically significant increases in four forms of cancer (colon cancer and leukemia in adult men, lung cancer in women, and kidney cancer in both men and

women, none of which cancers are not at issue in this case[48]), the 1994 study found no pattern consistent with exposure from the Landfill and the four forms of cancer which had elevated levels had no common mechanisms or exposures (751, 808, 822-23 808, 896e).

D. <u>The Quality and Relevance of Specific Studies</u>

To suggest that a type of study, in this case an "epidemiological study," is generally accepted and, therefore, satisfies all logical requirements in this matter, would be a meaningless statement, and improper, if made by itself. The proper question is whether this study is accepted as a proper epidemiological study? Calling something an "epidemiological study" and arguing from that to "generally accepted" is erroneous. The next question would be accepted for what purpose? It might be merely a "hypothesis generating study," only of use to *generate* a hypothesis for study for a future blind study, but of no use whatsoever in *proving* an hypothesis. That would be like saying, "because experiments are generally accepted scientific approaches, the results of any experiment should be generally accepted for any purpose."

[48] None of the plaintiffs has any of these four forms of cancer (751). All are children suffering from ALL or Hodgkin's disease only (751).

The following study illustrates this. In a published peer-reviewed study[49], the investigators were asked to evaluate a cluster of leukemias associated with residential proximity to a benzene-emitting coke oven. In 1989-1996 there were 12 cases of leukemia in close proximity to that source, while 3.4 cases were expected based on the population. Thus, there was a statistically significant greater incidence of leukemia in the "exposed" group. However, the investigators knew the levels of benzene in the air, which was quite low, and they modeled, using the United State EPA cancer risk assessment model, the cancer risk at those levels. They found that there was no increased risk from that low level exposure and concluded, therefore, that the cases, more probably than not, represented an associated cluster, rather than a group whose cancer was caused by the exposure. The conclusion of that paper was:

> The excess occurrence of leukaemia in the Warrawong area in 1989-1996 is highly unusual. Current environmental benzene exposure and the reconstructed past environmental benzene exposure levels are too low to explain the large excess of leukaemia. The cause of the cluster is uncertain.

The United States Centers for Disease Control and Prevention ("CDC") have developed advice concerning Cancer Clusters[50]:

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[49] V.J. Westley-Wise, B.W. Stewart, I. Kreis, P.F. Ricci, A. Hogan, C. Darling, S. Corbett, J. Kaldor, N. H. Stacey, and P. Warburton, "Investigation of a cluster of leukaemia in the Illawarra region of New South Wales, 1989-1996," 171 *Medical Journal of Australia*, 178-183 (1999).

[50] http://www.cancer.gov/cancertopics/factsheet/Risk/clusters

> The complex nature of cancer makes it inherently challenging to identify, interpret, and address cancer clusters.
>
> Cancer in general is common. In the U.S., 1 in 3 people will develop cancer in his or her lifetime. According to the American Cancer Society's Cancer Facts and Figures 2005 about 1,372,910 new cancer cases are expected to be diagnosed in 2005.
>
> Cancer rates vary by age, race, gender, risk-factors, and type. We know that risk for cancer increases with age and that cancer is caused by both external factors (e.g., tobacco, chemicals, radiation, and infectious organisms) and internal factors (e.g., inherited mutations, hormones, immune conditions). Nutrition, physical inactivity, obesity, and other lifestyle factors also play a role in cancer risk and outcomes. These factors may act together or in sequence to initiate or promote cancer. Ten or more years often pass between exposures or mutations and detectable cancer.
>
> Some racial and ethnic groups have a higher incidence of and deaths due to cancer. Such disparities may be due to multiple factors, such as late stage of disease at diagnosis, barriers to health care access, history of other diseases, biologic and genetic differences, health behaviors, differences in exposures to carcinogens in the environment and the workplace, and other risk factors.

What may first appear to be a cancer cluster may not be one after all. A review of the situation may show that the number of new cancer cases is in the expected range for the population and therefore that the cases do not represent a cancer cluster. Cancer cases are more likely to represent a cancer cluster if they involve (1) a single type of cancer, (2) a rare type of cancer, or (3) a type of cancer in a group not usually affected by that cancer, such as a cancer in children that is normally seen in adults.

However, cases of common cancers are those most often perceived and reported by the public as being part of a cancer cluster.

Moreover, confirmation of a cancer cluster does not necessarily mean that there is any single, external cause or hazard that can be addressed. A confirmed cancer cluster could be the result of any of the following:

1. chance;
2. miscalculation of the expected number of cancer cases (e.g., not considering a risk factor within the population at risk);
3. differences in the case definition between observed cases and expected cases;
4. known causes of cancer (e.g., smoking);
5. unknown cause(s) of cancer.

Similar criteria were discussed by Neutra[51] . Very few cancer clusters are found to be causal when properly discussed by these criteria. The best known is the cluster of mesotheliomas in the Turkish village of Karain[52]. In that village

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[51] R.R. Neutra, "Counterpoint from a Cluster Buster," 132 *American Journal of Epidemiology* 1-8 (1990). Neutra called the procedure "Distinguishing giants from windmills." *Amici* urge the court not to follow Don Quixote and tilt against the windmills.

[52] I. Baris, L. Simonato, M. Artvinli, F. Pooley, R Sarracci, J. Skidmore and C. Wagner, "Epidemiological and Environmental Evidence of the Health Effects of Exposure to Erionite Fibers: a Four Year Study in the Cappadocean Region of Turkey," 29 *International Journal of Cancer* 10-17 (1987).

natural outcroppings of fibrous erionite had been used by the villagers for paint and other purposes. The risk of cancer was 1,000 times normal. The Neugebauer study about what would be (at best) a cancer cluster around the Pelham Bay landfill came nowhere close to such reliability.

As noted above, Plaintiffs failed to address either of the two generally recognized and accepted components to causation assessment: neither "general causation" – the determination that a chemical or group of chemicals is capable of producing the disease(s) at issue, nor specific causation – the determination that the agent(s), known to have such a capability, did, in fact, cause the diseases in these particular Plaintiffs.[53] In this case, neither requirement is fulfilled.

Experts commonly extrapolate from existing data. But nothing in either requires a court to admit opinion evidence which is connected to existing data only

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[53] The reply affidavits of Bidanset (1025-27), Trainor (1086-91), and Neugebauer (1065-84) failed to address or correct the fundamental methodological shortcomings of their "analyses" and conclusions. The Trainor and Bidanset opinions rely on assumed exposures, without any evidence of dose, and were, therefore based on reasoning that was not methodologically sound. The Neugebauer reply affidavit again failed to provide sufficient information about his methods and analysis; Neugebauer also failed to provide confidence intervals for his comparisons, which is contrary to generally accepted scientific methodology. A confidence interval is a range of values calculated from the results of a study, within which the true value is likely to fall; the width of the interval reflects random error. It provides the magnitude of the association found in the study or an indication of how statistically stable that association is. A confidence interval for any study shows the relative risk determined in the study as a point on a numerical axis. It also displays the boundaries of relative risk consistent with the data found in the study based on one or several selected levels of statistical significance. *See* V. M. Brannigan, *et al.*, "Risk, Statistical Inference, and the Law of Evidence: The Use of Epidemiological Data in Toxic Tort Cases," 12 *Risk Analysis* 343, 344-345 (1992).

by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered. Reference Manual at 14-15 (2000 ed.)

II.

PLAINTIFFS' "SCIENTIFIC" EVIDENCE WAS UNRELIABLE, INADMISSIBLE AND LEGALLY INSUFFICIENT TO AVERT SUMMARY JUDGMENT

This Court should articulate a test for determining the admissibility of scientific expert testimony that ensures that causation evidence is reliable and genuinely scientific. *People v. Wesley,* 83 N.Y.2d 417 (1994); *People v. Wernick,* 89 N.Y.2d 111(1996); *Frye v. United States*, 293 Fed. 1013 (D.C. Cir. 1923).

When expert scientific testimony is necessary to establish the causal connection between plaintiff's alleged injury and defendant's product or conduct, the decision whether to admit or exclude such evidence is of critical importance. (*People v. Wesley, supra*; *People v. Wernick, supra*; *People v. Angelo,* 88 N.Y.2d 217 (1996); *Lara v. New York City Health & Hosps. Corp.,* 305 A.D.2d 106 (1st Dept. 2003); *Frye v. United States, supra*; *Daubert v Merrell Dow Pharmaceuticals, Inc.,* 509 U.S. 579 (1993); *General Electric Co. v. Joiner,* 522 U.S. 136 (1997). As this Court noted in *Parker* at 447, "As with any other type of expert evidence, we recognize the danger

in allowing unreliable or speculative information (or "junk science") to go before the jury with the weight of an impressively credentialed expert behind it."

The inquiry as to the scientific reliability of expert evidence goes to the admissibility of that evidence, not simply its weight, and is thus not a question for the jury. *See People v. Wesley*, 83 N.Y.2d at 425, *Parker*, 7 N.Y.3d at 447. As this Court held in *Parker*, the admissibility question that applies to all evidence -- whether there is a proper foundation -- is to determine whether it is reliable. *Id*. In the case of scientific evidence, the question is whether the accepted methods were appropriately employed to reach the conclusion in a particular case. *Wesley*, 83 NY2d at 429. "The focus moves from the general reliability concerns of *Frye* to the specific reliability of the procedures followed to generate the evidence proffered and whether they establish a foundation for the reception of the evidence at trial." *Parker* at 447, quoting *Wesley*, 83 NY2d at 429.

This is consistent with the general rule that courts must exclude an expert's opinion if it is not based on any supporting evidentiary foundation, facts or data. *See, e.g., Buchholz v. Trump 767 Fifth Avenue, LLC*, 5 N.Y.3d 1, 11 (2005); *Franchini v. Palmieri*, 1 N.Y.3d 536, 536 (2003); *Diaz v. New York Downtown Hospital*, 99 N.Y.2d 542, 544 (2002); *Romano v. Stanley*, 90 N.Y.2d 444, 451-52 (1997).

To defeat a motion for summary judgment in a case requiring expert proof, the expert's opinion must be predicated upon reliable facts and data. (*Alvarez v. Prospect Hosp.,* 68 N.Y.2d 320 (1986) (opponent must present evidentiary facts); *Amatulli v. Delhi Constr. Corp.*, 77 N.Y.2d 525 (1991) (unsupported conclusory allegations of plaintiffs' expert insufficient to raise triable issue of fact); *Romano v. Stanley,* 90 N.Y.2d 444, 451-452 (1997) (expert's affidavit proffered as the sole evidence to defeat summary judgment must contain sufficient allegations to demonstrate that the conclusions it contains are more than mere speculation and would, if offered alone at trial, support a verdict in the proponent's favor.)

To prove causation under New York law, a plaintiff must refer "not only to court opinions, but texts, laboratory standards or scholarly articles, as well, in an effort to determine whether a particular concept has been generally accepted by the relevant scientific community." *See Wesley*; *Lewin v. County of Suffolk,* 18 A.D.3d 621 (2nd Dept 2002); *DeMeyer v. Advantage Auto,* 9 Misc.3d 306, 315 (Sup. Ct. Wayne Co. 2005). As in *Parker*, Plaintiffs' experts in this case failed to identify a single epidemiologic study finding an increased risk of ALL or Hodgkin's Disease as a result of exposure to the chemicals in the Landfill. In fact, *amici* have found no scientific studies, reports in the literature, or other court judgments which suggest that

occupational or residential exposure to the chemicals contained in the Landfill cause ALL or Hodgkin's Disease.

From both the perspective of the relevant fields of science and the law, an opinion on medical causation must be based on Plaintiffs' exposures to a toxic chemical, that the chemical is capable of causing the particular illness (*i.e.*, general causation), and each Plaintiff was sufficiently exposed to the toxin to cause the illness (specific causation). *Parker* at 448; *Heckstall v. Pincus,* 19 A.D.3d 203, 204 (1st Dept 2005).

The expert opinions proffered by Plaintiffs do not state the level of exposure necessary to cause the diseases at issue, nor do they specify that Plaintiffs' exposure reached that critical dosage. Plaintiffs lack epidemiological evidence to support their claims. The affidavits submitted by Plaintiffs in this case are very similar to the submissions of Dr. Landrigan, one of plaintiff's experts in *Parker*, soundly rejected by this Court. *Parker, id.* While it is often difficult or impossible to quantify a plaintiff's exposure to a toxin with numerical precision, Plaintiffs' experts must demonstrate that exposure to particular chemicals in the Landfill caused the ALL and Hodgkin's disease diagnosed. As in *Parker*, the general, subjective and conclusory assertions by Plaintiffs' experts, not based on any thorough empirical examination of

the Plaintiffs or the Landfill, and not based on a thorough examination of the scientific literature, "is plainly insufficient to establish causation." *See Parker* at 449.

The expert opinions proffered by Plaintiffs in this case do not establish general causation, and do not even state the level of exposure necessary to cause the diseases at issue, nor do they specify that each of Plaintiffs' exposure received that level. Plaintiffs also lack reliable epidemiological evidence to support their claims.

It is palpable that in this case the Appellate Division majority was acutely sympathetic to the Plaintiffs, as are *amici*, but that sympathy for a claimant is not a substitute for evidence, and should not eliminate the requirement in tort law that the plaintiff prove causation -- that is, that the alleged cause was more likely than not to be the actual cause of the injury.[54] Sympathy cannot and should not displace a proper regard for the truth and for principles which are designed to ensure a just result when fault is to be found and liability is imposed on those plaintiffs seek to hold responsible for their ill fortune.

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[54] *See, e.g., In re Agent Orange Prod. Liab. Litig.,* 611 F. Supp. 1222, 1249 (E.D.N.Y. 1985), *aff'd* 818 F.2d 187 (2d Cir. 1987), *cert. denied* , 487 U.S. 1234 (1988).

CONCLUSION

Amici are convinced that the trial court and the Appellate Division misconstrued the necessity for the court to determine whether proffered expert evidence is reliable, that had those lower courts correctly understood the scientific principles involved, they would have found that Plaintiffs' expert opinions lacked foundation, and therefore they erred in denying the defendant's motion for summary judgment dismissing the cases. The decisions below should be reversed.

April 20, 2007

Respectfully submitted,

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Nonnon v. City of New York

***Amici* Biographical Addendum**

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PATRICIA A. BUFFLER, Ph.D. is Professor of Epidemiology at the School of Public Health of the University of California at Berkeley, and is a former Dean of the School of Public Health of the University of California at Berkeley. Among many honors and activities in the field of epidemiology, Dr. Buffler is a Fellow of the American College of Epidemiology, and was President of that organization in 1991-1992. She is a member of the Institute of Medicine of the National Academy of Sciences and a Fellow of the American Association for the Advancement of Science.

RONALD E. GOTS, M.D., Ph.D. specializes in toxicology and environmental medicine, and is board certified in toxicology. He is Principal of the International Center for Toxicology and Medicine and Medical Director and President of the National Medical Advisory Service. He is also Lecturer in and Adjunct Professor of Pharmacology, Department of Pharmacology, Georgetown University School of Medicine. He has been Coordinator, Pharmaceutical Class Labeling Project, of the U.S. Food and Drug Administration, Medical Director and Examining Physician of the Occupational Health Units, Bureau of Economic Analysis, Census Bureau and Immigration and Naturalization Service, Senior Investigator/Chief, Department of Gastroenterology, Walter Reed Army Institute of Research. He was Conference Chair of a conference on "Multiple Chemical Sensitivities: State-of-the-Science Symposium" co-sponsored by the International Society of Regulatory Toxicology and

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PHILIP GUZELIAN, M.D. is Professor of Medicine at the University of Colorado Health Sciences Center, Denver, Colorado and Chief of the Section of Medical Toxicology. For the 17 years before moving to Colorado, he was Professor of Medicine in the Departments of Internal Medicine, Pathology, and Pharmacology & Toxicology at the Medical College of Virginia/Virginia Commonwealth University in Richmond, Virginia. He received his M.D. degree from the University of Wisconsin at Madison in 1967 and trained at the Cleveland Metropolitan General Hospital, the National Institute of Child Health and Human Development, National Institutes of Health; he was United States Public Health Service Clinical Fellow in Liver Disease at Yale University, New Haven, Connecticut and United States Public Health Service Liver Research Fellow in the Gastrointestinal Unit at the University of California, San Francisco, California, from 1972 to 1974. Dr. Guzelian has authored or co-authored over 150 abstracts, peer-reviewed articles, or book chapters in the area of toxicology, with a major emphasis on the effects of chemicals on the liver. He is board-certified in Internal Medicine and is a member of the Society of Toxicology, and the Association of American Physicians. He has served on such

national committees as the National Academy of Sciences Committee on Toxicology, the United States Environmental Protection Agency (U.S. EPA) Science Advisory Board, the National Advisory Environmental Health Sciences Council of the National Institutes of Health (NIH) and of the Threshold Limit Value (TLV) Committee of the American Conference of Governmental Industrial Hygienists (ACGIH). Because of his accomplishments, both in medicine and in basic science and toxicology, Dr. Guzelian received the 1984-1989 Burroughs Wellcome Toxicology Scholar Award given through the Society of Toxicology.

LEONARD D. HAMILTON, M.D. is Professor of Medicine at the State University of New York at Stony Brook (since 1968) and Adjunct Professor of Biometry and Epidemiology at the Medical University of South Carolina at Charleston. He was formerly Head of the Biomedical and Environmental Assessment Group at the Brookhaven National Laboratory. He received his doctorate in medicine from Oxford University and a Ph.D. in Experimental Pathology from Cambridge University. He has been a Diplomate of the American Board of Pathology in Hematology since 1966.

LAWRENCE S. LESSIN, M.D. received his Doctorate in Medicine in 1962 from the University of Chicago School of Medicine, where he was elected to the Alpha Omega Alpha Medical Honorary Society. Following medical school, he

trained (residency in Medicine and fellowship in Hematology) at the Hospital of the University of Pennsylvania, where he served as Chief Medical Resident. He is certified by the American Board of Internal Medicine in Hematology, Medical Oncology and Internal Medicine. Dr. Lessin was awarded a special fellowship from the National Heart Institute (United States Public Health Services) in the Institute for Cell Pathology at the University of Paris, for advanced research training in recognition of his research accomplishments and innovation. Dr. Lessin has participated in the training of over 75 Fellows and research personnel in Hematology and Medical Oncology, as well as countless medical students and residents. For more than 35 years he has been a Professor of Medicine, Division Head and Medical Director at prominent medical centers, including Duke University Medical Center (Durham, N.C.), Veterans Administration Hospital (Durham, N.C.), and The George Washington University Medical Center and its Cancer Center(Washington, D.C.). In 1993, Dr. Lessin became the Medical Director of The Washington Cancer Institute at Washington Hospital Center, which has become a leader in comprehensive cancer care under his leadership. Dr. Lessin established the interdisciplinary team care model for treatment and support of cancer patients and helped to recruit top physicians to lead these programs. Over the past decade he has helped to develop an extensive research program, now conducting over 40 clinical trails per year and

bringing cutting edge therapies to over 200 patients annually. Dr. Lessin has a national reputation as a medical educator, cancer program administrator and provider of high quality cancer care in control, diagnosis and treatment of cancer and related blood disorders. He is the author, co-author or editor of books and articles in more than 120 national and international publications, books and journals. He has severed on numerous national boards and committees for organizations such as the National Heart Lung Blood Institute, the National Cancer Institute, the American Society of Hematology, the American Society of Clinical Oncology, the American College of Physicians, the American Blood Commission and others. He served for six years as a member of the American Board of Internal Medicine, subcommittee on Hematology. In 1999, he was awarded Mastership in the American College of Physicians. He has been repeatedly recognized as one of the Best Doctors in the United States of America, Best Medical Specialists in the United States, and Who's Who in America.

SALLY L. SATEL, M.D. is resident scholar at the American Enterprise Institute. She is also lecturer at Yale University Medical School, and was assistant professor of Psychiatry at Yale Medical School from 1988 to 1995. She was also a visiting research scientist at the University of Pennsylvania Medical School. Dr. Satel has written numerous monographs and articles on drug treatment, the

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RICHARD WILSON is Mallinckrodt Research Professor of Physics at Harvard University and immediate past Director of the Regional Center for Global Environmental Change at Harvard University. He is an Affiliate of the Center for Science and International Affairs and the Center for Middle Eastern Studies at Harvard University. Professor Wilson is a past Chairman of the Department of Physics at Harvard University, a past chairman and currently a member of the Cyclotron Operating Committee. He is a founder of the Society for Risk Analysis. He is and has been a consultant to the United States government and the governments of numerous foreign countries on matters of toxicology, epidemiology, public health and safety, nuclear safety, and risk assessment. Professor Wilson's areas of expertise include elementary particle physics, radiation physics, chemical carcinogens, air pollution, ground water pollution by arsenic, and human rights. He is the author of many articles on high energy physics, environmental pollution and risk analysis, including PARTICLES IN OUR AIR, EXPOSURES AND HEALTH EFFECTS (with Editor

John Daniel Spengler) (Harvard University Center for Risk Analysis, 1986) and RISK-BENEFIT ANALYSIS (with Edmund A. C. Crouch) (Harvard University Center for Risk Analysis, 2nd ed. 2001). Professor Wilson is the author or co-author of more than 880 published papers on subjects including atomic particles, radioactive particle decay, acute toxicity and carcinogenic risk, carcinogenicity bioassays, statistical distributions of health risks, public health, cancer risk management, shielding of particle accelerators and nuclear reactors, nuclear energy production, health risks of nuclear power plant accidents, health effects of electromagnetic fields, risks and health impacts of radiation, risks of nuclear proliferation, risk benefit analysis, and global energy use and global warming.