

TRANSCRIPT (edited for clarity)

American Society for Environmental History, Toxic Environments/Toxic Bodies Workshop, Baton Rouge, Louisiana, March 2, 2007. Morning Session #1, Endocrine Disruptors and Biomonitoring: Scientific and Historical Perspectives in Dialogue.

NANCY LANGSTON: Welcome to “On the Edge on Uncertainty: Biomonitoring, Body Burden and New Twenty First Century Bodies.” This morning will be devoted to a roundtable discussion among environmental scientists, science and technology studies scholars, and historians, who will be reflecting on biomonitoring and changing toxicological frameworks. We will address the profound uncertainty that these new ideas are bringing to us. We’ll talk about ways that historical examinations of chemicals and of toxicology might actually help us devise more effective policy, and more effective ways of responding to these new chemicals. We’ll also talk about specific historical examples that might be helpful in thinking about this.

Then in the roundtable after the break we’ll focus on questions of communities:

- the ways that scholars, affected communities. and scientists can all work together
- what we have to offer each other
- how we can communicate better
- the ways we know what we know and how that might transform the world.

I’ll introduce people very briefly. I’m Nancy Langston, about to be president of American Society for Environmental History. My work focuses on endocrine disruptors and the kind of radical uncertainty that leaves in our own bodies, and I focus my work on the nineteen thirties and nineteen forties.

Jody Roberts is the 2006 to 2007 Gordon Cain Fellow at the Chemical Heritage Foundation. He has his Bachelor’s in chemistry and his PhD and Masters in science and technology studies.

John McLachlan (I think probably needs no introduction to most of you) is the Weatherhead Distinguished Professor of Environmental Studies at Tulane and the director of the Tulane -Xavier Center for Bioenvironmental Research. He actually received his Bachelor’s in liberal arts from Johns Hopkins and then a doctorate degree in pharmacology from George Washington University and for two decades he was at the National Institute of Environmental Health Sciences, one of the leading center for environmental health research., which he directed before he came down here in 1995. He has done some of the really truly groundbreaking work on diethylstilbestrol—DES—and on endocrine disruptors in general, and he’s also been extraordinarily involved with communities as well. He received the Hero of the Year Award from the Breast Cancer Fund in 2000,

Sara Vogel is doctoral candidate at Columbia University in the department of sociomedical sciences program. She holds a Masters from Yale in public health and another in environmental management, and she’s working on endocrine disruptors, bisphenols, pollution and production , focusing in the nineteen seventies

Tyrone Hayes is professor of integrative biology at University of California, Berkeley. He studies hormones and sexual differentiation in amphibians both in the US and in Africa. He has become quite well known for some of his research both on basic endocrinology and also on applied work looking at the affects of atrazine on amphibian development. On his website he said, “Tyrone Hayes began as a boy who loved frogs,” and today he hopes to shed light on human problems ranging from infertility to hormone implants diseases so I love the frogs but thank you so much for coming.

Arthur Daemmrich is currently director of the Center for Contemporary History and Policy at the Chemical Heritage Foundation and he works on regulatory policies—politics and history. Starting in July he will be assistant professor at Harvard Business School studying regulation.

Jody will get us started off. We will have brief comments for about ten minutes each then we’ll have some time for discussion. After the break—another short roundtable and then we hope to have at least forty-five minutes for a very broad ranging discussion. Thank you, Jody.

JODY ROBERTS: I think actually if it’s all right—unless people can’t hear me—I’ll stay here—I don’t have any slides or anything fancy and I want to make sure to save as much time as we can for some of the other presentations and a lot of discussion. I just have a few comments that I’ll try to stick to just so I don’t wander off on too many tangents, but I want to thank everyone for coming and thank Nancy for all of her help—and all of the work that she’s been able to do, to bring us to this via ASEH. I thought I’d speak a little bit about how I came to these converging issues of biomonitoring—body burden and toxicology—endocrine disruptors—public health and environment before saying a little about what I think we might be headed for from here.

As a graduate student in science and technology studies at Virginia Tech I had the opportunity to hear a lecture by Tyrone Hayes on his work on the effects of low dose atrazine exposure to frog populations globally. I was astounded, and over the next couple of years the topic of environmental endocrine disruptors stayed with me as a topic that seemed to be one that required immediate attention, but from the perspectives of STS I wasn’t quite sure what that meant I could do. During the course of my doctoral work on green chemistry I kept running into traces of the environmental endocrine disruptor issue in people in institutions and in situations where it was very clear that it wasn’t being discussed very purposefully.

As a fellow at the Chemical Heritage Foundation, I found myself presented with a very unique opportunity. First of all, I had the opportunity to leave my dissertation work behind for a little while, which is nice as soon as you have the dissertation done to think about what might come next. As an added bonus, I also had the opportunity to organize a conference around whatever research topic I might find interesting for the year, which also poses its own

problems in organizing a conference around research you haven't quite done and getting that done in nine months. I knew I wanted to find some way to revisit and explore the issues of environmental endocrine disruptors that I had come across before from the perspective of how these issues and developments were creating an alternate landscape for the chemical community—that is, how were chemists in the chemical sciences being forced to rethink some of their own work in light of what was happening in endocrinology and toxicology. Somewhere in and around this time, I met Arthur Daemrich, who introduced me into the topic of human biomonitoring or the analysis of human liquids and tissues—normally blood and urine—for the presence of synthetic chemicals. The topic has pretty much consumed me ever since.

Over the past year I've been working to integrate the various themes and topics into what I've been calling—at least for the purposes of my upcoming conference at the end of March—our “new chemical bodies,” because after all that's precisely what we're dealing with here. The physical experience of our world normally overshadows what is our more intimate contact with our environment. We forget—or perhaps never quite realize—that we are large chemical structures who interact with the chemical world around us at every moment. Breathing—smelling—eating all bring us into intimate contact with our surroundings, but these surroundings have been changing in recent decades in ways—at least as far as we can tell—we never before imagined. New chemicals enter the world each year by the thousands. We've become accustomed in recent decades the existence of these chemicals in the air, soil and water) and perhaps we're even okay with them being inside of other organisms. But we're wholly unprepared to be thinking about these chemicals in our own bodies, as we're also learning that these chemicals might be sticking around inside of the organisms in which they enter, including humans.

As the CDC prepares to release its fourth annual—or fourth report—on human exposure to synthetic chemicals later this year, a host of problems confront us, which is part of the reason I wanted to organize this workshop and part of the reason I'm hosting this conference at the end of the month. Because a lot of the participants here will be discussing a lot of the issues that I'm concerned with, I don't want to go on with them too much, except to think about what are some of the pressing questions that I see coming out of this that we might begin to talk about as a group and discuss. So, I'll just talk about two.

First, human biomonitoring has its roots in occupational health of roughly a century ago when workers when dealing with lead, for instance, were routinely tested as a way of managing the workers health. My assumption up to this point—and I hope other people will correct me if I'm wrong on this—is that something happened with the birth of the more formal environmental movement. Whereas occupational health dealt very explicitly with bodies in very clear environmental situations, these two topics were bifurcated, and we began to think about the environment on one side and public and human health on the other. If I'm right

that something like this has happened, then what will happen in recent advancements in human biomonitoring coupled with that related to environmental endocrine disruptors mean for the future of this parsing? My hunch is that in a time when many are already questioning the direction the environmental movement, that human biomonitoring studies will once again put bodies back into environments and we will be forced to start thinking—linking the research that we have been conducting on environmental toxins over the past three decades with not only what it means inside of these larger ecosystems, but these ecosystems will once again have to confront having humans inside of them.

My second kind of question that I had put out for the folks here is at the same time that these new studies are coming out on human levels of synthetic chemicals, serious questions are being asked within fields such as endocrinology and toxicology about how organisms may react to these chemicals that are being found so pervasively in the world. Other panelists here will discuss some of these issues in greater detail but for the purposes of discussion I'll ask here—what will happen with a new low-dose framework? That is, how do we begin to deal with chemical toxicity when parts per billion—parts per trillion and perhaps even parts per quadrillion are increasingly measurable on the one hand but also seen as potentially harmful? How will regulatory systems need to be reconceived? How does one remediate as we normally do with places where pollution is prevalent? How do we remediate bodies and landscapes when such miniscule amounts of a chemical can potentially cause harm? So, these are some of the questions that I'm thinking about that—that led me into wanting to—to hold this workshop. You know, we are very excited about having a wide perspective on some of this who can speak about them—both the very technical aspects but also how we deal about these issues in terms of environmental justice—in terms of informing communities—etcetera so, thank you.

NL: Thank you. John—

JM: For ten minutes what I'll do is talk about three different points of histories. I'm especially delighted to be here because I like to think in historical context and perspectives about what it is we're doing and what I'm doing, and so to actually have professional historians to talk with and consult with is really exciting for me. I hold this up because even though I'm talking about history, my own ability to keep up with my thoughts for more than thirty or forty seconds is really deteriorating so I always hold up fingers for myself to try to remind myself what are the three points I was going to make or how many points it is that I do have to make. There are three and I hope when I get to the third I'll remember what the little finger's all about.

So, there are different levels of history obviously. The three points are historical in different ways. I'm a basic scientist who studies cellumolecular biology of hormones and I've actually lived a lot of the history that I'll be talking about except my third finger is going to be talking about evolutionary history and

I have not been around for the last four hundred million years, but I think that that evolutionary history helps us understand why so many chemicals in the environment work like the female sex hormone estrogen—so that's one kind of history we'll have to talk about.

The second kind is the fact that time, as it goes on, gives us a whole new set of things to know and to think about, and what's happened in this field of endocrine disruption or environmental hormones or even endocrinology has been remarkable. Having lived some of the history myself, what I'm going to talk about on my second point is the fact that things that we did in laboratory experiments in the nineteen seventies and nineteen eighties and tried to project to human beings but we really couldn't do it because the human beings we were looking at were only twenty-four years old or thirty-two years old and we had two-year-old mice that had tumors or had some other—they had early menopause as a mouse. Now those same things are happening forty years later we're finding that history playing out in humans, so that's another kind of remarkable sort of issue when you're looking—as you said—at low-dose effects or things that are distributed ubiquitously throughout the environment if there are developmental or time dependent changes how do you even conceive of that and we're struggling in New Orleans with how do we conceive of a recovery period of twenty-five years when few of us really plan more than three or four years in advance? So these issues of time and that temporal scale in research and science is really I think is going to be critically important to this whole area of endocrine disruption.

The first point is really how technology changes and by “technology changing” it really changes. How we can think about the problems we're dealing with and so the history of technology and the history of concepts sort of come together back and forth? What I always tell my students is you can only know what you know and so you have to have a context and a framework to look at the problem you're actually dealing with, so let me come to that first point as it relates to what I think endocrine disruption's about and hopefully Scott Frickel who's a real sociologist and historian who's going to be in the later panel will address some of these issues, too. He wrote a really beautiful book about the history of sociology of genetic toxicology—about the whole issue of the society of mutation and how mutation and the formation of DNA adducts with environmental chemicals causing mutation in bacteria was then extrapolated to humans.

Those of us who worked in environmental toxicology back in the seventies only could see the interface between our chemical environment and our own environment as a function of a chemical being bioactivated—it would stick onto DNA covalently—that would cause a mutation and that mutational event was how you got sick—that's how you got cancer—everything else. And so when I first started studying diethylstilbestrol and other synthetic estrogens and ultimately environmental estrogens, we were obsessed with looking for metabolites that would bind DNA and stick to DNA and that's how DES caused

cancer. Diethylstilbestrol as you all know was prescribed to over six—eight million women in the United States for pregnancy problems and then their offspring—primarily female offspring—had a high occurrence of very rare cancers that would show up fourteen years later—in some cases there're still primary cancers showing up in forty-two year old women who were exposed in-utero.

Talk about history . . . we could never really show any of the kinds of things that chemicals did in terms of being bioactivated and sticking on DNA were estrogens, and this then led us to a whole series of studies to come up with the idea that what estrogens really do is perturb the process of cell differentiation through a whole phenomenon called epigenetics that we can talk about later, which is really the cutting edge of thinking about how our environment can interact with our own bodies and the bodies of every other single species that's around. But until we had a context to look at epigenetic change and how that can be passed on through generations. We had a paper where we showed that the granddaughters of DES treated mice would get tumors that the offspring had had, but I didn't know how to even to publish that because I had no technical framework to say, "Oh, that's probably how that happens," and it wasn't until we actually had the molecular studies come out that we could let that paper go out and now there are findings with humans exposed to DES the granddaughters have some of these same kinds of things so—so it's that parallel history of technology and what we know technologically with the concepts that we then use to look at this interaction that I think is a critical historical framework.

The second point, which I had eluded to and I'll just very briefly touch on, is animal studies done with diethylstilbestrol in the seventies and eighties. One of the things in mice we discovered with female offspring is they cease their reproduction at a early age—if we gave their mother DES at higher and higher doses, the female offspring would reproduce for a certain period of time—they would cease ovulating and they would essentially have a mouse menopause and that would be earlier and earlier totally dose dependent. But when we looked at humans as a parallel—the women who were treated with DES in-utero were twenty-four years old—twenty-eight years old—fourteen years old—so you can only say from a biological standpoint this is something that should happen but we really can't see that—we could see the tumors and we had exactly the same cervical vaginal tumors in mice that humans had, so we can make those correlations and just this last year five papers come out within a six-month period showing increased breast cancer—early menopause—uterine fibroids—everything that had been predicted by mice and those experimental studies. It's like a personal history— if you stay alive long enough then you can actually see some of these things play out, but the same sorts of things they're now seeing in humans because women are now in that age group where they'll have more uterine fibroids—breast disease.

And then the third one: Evolution. I wrote a review in 2001 called “Environmental Signaling: What Embryos and Evolution Teaches About Endocrine Disruption,” and I was asked by the journal *Endocrine Reviews* to write some history and philosophy in the first few pages, and one of the issues that I addressed was why so many chemicals in the environment that interact with our endocrine system mimic estrogen—the female sex hormone estrogen. There are no such things as environment androgens—there are no environmental progesterines—there are no environmental glucocorticoids [unclear] adrenal hormones. Maybe thyroid but that’s questionable, and so we started looking and it turns out that plants that make phytoestrogens—all legumes—soybeans—everything else that makes a phytoestrogens—they preferentially make this thing that we interpret as a hormone or a frog interprets as a hormone or a snake interprets or a bird interprets as an estrogen—plants have been doing this for hundreds of millions of years and they squirt these signals out their root tips—that chemical interacts with the rhizobial bacteria and sits on a little molecule called the transcription factor that looks a lot like an estrogen receptor—it causes a whole bunch of genes to be turned on—the bacterium goes up the—into the root—forms a nodule and in its happy metabolism fixes nitrogen so nitrogen fixation in legumes is a result of a chemical coming out of the root tip of that legume with a signal for specifically a bacterium. Then over the years we’ve adopted some of that same biochemistry and we recognize that and instead of be, you know, moving up and going into a root nodule your breast grows or, you know, something happens to a sperm. So, there’s a kind of ancient history or an evolutionary history that also comes into play in how we look at this very complicated issue of how the external environment really interacts with the internal environment. At the molecular level there is no internal and external environment—there’s just an environment.

NL: Thank you.

SARA VOGEL: Okay, so I have to [laughs] follow John McLachlan, but I would recommend everybody reading that paper—it really transformed the way I thought about things and I think it was Russell actually who wrote a paper many years ago—I don’t know how long ago actually it was—that put a call out to environmental historians to try to grapple with this issue of evolutionary biology. Endocrine disruption is really at the heart of this and I’m just going to—hopefully not selfishly—tell a little story about the dissertation work that I’m doing as a way of to kind of giving us maybe a case study to think about some of this stuff.

So, a couple years ago the CDC published a paper that showed really persistent ubiquitous exposure to a chemical called “bisphenol A,” and they’d been doing a lot of work on other chemical agents. They just found this one and they found it in 95% of the samples of adult humans around the country, and so it raises this obvious question when you find out that you have low levels of this chemical in your body and most everyone has it, where is it coming from and what are the risks at this really low level? And my argument is that this isn’t just

a scientific question with scientific answer but if this chemical really has a history and so how did it become ubiquitous in the environment?

That's really what my dissertation's trying to do—it's trying to say what's the history of this chemical and I've actually been pushing it further back [laughs] in time and now it's about the—really the late fifties for regulatory reasons and so let me just explain why this one chemical and bisphenol A is synthetically produced and it's used for two plastics. It was originally sort of synthesized at the turn of the century but it doesn't come into commercial production really until they're basically discovering the first plastic use in the thirties, when it's also identified as an estrogen, but it gets used for epoxy resin, which are these really powerful adhesives—combine metal to metal and holds airplane wings together basically—but it also is used in the lining of food cans. It's used in dental sealant for cavities in children and then the other plastic sort of end products are these polycarbonate plastics—hard—translucent—heat resistant plastic—it's plastic that's so strong that it can actually replace metals, but at the consumer end the concern of late is that it's used to make baby bottles, in part due to heat resistance. They're also used as flame-retardants in blends, which are really popular plastics and the production has reached like basically over six billion pounds globally. About 2.3 or five billion pounds are produced in the United States for the huge Asian market obviously. So, my dissertation asked why is—it's a tautological question—how—why is there now this really contentious debate over the kind of science and the politics of this chemical. . . .

Now let me skip to the nineteen nineties and give a little context for sort of what we're all talking about here. Beginning in the early nineteen nineties BPA's estrogenic properties had kind of been in a way forgotten for a really long time and then it sort of emerged from the literature but it kind of becomes visible again in an experiment—it leeches out into some— from plastic flaps in a experiment of some endocrinologists from Stanford University and they were lucky enough that a similar incident happened a couple years ago in Anna Soto's lab. She discovered that it was a different estrogenic chemical leeching out of her petri dishes and interacting with the breast cancer cells which she was looking at. So, all of a sudden the BPA comes back onto the scene and it had been assumed to have really weak estrogenic properties, so thus it was held as having sort of insignificant health effects and thus we can be exposed to it at low levels through our food—through the products that we use and some of the most alarming studies of late have linked low dose, very low dose, BPA exposure to reproductive abnormalities.

These are in animal model studies so using rats and mice—most recently increased risk of breast cancer—prostate cancer—potentially increased risk of metabolic disorders like diabetes and obesity and most recently have some chromosomal abnormalities. So, I think that this is a useful example to think about and I'm not making the assumption that it's somehow is unique among a whole host of chemicals that are concerned but I wanted to use it as a way of

cutting through some of this stuff. Well, let me explain a little bit of why some of these studies are so controversial and part of it is that—just of broadly speaking—a lot of the work is in part being done by people that aren't traditionally toxicologists and they're working with incredibly low levels of exposure.

These are amounts of chemicals that are understood to be pharmacologically relevant so that they're known to turn on a system or change the development pattern but a level that a toxicologist wouldn't consider having a toxic effect seems to have a lot to do with the history of the development of ideas about what are the toxicological properties. They're also exposing these animals during early development, which wasn't a framework that was used prior and then most recently based on some funding that was made available from the NIEHS they're following these animals all the way to the end of their life and so they're able to look at really different disease outcomes. They're not just sacrificing the animals right after they're born and saying, "This is the reproductive [unclear]," were there—these animals get born, you know, with a—if the mother fertile so basically what's happened I think and—and I can be corrected—that over really the past fifteen maybe twenty years that there's been emerging these kind of series of criticisms of toxicology and I'll just kind of briefly go through them so that we're—maybe it can be added to but so we're all sort of on the same page and one of the main criticisms is that toxicology has dealt with chemicals individually—sort of a chemical by chemical route which is my [laughs] [unclear]. So the criticism here is that this really falls short of real world exposures which come in—and the term specifically used complex mixture, which would result in really different effects.

The second main criticism is that toxicology traditionally have looked at very high doses of exposure and then had made different kind of extrapolations down to what might be happening at these very low doses. What happens then of course is that area is a bit of a black box and so that's where all your different theoretical understandings about toxicological [unclear] on metabolism and what's going on once the chemical enters the body and how is it being excreted. So, the criticism there is that in that black box you have all this space for political negotiation and so that's where all the tolerance limits get set—that's where risk benefit analysis gets done and it becomes very much who's sitting at the table—who's saying which studies are sound science and which are weak or not rigorous.

So then the call has been that toxicology really should look at very low levels such as the work that's being done now—what's being called environmentally relevant doses—doses that would reflect some of the biomonitoring that's going on in humans. And then a third criticism is that toxicology has traditionally used adult models meaning they expose adult animals and look at the effects even if they're looking [unclear] effects as to offspring. The criticism here is that you would miss what has been called the kind of critical windows of development where a really low dose when you're giving it to the developing fetus that might have the same level of your chemical as it has

circulating estrogen in its own body would have a much more profound irreversible effect than if you're giving it at a later stage.

So, this is also led into criticisms that the toxicological model has looked at endpoints that aren't relevant—as relevant anymore—that it's just thought about death and gross malformation—weight loss—maybe even organ weight—gross organ weight changes and not more maybe [unclear] effects where you're looking at developmental change. You're looking at how the development of the organ has changed—is that leading to higher risk of development of cancer later in life—is it affecting the way the body metabolizes glucose or something like that? So, I think these are all really important factors that have come out of this field, but I think there's also interesting historical reasons for why toxicology has failed and I think a lot of them have to do with the technical issues and the changing ideas about what we know and how we can know it and how we can conceptualize it.

I want to close with two important features about toxicology that have been well documented by historians Gerald Markowitz and David Rosner, my advisor, and what's coming out in my work is that the disciplines always really been heavily influenced and shaped by the chemical industry. And second and related is that toxicology is a discipline that hasn't been defined by really fundamental biological questions, but really is interested in meeting and shaping regulatory demand to limit liability and to secure assumptions about chemical safety, thus industry's interest in controlling regs.

I think that raises a question — and I just put this out there — may be totally off base but it's a question of whether is toxicology the appropriate discipline to even be asking these questions? I mean so much of the interesting work has come from people in really different disciplines— there's a reason you were trained as a pharmacologist [laughs] and work on molecular biology and so my ending question is that what would an alternative discipline look like and I think that's the part that endocrine disruption world is trying to develop a discipline so what would those boundaries look like? And if toxicology and regulatory policy have been wedded together what then does that policy begin to look like when we change the base of knowledge that supporting it?

NL: Thank you Sarah. While Tyrone's coming up I want to give a quick advertisement—tomorrow morning at 10:30 Sarah's giving a much fuller version of her paper and I'm also talking about DES and epigenetics and regulatory arguments, so if you want to hear more—come tomorrow morning.

TYRONE HAYES: What I want to do is just briefly introduce you to atrazine and then put that into a historical perspective. So, just to let you know what atrazine is — it's an herbicide or weed killer that's primarily used on corn in the US; it's been used for forty-eight years. I guess that's our first bit of history—and we use eighty million pounds annually in the United States making it now the second

biggest pesticide in the world, second only to glyphosate (because of the GMO use of glyphosate now) and it's used in more than eighty countries. It's now outlawed in all of Europe so—again—just to give you a historical perspective in the same year—2001—that the Environmental Protection Agency re-registered atrazine, the entire European Union banned it and that's significant of course because the manufacturer—Novartis Syngenta—is a Swiss-based company.

So, here's a compound that's illegal in the home country, but we use eighty million pounds a year, and I usually like to point out at this part of the talk that one of the other countries that's banned atrazine is Angola. I don't know if you guys know, but again from a historical perspective Angola has the longest standing civil war in all of Africa and if they could get together with their gold, oil and diamonds, they'd be the richest country in the world. They can't do that but they know they don't want atrazine. Again, we're using eighty million pounds a year. So, just to cut to the chase here this is about half a dozen papers and about five years—ten years worth of work summarized in one slide—here's what atrazine does to frogs at .1 parts per billion. This is a male leopard frog—those are the testes—and all of this junk in the trunk as I usually refer to it are eggs that have developed in this males testes that have yolked up and that are bursting through the surface of this males testes at .1 parts per billion -- an effect that the EPA now recognizes as a real effect but has not yet ruled on whether or not it's an adverse effect that would lead to the regulation of the product. The women are laughing but all the guys are thinking about a dozen chicken eggs in their testicles. The EPA says it's not an adverse effect . . .

What I want to show you is that atrazine works a little bit differently compared to the environmental estrogens that we've heard about already and atrazine doesn't follow—it doesn't in itself act like an estrogen, but when atrazine is present it reduces testosterone or chemically castrates animals and it does that because it induces the enzyme—or the machinery if you will—that's required to convert testosterone into estrogen. As a result in frogs you see a reduction of androgen dependent characters. You see a reduction of sperm and a reduction in the growth of the larynx or voice box and you see an induction of estrogen dependent responses so the growth of eggs or ovogenesis as well as the yolking up of those eggs or vitellogenesis in males. Now, this slide should give renewed meaning to the term “red states.” What this is showing in red the states that use most of the atrazine—where most of it's used. I guess what's relevant is—of course it's used on corn but if you also follow—this is actually the Mississippi River here—so not only is all of the atrazine being dumped into the Gulf of Mexico primarily because it's flowing down the rivers here, but if you also look straight down there's a lot used along the river and I understand there's a field trip here where we can go visit the Syngenta factory and I saw it—oh, a few months ago and there's a big pipe that runs right out and right into the Mississippi River. I'm not sure what it's dumping but it's there. Also relevant to this group I want to point out that this is a statement by Glen Fox, “In ecoepidemiology,” he says, “the occurrence of association in more than one

species and species population is very strong evidence for causation,” and I won’t have time to talk about it but the induction of aromatase and production of estrogen has been shown not just in multiple species but in every vertebrate class that’s been examined including mammals.

I want to show you a little bit of the evidence of what happens in laboratory rodents and some of the associated problems that you see in humans — and I’d like to always put up a statement here—when I presented this to the Minnesota State Legislature, EPA commented that there is no direct information to assess this hypothesis that these effects occur across vertebrates—somehow they’ve missed about forty publications. So, the evidence that atrazine reduces testosterone and testosterone dependent features in rodents is that in rats there’s a decrease in testosterone and there’s a subsequent decrease in sperm number—this is from one of many papers that was published in Europe and [unclear] Shauna Swan has shown—these are atrazine levels in the urine of what she calls control males in Missouri and the level of atrazine in sub-fertile men—men who have trouble getting their wives pregnant—men who have low sperm count.

Again, I don’t know how to translate the doses—but these men have atrazine levels in their urine that are equivalent to what it takes to chemically castrate a frog. And now watch the Y axis because I’ve reduced the data because I’ve increased the numbers on the Y axis because another study showed that these are atrazine levels in the urine of field workers and now watch the axis again—these atrazine levels in men who apply atrazine, okay? These men who work in the corn fields applying atrazine have twenty-four thousand times the atrazine in their urine that we know is associated with sub-fertility in men in Missouri who just live in this agricultural area. Or as I usually tell my general audiences—these men who apply atrazine have twenty-four thousand times the atrazine in their urine that it takes to chemically castrate and make a male frog make eggs. One of these men could pee in a bucket—I can dilute it twenty-four thousand times and use that to chemically castrate and produce eggs in twenty-four thousand tanks of thirty tadpoles each.

That’s the level, but nobody knows anything about the health care of the men shown here because they’re primarily African-American and Mexican-American farm workers and nobody follows their health, but we know that twenty-four thousand times less is associated with low fertility. The other half of the equation that atrazine induces aromatase and causes estrogen like effects including mammary tumors in rodents has also been published. Here [unclear] levels of testosterone—this was actually done in an EPA laboratory showing that atrazine again is reducing testosterone in male lab rats and that there’s an associated increase in estrogen—not a coincidence—same thing we’ve seen in frogs—in fish—in reptiles—now also in rodents.

Also, in rodents there’s an increase in estrogen dependent mammary tumors associated with that increase in estrogen in rats that are exposed to

atrazine. Now, the company argues that this is not an increase in the incidence of breast cancer because they argue the controls would eventually get the same incidents of cancer if they lived long enough—but again that’s the equivalent of saying “Ah, you’re going to die anyway—we’re just making it happen a little bit earlier.” In *Human Cell Lives* published in 2001—we’ve now published a paper showing the same thing—in human cancer cell lines atrazine induces aromatase and a study of two in 1997 showed that breast cancers were associated with atrazine contamination of well water with the p value of .0001—and this is when women were compared to women in the same community but who didn’t drink that well water. EPA says there’s no direct scientific information to test this hypothesis but here I think is the most important information—cancer is not caused by estrogen or—or atrazine—cancer happens when you get enough damage to the cell that it simply doesn’t grow properly; it grows out of control. But we now know that aromatase locally produced in breast cancers is very important in causing those tumor cells to grow out of control once they’re damaged.

Here’s the evidence—even if I didn’t tell you about the forty papers that I just put up, here’s how much we know about the importance of aromatase in breast cancer. Right now there are companies developing chemicals like letrozole, for example, and they claim that this chemical works a thousand times better in blocking the estrogen receptor because this chemical knocks out aromatase, which reduces estrogen, which prevents the cancer from growing in the first place. At the same time that these companies are now selling you—or want to sell you—letrozole to block your aromatase when you get the number-one cancer in women another company is producing a compound that a million people a day—60% of all Americans—is exposed to. Now, I just told you a lie—it’s not another company—it turns out that Novartis oncology offers treatments for cancer [unclear] to breast cancer. The same company that has given us fifty years of an aromatase inducer is now trying to turn around and sell you an aromatase blocker and the best part of it is if you go on their website and read about my work they’ll say “Oh, atrazine and aromatase isn’t important for breast cancer.” You go on their website—their pharmaceutical branch is telling you, “This is a thousand times better at treating breast cancer blocking aromatase.” Now, putting it in historical perspective here’s the problem—a law was passed in 2001 and my data were the first actually to use what’s called a data quality map—the Center for Regulatory Effectiveness basically says no primary literature—no primary studies can be used for regulation of a chemical—that the EPA has to develop a standard protocol first.

Here’s what the EPA’s done in response: instead of looking at the primary literature, they have what they call a tiered model whereby the company now is required to repeat for example my laboratory data before they’ll consider regulation of atrazine. If the company can’t repeat the laboratory data—which believe it or not I bet they won’t—then you stop. If the company does, then they have to now go repeat our field data and by the way it took the company six years

and they still haven't produced the data to do what it took my undergraduates three months to do so how long is it going to take them to do a field study? But even if they get a 'yes' answer then the EPA requires that they do a series of studies that will explain the mechanism of how atrazine works before they'll even consider regulation.

Now people say, I don't like atrazine so I want to—put this in historical perspective—let's say that the EPA had existed in 1973 when DDT was banned. If the EPA had existed in 1973 with their tiered model we would have had seven more years of DDT because it wasn't until 1980 that the laboratory data were produced that showed that atrazine caused eggshell thinning in birds. We would have had twenty-five more years of DDT because it wasn't until 1998 that the field studies were done that showed that when you removed DDT, eggshells returned to normal because ironically you had to get rid of the DDT before you could do the study. And then finally in 2006 a paper was published that showed that the mechanism for contaminate induced eggshell thinning in wild birds remains to be clarified. So, if the EPA had existed in 1973 DDT would still be on the market as would Tributyl-10—as would DES—we still don't know how it works. And somebody mentioned mixtures—imagine if we had to go through this model for mixtures of compounds—imagine if we had to go through this model to not only address how the compounds work but go through three generations to get to your DES grant dollars.

So, speaking of history we've moved backwards, I would argue, and not forwards in how we regulate chemicals and not to make light of it—imagine if we used a model like this for other things that we already know—for example, that speeding cars kill children. We'd first have to put some kids in a parking lot to show that in fact speeding cars do kill children; then we'd have to do the field study—we'd have to put a few out on the highway. . . But even if we showed that we'd have to go through the mechanisms—were they crushed by the front tires—no—crushed by the back tires—no—hit by the bumper and dragged—it is not enough; when you know there's a problem you simply put a stop to it—you don't keep testing it. And finally, I just ought to point out recently after this pretense that all of this science is going into how the EPA's going to regulate chemicals they said the following in an article—a newspaper article December 3, 2006. “The ultimate decision,” the EPA says “is much bigger than science and waves of public opinion,”—so they're counting on you to decide what should be in environment and what shouldn't be. Thanks.

NL: Atrazinelovers.com [laughs]?

TH: Oh, and if you do want your opinion, there is a website atrazinelovers.com has legislation coming out in several states and you can go out and endorse that legislation. All the literature on atrazine is available for you to read on your own, in case you think I made it up—atrizinelovers.com.

ARTHUR DAEMMRICH: All right, so this is a somewhat different take across the same territory. My background's mostly in science studies with a focus on political science and regulation and I created a huge title for myself just in case we completely run out of time . . . but I think we have few minutes so I'll try to get through my talk very quickly. So this is ethic—biomonitoring at the crossroads of advances in analytical instrumentation market success of second generation chemical industry synthetics and shifts in social and regulatory rules occupied by engios, the government and industries. I'm going to now take that [unclear] and turn it into my talk—within the whole talk, right? So, let me go through a little bit of the longer history of what has become biomonitoring today and we can track its origins back to the nineteen twenties going up to the seventies which had to do with lead studies and those were prompted by health concerns in Baltimore—Boston and other northeast cities and at the time.

If you go back to the twenties—thirties—forties—you were talking two plus days per sample and tens of thousands of dollars to run the test. Since Mike Egan's here I'm going to take as my second major turning point in the longer history—the study of nuclear radiation and it was of course—say the name—Barry Commoner—whose different studies of children's teeth—baby teeth in the nineteen fifties and sixties finding background radiation and the impacts of nuclear testing and that set of studies actually contributed to the atmospheric nuclear test ban. From the nineteen sixties on we start seeing increasing numbers of government surveys, including my state fish and wildlife agencies who are now beginning to track not just animal populations but also the presence of chemicals in the environment over time.

Coming forward to the nineteen seventies, there's a vinylchloride debate and concern and the workers safety issue that now for the first time really involves the chemical industry and here we see the period of expansion in the field of toxicology as an academic discipline. So, those are sort of background larger scale who's doing what population level surveys focusing in on what would become biomonitoring—in '67 you have the launch of the National Human Adipose Tissue Survey, which collected fat from cadavers eventually built up a set of twelve thousand samples and by the late nineteen eighties they could identify seven hundred substances. But the study was not structured in a way to really match those two known chemicals and the entire program was discontinued in 1987.

In the meantime, CDC had the gun, including environmental chemicals in its long running National Health and Nutrition Examination Survey. A couple other quick historical points: 1980 the case of animal feed in Michigan contaminated with PCB's and CDC at that point then began expanding and including new compounds in the NH study. In 1992 the National Human Exposure Assessment Survey was initially launched—in '99 CDC [tape off/tape on] [End of Side A, Beginning of Side B]—that suggests particular compounds to CDC to include in its report. In 2001, the first CDC [unclear] report was released

which covered twenty-seven chemicals. By 2003, the second report expanded that to a hundred and sixteen and the most recent one covered a hundred and forty-eight. What that historical sequence suggests first is a growing realization that humans absorb chemicals from consumer products—from consumer goods and second a fairly broad kind of cultural focus on the body even within the environmental movement and that human health can be kind of a surrogate for measuring environmental health.

Where is biomonitoring today? What we're seeing are a number of different national level surveys—population based surveys especially done by CDC. We're seeing activist type surveys, which [involves] various NGO's conducting very specific studies not so much of populations but of specific individuals, and we're seeing a number of interactive websites around the issue. I'm going to show you a couple examples of those to give you a sense of where the politics of this dispute are today. I think in terms of broader questions there's a real big question here on peer review of the studies—studies are being done within industry—within government—within NGO's—within academia, and there's very different kind of assessment of what counts as a good study.

Linked to that there's a big question of looking forward of standardizing and making different specific studies comparable so you can study any one of several thousand chemicals . . . And there's a huge question around which compounds you're selecting—EPA has eighty thousand chemicals licensed for manufacture or sale in the United States—nobody's studying eighty thousand compounds, so which ones are you going to do and what is the threshold for regulatory action? We saw that with the two examples given, at what point are they crossing that ground? And we come to that last point last—here's a first example of an environmental NGO addressing [unclear]—the Coming Clean Network and what you see here—this is a visual pulled off their website—the toxins are located all over the body—they're not just located in the woman's breast—they're sort of everywhere in your body now, and the focus is of course on coming clean—on removing the toxins from your body.

A major study put out by the Pesticide Action Network raised the question of chemical trespass and the people I've spoken to within the chemical industry—this is of course a sort of frightening notion to them because it's now going from a scientific to almost a legal framework. Taken quite literally, this would open companies to potential litigation for compounds identified in your body that you didn't allow them to put into your body. I mentioned that there are interactive games on the web built around this notion so here's—by the WWF—the World Wildlife Fund—they've created a playful sort of asteroids like game called Toxic Blaster in which you pilot a spaceship and you go into first a polar bear and then a whale and then ultimately a human, and you destroy different toxins. In between there are moments where you can take action now so that there are different windows that pop up as you play inducing you to learn more about the topic and also e-mail or write your congressman or congress person. Just to show you a

second slide on this—this is what happens if you play the game [unclear] iceberg—polar bear is full of flame retardants—PCB's and PFC—his immune system is shot—you actually then sort of enter the polar bear—I won't tell you exactly how—you have to go to the website [laughs]—and then you're inside and you get to blast these . . .

The final one in terms of activist NGO's is the famous Body Burden site from Environmental Working Group, and this is around the point I made about them focusing on specific individuals. Here they found—they took a set of twelve people—I've clicked on Bill Moyers—you see him highlighted—he's of course a perfect one to show people within the chemical industry because he did this PBS special a few years back on toxins in the environment. This highlights then—it's set up visually to look like a periodic table—of course it's not because it's all variants of PCB's—purans and other organic compounds—PCB's—and if you click on any one of them a window will pop up showing you the specific compound and also what chemical companies have made it. So, this is a very powerful interactive kind of visual data tool. Ironically of course what this now means for the chemical industry is the longer Bill Moyers lives the better for the chemical industry because it makes these compounds look less toxic [laughs].

All right, shifting gears completely, here's how the chemical industry is beginning to represent itself in relation to these sets of issues. This is not technically a [website?] put up by industry but it is heavily industry funded. This is a group that's run out of a number of government labs and what this is showing is sort of a perfectly pristine clean chemical plant—sort of isolated from its environment, and yet part of it. If you click on any of the red dots that show these, you know, cooling towers etcetera—etcetera a window pops up showing you how that element is regulated so the point this conveys is this is a highly regulated, highly controlled industry. Dow Chemical has recently launched a set of ads many of you have probably seen—the human element—really trying to give a kind of human story and human touch so that's not focused to the content or controversy, but rather saying there is a human element binding us all together with common hopes and aspirations so it's a kind of feel good campaign but its focus is on cleanliness, safety and the future and offers a kind of conservation vision about people not about external nature. And third, the chemical industry has launched a big advertising campaign called the Central Two—you may have seen some of the ads—some show sort of an alarm clock melting away because the chemicals that are needed to make the plastics in the alarm clock disappear so then of course you can't go to work so it's disrupted your life. This is the piece of it that's very sort of external environment focus so I've been saying that a lot of this is about the human body here's an example of sort of external environment—but here again it's sort of our air, land and water are cleaner now than they were thirty years ago.

So, let me close with some analytical perspectives as a kind of policy analyst. Number one around the issue of visibility—so every consumer product

you encounter and increasingly building materials and other really sort of the material world in which we live is packaged in or manufactured by the chemical industry. Only rarely are most of these materials labeled Dow—DuPont or BASF, so unlike in other sectors by the time the product gets to the Walmart or other store shelves these compounds have been traded among three or four companies. NGO's have worked very hard in recent years to make those supply chains—to make the products visible in their environmental and human health impacts more visible to the public.

An important turnabout since the nineteen eighties, the chemical industry itself has come to the conclusion that it is misunderstood because its innovations are not sold directly to the consumers so the industry too is interested in increasing its visibility to the public. You've probably all heard the BASF advertising "We don't make the products you buy—we make them better," so that kind of raised the visibility of who we are as a sector. Number two as an analytical perspective—changing concepts of risk so historically risk in how we regulate was this classic hazard times exposure equation. If you're thinking about biomonitoring exposure tervasine, which means risk now becomes some form of equivalent to hazard. Likewise how we regulate risk has been around materials and chemicals physically in the wrong place.

If you think of the entire legislative history in the nineteen seventies around Clean Air Act, Clean Water Act, Toxic Substance Control Act—it's get these things into the right physical place and it will be under control. The biomonitoring area really challenges that on a very fundamental level, and risk is also of course increasingly understood as linked to values, so the very way in which you're going to do the tests in biomonitoring is challenging because animal rights have come to the floor—the way in which we do laboratory testing has become contentious itself. So the knowledge production of biomonitoring is intertwined with how we understand risk related to values. And my last point on the risk one is biomonitoring poses a huge challenge to how we understand risk in that greater wealth now means greater exposure so for the last several hundred years of human history wealthier people were able to pull themselves out of most of the risk zones ,so you didn't live adjacent to a chemical plant—you didn't breathe the fumes and fogs but now of course it's the wealthier you are the more you consume—the more exposure you have—it's in the plastics—the more you drink bottled water as opposed to tap water, the more exposed you are. That's a real challenge to how we think about it.

My final point and then I'll sit down and we'll open a discussion, is that in the last couple of decades we've really shifted how we think about regulation in the US and I think this might be a contentious point to this audience but EPA is no longer the center sight—EPA is a kind of data monitor and neutral meeting ground—a place in which results get debated, discussed and contested but effective regulation going forward—I would argue—really needs industry; NGO's and EPA to sit down and hammer out together what will count as an

appropriate test for this area—run the test and then make the set of decisions. We're increasingly relying—for better or worse—on industry to self-regulate, self-monitor and do so under NGO eye so that there is an observation mechanism—a form of control going on and yet it's not the traditional command and control regulatory. Where that leads is f open to discussion I would say but my sense is that's where we're headed as a society for better or worse and we need to come to grips with that—understand those different roles occupied by those different major groups and help support and analyze that. So, let me end there. Thank you very much.

NL: Thanks everyone for lots and lots of really provocative comments. I want to make a couple quick comments in response and then ask for feedback from each other and then we'll have some time for response. Then we'll have, I think, a whole hour later after the second roundtable.

I was struck by so much of what you all said, especially the idea of this new model coming forth in regulation. One thing we didn't talk about a lot was politics—Tyrone mentioned a little bit—well pointed obliquely. I think that the question of political influence is important: —how politics in these conceptual models are interrelating, how different groups with different amount of money, different amounts of power, and different amount of pressure—are able to manipulate the uncertainty. That's a lot of the focus of my research, and trying to figure out these interrelations between emerging conceptual models, and politics, and money is a huge challenge.

One question I have for all of us is how much are politics and money manipulating this radical uncertainty. The other thing I think is really intriguing that came up in all these is the question of visibility—what is visible, what is known, and what is invisible. It was so intriguing to me, Arthur, that you mentioned that the chemical [industry] is beginning to think they're not visible enough, because in some ways I would think visibility is not in their favor, really. One of the ways I got interested in this was when I saw the Bill Moyer's special, "Trade Secrets." I was shocked, and I started looking around, and I looked at my students who were working on a project on the Fox River and the PCBs Superfund site, and I was thinking "Well, that's fifty miles away." And then I looked at the Superfund registry and I realized "My realtor forgot to tell me that, literally, a thousand meters behind my house there's a Superfund site." And I called up EPA to see what was known, and they were saying, "Well, we haven't shown any effects in the well water." And I asked, "Well, have you tested our well water?" "No, but we haven't shown any effects. . . so it must okay." And I said, "Okay guys." And I popped up the atrazine map thinking "Oh, those poor people exposed to atrazine." And I looked and I thought oh, that's the field back there—oh, wait—I'm in Wisconsin—that's the reddest of it all.

And then, I started working on endocrine disruptors as a response to this, and this project has taken long enough that I developed into the woman in her

forties and had a lot of cancer issues and, you know, learned I was likely DES-exposed and went through a whole bunch of stuff with dealing with that. All of a sudden realizing that these things that you think you're studying out there are literally popping up inside you. . . . But the uncertainty is there; my mother doesn't exactly remember what was prescribed—the doctors can't exactly say—everything becomes very uncertain, and very invisible, and so there's no real responsibility in some ways. . . . I think this is happening to so many people; their responses to toxins begins as a kind of intellectual issue, and then we start realizing we're enveloped in it, . . . realizing that we're all in this together is, I think, one of the most profound things coming out of this research.

Do people on the panel want to respond to each other, or just open it up for comments and questions right now? Open it up. I'm going to ask you to please speak as loudly as you can, because we are tape-recording this, and please give your name.

FAUDIENICE: My name is Mathews—and I have a question about the exact flip side of what you just mentioned and that's the idea of complacency and the role that people think technology can play, because I think that there are people that live in the red states or, you know, they have the dumps in their backyard or, for whatever compelling reason they get involved and they get interested but then I think there's another segment of the population that doesn't live in a red state and they develop a certain complacency about it, because it's not in their backyard or because they have this misguided notion that some pharmaceutical will come along—some technology will come along, and they will make it all better. So I was wondering if you can comment on some of those issues. . . .

NL: Anyone want to jump in? John.

JM: Sort of coming to what you had said about politics and money and coming back to your comment. I mean obviously politics and money drives everything and to think differently would not be sensible, and every industry is out to make as much money as they can for as little cost and going back even to diethylstilbestrol—Eli Lilly made DES—they were sued and there were all the same sorts of things that are happening with atrazine or anything else where different people's results couldn't be replicated or this gynecologist in Stamford would say, "Oh, that doesn't happen this way," and then you find that he's been paid by Eli—Eli Lilly. This is just sort of a recurring kind of theme that's there.

One of the things that's happened with—I don't like the term endocrine disruption, but—if you take this idea of environmental signaling or hormonal activity or biological activity of environmental chemicals—however you want to call that—what that's done is break through the idea that there is a safe level—that up until then I think part of the propaganda that was supported by science really is that you need a certain level of chemicals to do certain things that are pretty easy to monitor to say there's going to be a risk. What things that effect the

endocrine system have shown us is that very small amounts can have lasting effects and that the developing organism—whether it's a frog or a human—might be greater risk—what hasn't really been done much, which I think is going to be the new generation of this kind of work is looking at the immune system—the nervous systems—it would be incredible to me if there weren't synthetic environmental chemicals that didn't affect the nervous system—the immune system—some hints of that have started showing, so endocrine disruption I think is just going to be one of those areas that because we discovered environmental estrogens a while ago that pushed that.

But I'm sure that we're going to see these misprogramming of a variety of other things so the difference between red states and not red states won't be so great but the whole issue of complacency is the fault of the scientists and the industry, and everybody else, because the industry obviously doesn't want you to worry about these things and there are all these reasons why oh, that data can't really apply to you but from the standpoint of scientists I know—this happens to me all the time—someone says, “Well, what should I do? Should I not use plastic bottles?” You know, and so I'm sitting there guzzling a plastic bottle and, you know, I'm doing this—doing that and somebody says, “Well do you ever do the following sorts of precautionary things?” And I don't, you know, so that in some ways the place historically we are not is what to do so I can tell you how these chemicals work—I can show you the genes—I can show you the rapid estrogen signaling route that I think is the new key to things, but I can't tell you what your behavioral changes should be and if I can't do that then why should anybody care. I think that's a big, big problem in this whole area generally of environmental health, but I think especially in this particular area.

FA: Well there's a whole lot of research now in the time how humans perceive risk to our evolutionary past. I don't know if you've read any of that—

JM: I haven't, no—no that's very interesting.

FA: Yeah, and the idea is that in our evolutionary past that the things that we were [unclear] into thee so called, you know, tiger [unclear] wood—and so the human stress reaction and stuff that we're worried about in terms of risk tends to be [unclear], and there's less of an issue of worrying about concerns not [unclear] in the future—

JM: Right.

FA:—like what you're [unclear].

JM: Well, it's like smoking, you know. . . If you're going to get cancer twenty years from now you can not worry about that, but if you're going to get hit by a car, then you're going to worry about that. I've also noticed that women worry about

kids and if you say, “This’ll affect your children,” there’s more concern in that respect—and again that’s another way to look at it.

NL: Okay. We have a bunch of people—Jody wants to make a quick comment—Arthur—Willy had his hand up and then Sarah.

JR: I think, just real quickly, I think you hit on a really key point. I think that part of bringing together these different perspectives is one this is a faith in technology issue, both by sort of communities but also by scientists, who assume that the stuff they’re doing they have faith in and that it won’t do harm and that if it does do harm they’ll find a way to fix it with more technology. But this is also an issue of communication, and I think one of the big issues is making this stuff visible. The way in which we make it visible—if you run the risk of democratizing too much and you say, “It’s everywhere—it’s in everything—we’re all, you know, we’re all kind of finished,” then there’s the complacency of—

NL: Yeah.

JR: —oh, well but it’s drawing up these sorts of connections that I think Tyrone did of, you know, the company that is producing the compound that is potentially producing the cancer is also producing the compound that is working on the same mechanism to treat your cancer and it’s making those sorts of connections visible, which is much more difficult. But is what’s actually necessary to start changing attitude and not simply doing sort of regulatory precaution.

TH: No and—sorry to interrupt but what you said was is just important—I’ve been working on this for nine years—I only recently discovered that a—

JR: Yeah.

TH: —few weeks ago, I was looking at a website and I said, “Hey, wait a second.”

AD: On the complacency point, which I think is a really good provocative question, so let me throw out a hypothesis for the future, which says within a decade you’ll be able to walk into some little storefront in one of these strip malls and get a set of tests done for under a hundred bucks that tells you every compound in your body. Or there’ll be a checklist out of a thousand compounds that they can run an assay on for under a hundred bucks—here’s what you’ve got and you do that every couple of years and you’re going to get pretty non complacent about it and the kind of people who go in and get those tests done are going to be middle class and above and those are the ones—this is a total cynical statement—they’re the ones who tend to have a pretty strong influence on what gets regulated, and so you may see a shift in that in a way that we did with smoking. For a long time smoking was an individualized risk choice—it was known to cause cancer and yet there wasn’t a broad societal concern and this may shift from that individualized

choice to a societal level of health care funders and the like may start saying, “Hey, we need to regulate this,” and that may shift the train quite considerably.

SV: Oh, can I add something?

NL: Yeah.

SV: Just on the uncertainty and complacency—I think it’s important because a lot of people brought this up and Tyrone’s if we had waited this long for DDT this is when we knew—is that we know a hell of a lot more about a lot of these compounds now than we did when we set up some of these regulations and I think that’s really important and so uncertainty gets produced and so—for example the—the kind of brain work for NIEHS right now is, you know, genes and the environment and there’s been this push really since it’s kind of a Clinton administration thing where it was we need to look at mechanisms of action so all of this research fell to answer these questions and then at the same. The chemical industry’s making memorandum of understandings with NIEHS and they’re supporting this work and then at the same time on the other hand they’re saying, you know, all this research is coming out and they’re saying “Well, you know there’s still so much uncertainty because we don’t know the mechanisms of action or we don’t really know what—we can’t really go from animal model to human model,” even though this has been the role [unclear] so suddenly that’s driving complacency, right? So, it’s saying if everyone had these, Bill Moyers seems to be fine, so for the guy who wrote article in *The Smithsonian* where he had a [unclear] really healthy, strong guy, start to think okay, well, does it really matter?

NL: We have a couple minutes more before the break and then we can continue the conversation informally during the break. . . I know a lot of hands are up—does anyone want to comment specifically on the uncertainty/complacency? Sara and then Chris. Yeah—and say your name loudly please—okay—thanks.

FAUDIENCE: I’m Sara [unclear]. I’m an anthropologist—I live in a small city that is—was [unclear] by a pharmaceutical company, which pulled up stakes several times and just did so again recently and a lot of people that I socialize with are chemists and they work for this pharmaceutical company, where I work with [unclear] at my university. I’m struck when I listen to this by thinking “How can they do this—how can they be [unclear] scientists and ignore this?” I think [unclear] the complacency issue really centers on how we train chemists in university and where they’re going to go to work after, [unclear] and the chemistry department is specifically small but most people that graduate with degrees in chemistry are going to work in the industry, so now this is my initial response and then I heard Arthur’s discussion. There’s going to be some sort of consumer demand that’s going to push the gate, but in the mean time I do wonder, what are the possibilities for reaching across the [unclear] university to try and find a chemistry department’s about how you educate your students so they make notes for the [unclear] the job that they’re going to get, you know, be able to make good

middle class livings on and reproduce, you know, internally what [unclear]. They give up their jobs as educators just to work within our own sort of industry but, bring ethics to some area . . .

NL: Rather than answer that right now on the panel I think we can talk about that on the field trip—I want to hear Chris—

FAUDIENGE: I actually have a different kind of comment that relates to the issue of uncertainty in knowledge and I want to say and I don't know whether you guys are aware of it, but there is a huge initiative in the EU called the Breach Initiative that's going to—assuming it progresses as planned, which is going to require all the chemical companies and the chemical users in the EU to study their eighty thousand chemicals. And this is going to result in a huge increase in [unclear] I'm assuming—in knowledge about the health impact of all these chemicals. Out in California where there's less [unclear] some interesting green chemistry where [unclear] but that's essentially changed the framework. The problems that we have with the EPA are really serious problems—the EU is stepping up and—and providing an alternative, and I'm curious to know whether you guys are thinking about that and [unclear]—the way things are here and the Europeans are trying to fix it and—and then you have this huge increase in knowledge and what do you do with it?

JR: I'm a bit cynical about the EPA but in the case of atrazine, for example, the data's there—what happens on these EPA panels on atrazine is they'll say, “Does atrazine cause [unclear] in frogs? And then of course I'll show up and there's a few other studies other than mine but then they end up bringing up nine non chemists and nine biologists won't say anything and so then what happens is the EPA hasn't even reviewed the [unclear] literature and all those literatures together and they make statements to the public like there is no science to assess this hypothesis. So even if we have the science—well, you saw [unclear] the ultimate decision is much bigger than science—I said, “How could he even say that?” When you supposedly have this model here you're supposed to be putting the science together then you make a statement like that to a newspaper—anyway—sorry. Don't get me started [laughs].

NL: Willy and then—okay.

MAUDIENGE: Willy Fontenot from Baton Rouge. I'm retired from the Attorney General's Office and I just want to say this is a fabulous panel—if we tried to pull a panel like this together thirty years ago we wouldn't have been able to get one—today we can fill this room with people like this panel and that's pretty exciting. The issue that you're struggling with is the regulatory bodies tend to become captive or captured by the people they regulate and that's true in the history of regulating the railroads and trucking industry and consumer safety, because the people in the regulating business develop relationships with the corporations—doing whatever work they're regulating, and they tend to develop social relationships and all sorts of things and so to be shocked by the EPA being

captured by industry means that you haven't read enough about how regulatory bodies work or don't work so you need to be as diligent—vigilant—

NL: [Laughs] [Unclear]

MA: —[unclear] are regulatory bodies free from being captured by the people that regulate this and worry about the materials they're regulating?

NL: Who wants to be the last commenter? Whoever raises their hand highest [laughs]—okay—you in the back there.

FAUDIENCE: Cynthia Rolette—University of South Florida. Seems to me that looking at the EPA as a really kind of distant windmill that we're all trying to joust against—it seems to me that the front line is in the doctor's office and if you are experiencing toxic reactions and—this has been my experience as well as many others I would imagine—and you want to get a test for a certain array of chemicals, the chances are if your medical practitioner's insurance payee or payor will not authorize it you don't have a choice and so how do we get access to this knowledge when really who's being captive is the medical practitioner with the drug company—with the insurance company. The frontline is right in your doctor's office and the idea that I could walk into a store and get a chemical test seems fanciful when I can't get one from my doctor, so I just want to bring that up [unclear].

NL: Okay, Jack really wants to make a comment—then we have thirty minutes for break and we'll be back again at 10:30 so Jack—yeah.

JM: I would say that the frontline is not the doctor's office—the frontline is in the medical school. Medical students are not being taught environmental health and the few public health and medical school departments in the country that do are under siege because they're not making—they're not falling into the parameters [unclear] care about the community [unclear] pharmaceutical companies.

NL: So, coming back to money is probably a good way to end this part of the session. There's coffee there—there's lots of coffee in the break room and I hope to see all of you back at 10:30.

[Tape off]

[End of Session #1]

American Society for Environmental History, Toxic Environments/Toxic Bodies Workshop, Baton Rouge, Louisiana, March 2, 2007. Morning Section #2, History, Ethics, and Environmental Justice.

NANCY LANGSTON: Okay, everyone—I'm going to get started now. I have a couple announcements—these are actually important announcements. Before we get started with the second roundtable—those of you who are on the fieldtrip—you

know who you are, right? Okay, those of you either you registered for the field trip or else if you're one of the presenters you're automatically on the field trip—in some of the initial information we sent to you it said 12:30—they've actually changed it to 12:15, so just get on the bus that says The Environmental Justice Chemical Field Trip rather than the Birding Field Trip. There'll be lunches—but 12:15 rather than 12:30—I want to make sure everyone knows that and we hope that most of you are also able to join us on that field trip.

Okay, the second roundtable will be focusing on communities—historians and scientists and two of the people who were going to join us, got stuck in snow so Michele Murphy is not able to make it and Pete Myers—they're both somewhere stuck in snowstorms, so instead we have the rest of the people on the panel—Scott Frickel has a Ph.D. from the University of Wisconsin—he's a political sociologist with strong interest in the politics of knowledge as it intersects environment—social movements—state theory—academic culture—STS and a lot of other things and John already talked about his book, which is—I haven't yet read it but I'm very excited—it's an extraordinary—I hear—book—about the intersections of much of what we're looking at. Linda Nash is an assistant professor at University of Washington—an environmental historian whose book *Inescapable Ecologies: A History of Environment, Disease and Knowledge*—came out last year with University of California Press and is a really extraordinary look at the links between permiobi—permeable bodies and environments and how those understandings have changed over time in the past century. Wilma Subra—we're very fortunate to have as we are for everyone—she's an environmental scientist and activist—she's a chemist, so I hope during the day today she can speak to some of the questions that one of the women in the back raised—she's also an environmental advocate—she received a McArthur Fellowship in 1999 and is president and CEO of The Subra Company, a firm based in Louisiana that has been extraordinarily active in working with community groups analyzing the scientific data—working with communities and really forming these links between sources of pollutants and effected communities. . . . And then Barbara Allen, who I think many of you heard at the Plenary yesterday—she is associate professor and director of the graduate program in science and technology studies at Virginia Tech. She got her PhD in STS from Rensselaer—from RPI. And finally, Michael Egan will be moderating today—he works in mercury—he's an assistant professor of history at McMaster University in Canada, and he works in science and technology and his book on mercury pollution will be out in a while [laughs]—

MICHAEL EGAN: A long while.

NL: —in a couple years. Okay. *A Global History of Mercury: The Alchemy of Nature*—and his book on Barry Commoner was published early this year just had come out.

ME: A little while—about May.

NL: A little while—oh, it's about to come out. Okay, thanks everyone and Scott. . . .

SF: Thanks. [Pause] As a sociologist, I study what I call—or have come to call—environmental knowledge politics, so I'm interested in understanding what kind of knowledge gets made—where it gets made—who gets to make it—who gets to certify and legitimate it—how it's used and who gets access to it once it's produced. I just have a couple of slides that I want to show—we got ten minutes, right—from a couple of different research projects that are currently under way. I live in New Orleans and after Katrina—as you might guess—my research agenda shifted radically to my backyard, and so two of the projects that I'm working on now are contained—encapsulated in the title of this short topic. The first project that I will show one slide of is a project about knowledge gaps, and in that project I'm looking at and trying to dismantle the EPA's environmental testing program that they conducted over the past year in Orleans Parish to try to understand what they know but really what they don't know in light of their contention that there's not really any problem with contamination or toxins in the city. And then the second project which I've talked about in more detail yesterday is a project I'm calling "Hazardous Legacies," where some students and I have gone back and tried to locate the places in Orleans Parish where hazardous industry has been located over the past fifty or sixty years and then we did a field survey to see what has become of those places in 2005—2006. So, I just want to show you a couple of slides from each of these projects that I think point to some important contradictions that really feed off of the earlier panel and particularly the questions of regulation—questions of the political structuring and unstructuring of knowledge and in this case as all of those things come together in the context of EPA's regulatory efforts.

So, this is a map that was produced for us by Rich Campanella, who works for The Center for Bioenvironmental Research—John McLachlan's group at Tulane, and what it shows is the cumulative sampling distribution of soil and sediment samples that were taken during a one-year period in Orleans Parish. Let me give you a little background about this: the testing program that was undertaken there following the flooding in two thousand—in August of 2005—was a program that was led by EPA but it involved, I believe, a dozen other state and federal agencies. So there was lots of expertise—lots of resources brought to bear on this project—testing evolved over a period of time there—it was a year-long effort and what you see here are the places in the city where soil and sediment samples were collected. There were in total over four hundred thousand analytic tests done for about two hundred different substances, from roughly two thousand samples and those samples are what you see here. So, this is the study on knowledge gaps and what I want to highlight for you are a couple of things that I think are distinctive about this. First, let me just say that from this slide that I've got the old Gentilly landfill—the Axe Street landfill and the Thompson Hayward—or is it Hayward Thompson—Thompson Hayward pesticide facility are three sites in Orleans Parish that are sort of known contaminated sights and you can see that there was a lot of attention around the Axe Street area, which is a housing development that was built on top of, ironically, a landfill from the hurricane debris from Hurricane—Betsy?

NL: Betsy.

SF: Right, so this is an old hurricane debris landfill—it was a site in the eighties and early nineties of a lot of environmental justice activism. Consequently, there is a lot of attention there, although a lot of that attention is because Wilma Subra made the EPA go there—she also made the EPA go to this Thompson Hayward facility and do some testing around the perimeters of this. This is a facility that's closed down now—if you go to New Orleans, it's a paved lot with a chain-link fence around it and some sort of stove pipe vents coming up out of the concrete. This was a facility where they mixed pesticides, including DDT and various other related compounds. The story I've heard about Thompson Hayward is that when they closed it down regulations for disposing of those chemicals were not on the books, so they just buried them there so there are tanks of DDT and DDE and various other pesticides on site there.

But I'm not so interested in where they tested them; I'm more interested in where they didn't test, so let me just point to two sort of spatial knowledge gaps that remain in the city. One is here—this is an area called Pontchartrain Park—you'll note there is no testing done in that area. Pontchartrain Park was a middle-class neighborhood developed in the nineteen fifties and sixties and marketed to and for middle class African-Americans before the storm. Pontchartrain Park was 98% black, I believe, and it borders the Industrial Canal, which is a sight as you might tell from the name. It's a site of heavy industrialization in the city, so we don't know anything about that part of town.

Today, Ponchartrain Park remains largely abandoned—there are very few people who have returned to Pontchartrain Park—it took a lot of water. But the other place that I want to focus on a little more carefully is down here in this area of mid city right sort of in the heart of New Orleans—not the cultural heart, which would be the French Quarter—but the residential heart of the city down here in mid city—stretching from just behind the Superdome all the way up to the edges of City Park and twenty or thirty blocks [unclear] wide there's a stretch there where no samples of any sort were taken. Now, one reason why they wouldn't sample there is because this is the bottom of the bowl and this was probably the last part of the city to actually become unflooded. But the entire city was unflooded in mid-October of 2005, and testing went on through June of 2006, so that doesn't really explain that. . . .

This is one slide from one project and let me show you another slide from my Hazardous Legacies project—this is a slide that I put up yesterday and this shows a random sample of one hundred hazardous industry locations in the city of New Orleans there. We found in our search through manufacturing directories two hundred and eighty-three traceable addresses but then we selected one hundred of those at random for more follow-up analysis. I guess the two things that I would note here is the concentration of industry along the Industrial Canal which we would expect—and also the concentration of industry of old hazardous industry. This is not to say that these places are necessarily there now but they have been there in the last fifty years—right here. It's the same place. [Pause] So, so those are the two slides I want to show and then think about—I just want to say another couple words about the logic of EPA's sampling.

If you read their documents and they're sort of the summary reports that they produced they state very clearly that the testing was primarily focused in residential areas because they're worried about returning residents' exposure to these hazards if they're there—so you see, for example . . . I guess the other thing about this map that I didn't say but I hope was obvious—is that it shows the extent of the flood, right? So, you can see there was no testing done in the part of the city that didn't flood, although historically and this is also true in my sample of industry sights—industry concentrated along the river because it was dry—but there was no sampling there and presumably there was little sampling up here, for the same reason. Maybe there was little sampling there for that reason although mid city—for anybody's who's familiar with it—there's a lot of industry there—there's a lot of commercial area—there's a commercial corridor going through there—but it's a very residential part of the town. At any rate, the idea or the logic that floods—that testing in industrial areas isn't important because people don't live there, is sort of belied by the systematic logic of the early phases of their testing, which was—now you can't really see it from this map because I've—it's a cumulative map—but if I showed you a map of the first stage of testing you would see very evenly spaced dots covering most of the city, because they took a very systematic approach on the logic that the flood waters diffused contaminants across the city. So, there's a fundamental contradiction in the logic they're using—on the one hand, they're saying we're worried that the flood water would diffuse the toxins or the contaminants if they're there, and in the same documents, they're saying we're only testing in residential areas because that's what we're concerned about. So, I just want to throw that out and—and I think I'm done so thanks.

NL: Thank you.

LINDA NASH: I don't have any fancy slides or anything, because I'm a historian and we tend to work with text, but I just wanted to say a few things about the usefulness of history because whenever historians get drawn onto the terrain of the contemporary world we're often seen as sort of interesting people to have around - I think we're considered interesting generally and people like to have us at cocktail parties because we know odd historical facts - but they're never quite sure that we have anything to tell them that is of relevance to contemporary problems like the one that we're discussing today. So, let me tell you what I think is useful potentially about history and . . . let me also clarify that I'm not talking about the kind of evolutionary history or biological history that John McLaughlin was talking about but more particularly about the cultural and social history, which is my own field, particularly the cultural and social history of the US. So, what that can reveal I think is, first, the contingency of the present, and by that I mean the fact that current events or the current situation didn't have to be as it is—in other words current environmental conditions did not necessarily have to be what they are, which I think we all would agree on, but also and perhaps equally important, our way of thinking about environment and disease did not have to be what it is. In other words, there are other possible ways that we could have come to think about environment and disease in 2007.

And that leads to my second point about the usefulness of history, and that is I think we can in some cases mine the past for alternative models or other ways of thinking about things, and I think that becomes possible once you let go of the progress model of history. Now, for environmental historians that comes somewhat naturally, right? We're not very invested in progress, but for medical historians and historians of public health I think it's a little bit harder, because I think that group of historians is somewhat more invested in the progress model as are those professions. So, for a brief example, let me just talk a little bit about my book—I try not to shamefully self-promote it, but it is available for your review on the Scholar's Choice table. My relationship to these issues comes through my book so—you'll have to bear with me. In that book I tried to historicize understandings of disease from the late nineteenth century down to the late twentieth, with a particular focus on how environment was or was not considered an important component of health and disease.

And when I looked at nineteenth century understandings of disease, what I found was that nineteenth-century individuals, both medical doctors and lay people, located disease and health in the environment, and usually we dismiss this as wrong, right? I mean they believed in the miasma theory—they didn't understand how malaria was transmitted, which is true, but what I ended up arguing at least in part was that the concern of nineteenth-century doctors with environment was nonetheless something very valuable, despite the fact that they didn't understand the malaria parasite and they didn't understand the mosquito vector. Nonetheless, they—while they were wrong in a sense from the contemporary perspective—they were, I think, right to pay very close attention to local environments and ask how environments affected both health and disease.

So these nineteenth-century doctors went out and they said, you know, "it's as important to bring along your thermometer and your rain gauge as it is your speculum and your stethoscope," right? I mean they really believed that it was their duty to measure and assess and pay attention to environments and to environmental change and how those changes registered in health. Now, germ theory and modern laboratory medicine—which arose gradually at the end of the nineteenth century—made possible many important discoveries (and I don't want to dismiss the importance of those), something equally important was I think lost or at least sidelined, and that was the concern of the medical and public health professional reputation, with environments—the explicit concern and focus on environment. Doctors moved into the laboratory. The laboratory became where you made your profession—where important discoveries were made—environments were left to people like sanitary engineers who have far less professional status than do laboratory doctors.

Now, when I talk about germ theory, I'm using it in a broad way— and fortunately we're not medical historians here—we'd spend all day talking about what it meant—but I use it just to indicate the belief that diseases are etiologically specific. That means are caused by a specific agent that it also refers to a very individualized model of disease. So under this model disease is contained in the interaction between this agent—this germ if you will—and an individual body—

in other words, the broader environment is no longer implicated in disease production. It's the relationship of this single agent—the germ with the individual. And also another implication of that is that the healthy body is therefore a pure body so in order to maintain health you need to keep these germs out of your body.

Now, germ theory is not wrong—let me be clear—I'm not arguing that germ theory is wrong—but it is just one model. But because it emerges at the moment of professionalization and the moment of the progressive expansion of the American state, I think it becomes easily intertwined with certain professions and institutions—medicine, public health, the regulatory state, and what you see is that medicine essentially takes over disease—says we're in charge of sick bodies and sick people, and it largely excises the environment from its realm of study. Not completely because it can't, but it really sidelines it and that has the effect, I think, of working much better for certain diseases than for others. Certain diseases really work well when you put them in a laboratory model—it's very good for smallpox, but probably less good for diseases like malaria, hay fever as Gregg Mitman is showing us, cancer and other types of diseases that don't fit neatly into this late nineteenth-century model. Now, environments—despite the efforts of these early late nineteenth- and early twentieth-century doctors—I don't think they ever are completely written out of the picture, but in many cases I think modern medicine and public health try to assimilate diseases like malaria to the germ model even though the fit is not that good and I think John referred to this when you talked about your early work in eco—whatever it's called [laughs]—but, you know, your early work tried to look at the effect of estrogen when you're using an older model derived, I think, in a large part from these early germ discoveries.

But that doesn't quite fit, so you have to keep expanding it to take account of the complexities that the original germ theories did not really anticipate or hold so well. So, consequently I think we, maybe less so the professionals like John who really have to look closely at what's going on, but I think the rest of us still tend to define the causes of disease quite narrowly and we want to locate it in specific germ like agents, for instance, in a chemical and again in an individual body—a susceptible body. So we want to say okay, the chemical gets into our body and makes us sick—that is the germ theory model. But the message of nineteenth-century medicine I think was that environmental arrangement always mattered. Not specific etiologic agents, but the broader relationship of environments and bodies together, and that those arrangements always had health effects.

Therefore, whenever you had rapid radical environmental change whether it was nineteenth-century deforestation or the proliferation of plastics after World War II, from a nineteenth-century medical perspective you would expect, you would assume, that those were going to have health effects. Modern medicine and public health I think are not at all that well equipped to address that fact the assumption is that environmental change does not necessarily have health effects, right? We have a different starting point. We assume that environments are not

necessarily related to bodies unless we can show the precise ways that they are, and if we don't have the precise mechanisms of causation, we don't really need to consider the environment.

And I think because of that particular history, modern medicine and public health still remain closely wedded to the germ model and even when environment gets taken into account, it is only when it can be abstracted into a single chemical—a single entity that can be moved into the laboratory and examined. This reminded me of Arthur's comment about body burden profiling as a way—as something that might in the next ten or twenty years make us all very aware of the environmental effects of chemicals on our own bodies, and might lead to a demand for political action. I think what Arthur is arguing there is that as we become aware of how infiltrated our bodies have become, we will redefine the problem as a social and cultural one, as an environmental and cultural one, rather than as an individual problem of health and disease. That's a very optimistic assessment and let me say I hope Arthur's right, but I would just point out there is an equally powerful model coming out right now, which is that of toxicogenomics, which basically moves us back to the individualistic model of health, which tries to assess people's susceptibility to individual chemicals.

Now, there's a lot of value in that on the one hand, right? To know whether you're susceptible to certain chemicals or to certain cancers might lead you to adopt certain behaviors that could protect your health. On the other hand, socially I think it has a radically different set of implications, because if we define the problem individually the solution again becomes individual: you need to protect yourself; you need to stay away from certain chemicals; you need to stay out of certain environments or eat certain foods. Whereas if we defined the problem socially and environmentally the implications are much different; corporations need to stop producing these chemicals; we need to pass a different set of regulations; we need a new legal structure that hold corporations and companies responsible for the environmental toxins that they release.

So, where does this lead? Are there any uses for history in the end? Well, there might be a few things that you could draw from this very brief overview, and one might be that we should have health officials more involved in [issues] like urban and environmental planning, and in fact I would point out that one of the NIEHS initiatives in recent years has been to actually do just that—to get public health professionals involved in urban planning as a way to prevent the development of diseases like diabetes and other things, which are associated with how people go about living their lives.

So, there's a new emphasis in public health departments to sort of assess—to try and get involved in the urban planning process. So that's one possible implication: an attempt to bring the disciplines that study health back together with the disciplines that study environment—the things that got separated I think in the early twentieth century. It might also argue for a different kind of education for doctors and public health experts as Scott mentioned at the end of the first session. It suggests that maybe doctors and public health experts need to actually think about environment as a fundamental part of their education rather

than coming to those questions quite late. I think it's a broader way of phrasing the question that Sarah asked about whether toxicology is the appropriate discipline to undertake this kind of research—I think we maybe need to think differently about how we organize knowledge production in the university if we want to address these kinds of questions.

It might also suggest the need to push for a chemical regulatory system that is not so wedded to the individualistic model of the germ, and this I think goes to Tyrone's criticisms of the EPA—I think if you look at the history of regulation within the FDA and the EPA, it's really quite clear that the individualistic model of disease that those agencies have based their regulatory structures on has been highly beneficial to the chemical industry, which has been able to manipulate the uncertainties in that process, and I can imagine or at least I can start to imagine ways in which we might design a regulatory structure which would be very, very different from the one we have, which wouldn't look at individual chemicals so much but which might be completely different, and I think that's perhaps another thing that history at least asks us to consider. Or like most environmental histories maybe this is just another argument to slow down the increasing pace of environmental change on which modern capitalism seems to thrive.

WILMA SUBRA: My name is Wilma Subra; I'm from New Iberia, Louisiana. The hurricane hit a year and a half ago yesterday. I was in the field within forty-eight hours doing damage assessments—needs assessment and collecting samples of sediment sludge, and this is what he was referring to which was brought on shore from the tidal surge that hit the whole coast. I was in Alabama, Mississippi, Louisiana and Texas and what you saw is just a little piece of that but today's not the hurricane issue—today is the issue of a company known as Shintech that Barbara featured in her book—the vinyl chloride and dioxin emissions. A troubling sort of process that's evolving in the environmental justice movement and then associating body burdens to an industrial facility so having the body burden data and being able to identify what industrial facility that came from. . . .

This is the Mississippi River corridor—these are the parishes along Mississippi—we are in East Baton Rouge Parish—the numbers there are the number of major industrial facilities that release toxins into the air, land and water in each of the parishes, and again, the facilities are located right along the Mississippi River. But what I'm trying to show you here is Shintech was proposed in St. James Parish in the community of Convent so that's Shintech One—and then it progressed over to Shintech Two and Addis in West Baton Rouge, which is just across the river—and then Shintech Three in Plaquemine—a potential Shintech Four again in West Baton Rouge, and then we go all the way back to the Shintech location where another industrial facility is being proposed. Shintech One was a vinyl chloride facility in Convent.

It was proposed to manufacture chlorine—EDC—vinyl chloride monomer and poly vinyl chloride. It went away in 1996 and then we have Shintech Two—vinyl chloride facility—it moved across the river to Addis—just across the river from here in West Baton Rouge, and Shintech said well, now that we have that

big facility we're just going to do the last process—take vinyl chloride and make poly vinyl chloride. They began operating in 2000, and the releases in the year 2004, which is the latest data—vinyl chloride—six thousand and eighteen thousand pounds a year of vinyl chloride . . . . Shintech Three was proposed in December of 2004 slightly before the hurricane, and they went back to the big facilities—all the units—chlorine—EDC—vinyl chloride [unclear]—poly vinyl chloride—public hearing in April 2005—current under construction proposed 2007 and this is where the EJ perspective stop. Shintech One in Convent—all the people were opposed to it after they learned what it was about. A lot of the national groups came in and supported them and it was a really big deal—when they moved across the river to West Baton Rouge and Addis the national groups didn't have that much interest but the community said no to Shintech—over and over again the community said no, and of course the agencies permit it. And then here comes along December 2004—Shintech comes in proposing the big unit again and everybody in the community from Chamber of Commerce—school boards to EJ communities said we told you no last time Shintech—we welcome you in and in exchange for that welcoming in, they didn't ask for anything, because Shintech had already told them they would get the jobs.

So, doing the technical working with Environmental Action Network, I sat down with Shintech and said these are the things we need: reduced emissions—reductions in all of these processes. If you're going to have your permit, the least you can do is control the emissions. And yet the community hated us—they said we want Shintech no matter what, and so that started the EJ movement just accepting without trying to make it a cleaner plan. Okay, can you go up? This [shows] the emissions—twenty-nine tons a year of vinyl chloride—vinyl chloride's a known human cancer causing agent and angiosarcoma of the liver—it's one of the few chemicals that we know of specific health impact and that EDC six tons a year and then dioxin they didn't happen to tell us what the emissions of dioxin were going to be.

They say in the one in Addis that they only make the final poly vinyl chloride so they're not releasing dioxin and I'll be going around and around looking again, but again the vinyl chloride and the dioxin are the real health-associated agents and then we have a potential Shintech Four—while they were buying up property on that side of the river, they bought the former Borden Chemical Plastics, which made poly vinyl chloride so that's a potential four. And then making the loop all the way back Tiger comes along late last year—wants to locate on the same property in Convent that the big one was going to be. They're going to make a hundred and ten million gallons of fuel grade ethanol—permit process was fast tracked—less than three months from the application to the public hearing—they projected to abide to a lesser apparent impact so they were reducing everything. They only applied for one so they were minor [unclear]—they obtained the minor permits that are less protecting.

Here the community said we love you—we want you in—we want parks and we would like a health clinic. Well, Tiger said we haven't been here all these years—we're not responsible for the health impact and maybe we'll consider a

park—so again the EJ community said come on in. Working with Lee, we asked for the better controls, but the EJ community is like settling for a park and not requiring reduced emissions and what I'm scared is going to happen is like benzene causes leukemia in kids—it's okay if our facility releases benzene as long as you put a health clinic, so when my kid gets benzene I can go to the health clinic.

And this just shows you the property at the St. James that was on health [unclear] and Wilkins Sanitation, and that's where the ethanol plant's going to be and if those of you from the area have been watching, our governor is proposing a steel mill and that's where the steel mill's going to be. The white people who still live there said buy us out—we're tired of fighting—we want out—and the African-American people are saying we want a park. So, vinyl chloride and dioxin air emissions are huge—West Baton Rouge is one of the highest areas—these show you what's coming out of the Shintech facility on a yearly basis—this is when they went online—Dial—and we're going to be going by Dial—that's a town made ground water a trailer park all the way from their facility. And here you see the quality of vinyl chloride and the—the next one will show you the dioxin emission. That just gives you some idea of what's in the air—the Georgia Gulf Facility in Plaquemine and then Shintech will have fifty-nine million pounds of vinyl chloride in the air.

MAUDIENGE: What's the difference between fugitive and stack emissions? . . . .

WS: A stack is what you usually see—fugitive comes out of a connectors go together—they usually closer to the ground—migrate out, okay? So, this just gives you some idea—we've done a lot of monitoring in the communities. We know that the concentrations are way over the edge in [unclear] and in other areas—West Baton Rouge—Giverdale area has high concentrations of vinyl chloride and dioxin being released—Longville—[unclear] Parish—that's where the body burden that I'm going to talk to you about—East Baton Rouge—Ascension. You see that there are cluster areas—hot spots—in Louisiana where the dioxin emissions and the vinyl chloride emissions are really, really high. So, APSGR did a study in Marksville Township on the Rusty Farber estate—they did ninety-nine—'97, '98 they did twenty-eight people—African-American people in Marksville—the average dioxin concentration was 68.5 based on TEQ's parts per trillion—more than three times the average of a national comparison group.

Then they came back in 2001 and they were hoping the levels had gone down, because we got the community relocated so at 2001 twenty-two of those twenty-eight people were tested again—the average came down to sixty-one; still three times the other comparison annual group. So, I went to APSGR and asked them specifically for the [unclear] the little pieces of the dioxin and they gave it to me for each of the individual people. So when you look at multiple blood serum this is the [unclear] that was responsible for the most toxicity in the blood—42.7% in the '97-'98 42.7—the second one—this is what people call TCDD and this is what all the [unclear] are calculated back to—eleven and 11% and then three—can you raise it a little bit—three of the—added up to another twenty [unclear] percent. I compared those, so seventy something percent of the

[unclear] that cause the toxicity in the blood will need—compared to Georgia Gulf which is the bottom [unclear] manufacturer—can you raise that a little bit—and you see the same thing—the that they reported—35%—34% [unclear] degree so Georgia Gulf matched the top so 70% in the blood and 70% of the emissions matched. So Georgia Gulf is the one most responsible for causing the elevated levels of dioxin [unclear] in the blood of the people in Marksville and remember when I showed you all the other hotspots? Those are the ones who were working with APSR to go back in and sample the people in those communities. . . .

NL: Thank you. Barbara.

BARBARA ALLEN: My name's Barbara Allen and I'm the director of the science and technology studies program at Virginia Tech's DC campus, and I've been studying the science wars in Louisiana for the last five or six years. There have been a number of projects that I've been doing and it's a good test set for a lot of the problems between doing laboratory or traditional science and more popular epidemiological or citizen science. My own interest is in the citizen use of science—citizen understanding of science and citizen actually making science, so I want to talk a little bit about it and see what I can cram into my allotted time. I'll talk a little bit about what's been happening down there in the past or in the recent past, and maybe say a couple of other things in response to my speakers here.

I want to talk about the politics of method. I call it the creation of fiction or is there really a Cancer Alley because the state will tell you there's no proof of the Cancer Alley area of petrochemical plants along the Mississippi River, where people live between Baton Rouge and New Orleans. They will tell you there's no truth that it's a Cancer Alley and I'm going to come back to that in a minute. This is where it is. First I want to talk a little bit about what popular epidemiology is—I think that citizens participating and making their own science and in collecting their own data is a very powerful tool, because it can go straight from the citizens' own laboratory if you will—the laboratory of the world or in the environment—to the policy makers. . . . The woman in the middle—her name is Florence Robinson—if you were at my talk last night, you know she's a retired professor of biology at Southern University. She lives in a very contaminated community—lived—she's actually moved—it's a Superfund site now, but where they collect hazardous waste and process hazardous waste.

So, she began just collecting data. She suspected that people that live near these sites, that live near the most polluted areas, were predominately African-American, so she put together a very simple chart. She would put together charts of her neighborhood, and she would actually have people go around and ask people in their houses what diseases they have—what illnesses they have—and collect this information and again take it straight to the policy makers. And you know, you would think in your mind the best thing to do is to get this and this would be a reason to get the state to do a study and wouldn't that be great then they would do a real scientific study—well, I'm going to show you why I think that's a bad idea—a really bad idea.

And then we have Wilma Subra . . . one of the things that Wilma is famous for in this state is she goes to these communities and she doesn't do their science for them in a sense—she might give them some data and it's up to them—it's up to the community members to begin to organize—to begin to put this in some mechanism—in some kind of format that they take to their policy makers, that they take to their legislatures and that they begin to use to fight the system to clean up their environment. So it's not that Wilma's out there doing their science and fighting their fight for them—she's providing them some baseline material so that they then can take to their fight. Another tool in this area and this is a map from the Shintech study of popular science and I say that in a positive way—popular science—it's putting the community at the center of investigation.

What the chemical companies will do for permitting is they'll come in and they'll say, "Here's what we're going to release." It's sort of like well, we're going to have this many tons of this and that many tons of that and what the citizens really want to know is not how many tons that plant has or this plant and that plant, but what's the cumulative effect at the center of their community. In this case the center of their community they decided was their school, so they looked at their school. They don't just want to know about Shintech; they want to know about the other twelve or eleven facilities and then how does that cumulatively—and those are a different set of questions and can be presented differently to policy makers.

Now I'm going to get in my little conundrum of traditional science versus the popular science. A number of years ago, in the late seventies, a woman by the name of Kaye Goday was a pharmacist—is a pharmacist in the St. Gabriel area—a place of about twelve to fifteen chemical plants. It had a huge concentration of chemical plants and they moved out there because they wanted to live out in the country and her and her husband were both pharmacists. They had a small business, but she started noticing and she's hesitant to say. I've interviewed her several times, because there're certain privacy rules in the US but in parts of her business and in part through being in the community, there're a number of miscarriages—they seemed really out of place—she was very concerned and she started talking to other pharmacists around, and she actually found other things like an elevated level of breast cancer drugs being distributed by the pharmacies and she just thought that there was definitely a problem.

So she collected enough information and she kept a journal—she journaled all of this and she would notice when there was really heavy releases these miscarriages would follow pretty soon after—she had really, really extremely detailed records. So, she went to the state and after a lot of threats, and she finally was actually going to go testify before a Washington committee . . . They actually hired the Tulane School of Chemical Medicine to do this miscarriage study and the study was—they picked an area was epidemiological area of study—they picked an area that was large enough to get a population that they could get statistical significance. So they couldn't just pick the population around the chemical plant; they picked the entire half of the county—the majority

of which didn't live by the chemical plant—so their statistical sample really was irrelevant. . . .

The second thing that they did is they went around and they sent these researchers—and they even admit this—the researchers were predominately white and there was an underreporting of African-American miscarriages, because in that cultural community to have a miscarriage was considered a failure and I'm not sure they would tell some white researcher about such a personal thing. So that the study itself admits that they grossly underreported African-American miscarriages; this isn't a mixed community. So, they came out with a study—it took them ten years—almost ten years—saying that there is no elevated miscarriage rate. It's all okay, you know; we did this study and so they used science in such a way to create such a huge population so that the study could be statistically significant—they collected it in such a shoddy manner that they didn't even show the miscarriage rates of the majority of African-American women and then they called that science and this was used as a tool to do nothing—well the science shows that, blah—blah—blah. So, for years citizens have had concerns about elevated rates of cancer in the corridor—they've even named it Cancer Alley.

There was actually a particular interest and I have so much anecdotal evidence about this—I mean it's overwhelming when I think about it, that there's really elevated rates of rare cancers in particular pediatric cancer I believe is off the charts in this area. So, about . . . eight or nine years ago a group of citizens and medical researchers brought a law suit against the Louisiana Tumor Registry who are the people that collect cancer data in the state paid for by the state tax payers. They wanted them to change the way that they release data so they could begin to see by zip codes can you see cancers in these zip codes around these chemical plants and in particular they were interested in these rare cancers that could be linked to the chemicals . . . —one of the medical researchers leading the group was really concerned with pediatric cancer and that's what she wanted to study. So, the citizens living near the plants wanted to know how their daily exposure of the plant pollution affected their family health—they were suspicious about the cancers, rashes, asthma and other neurological disorders, and could those be related to the chemicals. And was there some way to produce enough evidence to mandate stricter regulations for these communities?

So, here's what they wanted—this sounds pretty simple—they just wanted cancer collected in one year increments—at the time they collected cancer in five year increments—they wanted it collected by parish, which is like a county and zip code and they wanted the data for that and they wanted cancer data showing even rare cancers such as pediatric cancers and they wanted cancer data for Louisiana residents who died in out of state facilities—some of the big cancer treatment facilities—Indiana [unclear], Houston and there's other ones. They weren't collecting those people—I mean it was absurd. The way that they were collecting data—they were doing it in five year increments—they published the data in large geographic multi-parish regions and literally they gerrymandered these parishes together so that you would have an industrial parish all connected

with these sort of rural non-industrial parishes, and they would create this sort of mass data that meant pretty much nothing.

I interviewed the head of the LTR and well, you know, the important cancers are lung cancer from cigarette smoking and I guess those kind of cancers you can do that way, but that isn't what these people were interested in and then they only listed like the five most common cancers—well, in pediatric cancer there's sixteen kinds of pediatric cancer. I mean there's a million kinds of cancers, so by picking the five yeah you get the lung cancers and the breast cancers. But the really rare cancers that are related to these chemicals don't show up. Okay—so, here's the reasons they gave and I saw all this because the court documents—they went to court many times—multiple times—they said that to get the zip code data would be really expensive for the citizens of the state and that they only collected in five year increments.

How do you collect something in five-year increments and to release the data in any other form you'd have to get this software program and it would be really expensive as well and then—this is the best one—to release data of rare pediatric cancers it would violate a patient's right to privacy because you might have a zip code in which one of the sixteen pediatric cancers only occurs in one person and that's actually likely but if you start to see these different pediatric cancers for these. . . . I mean that's huge, because they shouldn't occur at all so that was astounding and then they felt that the scientific community regarded zip code-specific cancer as statistically and clinically meaningless that these people want to create junk science—that's the other reason we don't want to give it to them.

So, the citizens argued that they needed the information—that a patient's right to privacy would not be violated—because there's no way to tell—you'd have to know who this child was—and I mean you'd really have to be a detective to figure out who that one pediatric cancer was—that specific kind of cancer in a community, and they maintained they were not using the data as causal evidence. This is what the LTR was afraid of—they were going to start making causal claims, very expensive causal claims but they wanted the information to push for further funding or further regulation. So, in 2004 the state court made its decision—they must release the data by parish, so they had to give up that ghost of a giant multi-parish region. They had to release it for all cancers they couldn't just give the top five, you know, the top five greatest hits of cancer—they've got to give all the cancer, but they did not have to release it by zip code. They actually bought the cost argument—too expensive—even though the actual data form that the computer input people use because they had this [unclear] zip code is one of the criteria so I don't know how the judge got talked into that one. They agreed to finally go and collect data from out of state facilities where Louisiana residents had died.

So there are a number of issues raised by this, how right to privacy has been used to hide data from people—how lack of standards make aggregating data across like exposed populations different and the over interpretation by the Louisiana Tumor Registry. And it's odd that they're over interpreting because

what they used to say in all of their literature is there is no evidence that there is a Cancer Alley—well, the opposite claim—you can't actually make the opposite claim, either—you can't make a claim that there is no evidence for cancer in Cancer Alley can not be turned into which it has been by state regulators is there is no evidence of cancer in Cancer Alley. The reason there is no evidence of cancer is because they've hidden the evidence that we don't know—we can't even find out, so I think I want to end there and—.

NL: Thank you. Michael's going to moderate the discussion .

MICHAEL EGAN: Well, I want to start actually by thanking Nancy and Jody for putting together these twin sessions—they're absolutely tremendous [unclear] [tape off/tape on] [End of Side A, Beginning of Side B] so we should probably skip out of here so we are not late for the bus. Obviously, we can continue this conversation on the bus to and from. All kinds of things going on—I think what we might [unclear] do here in terms of question and answer is try to make connections about issues of science and uncertainty in science—increasing notion about, I think, expanding peer review with pros and cons involved in that kind of a process—how we interpret risk—whether there is a statistical or scientific method for doing so, or to what extent that's a social or a moral question or point of examination, rather than strictly a clinical one and countless more but I'm going to shut up because we've got a really interesting series of questions on the table [unclear]. Again, if you can please stand and give your name and speak clearly [unclear]—sorry Chris.

MAUDIENGE: I am Chris Sellers and it occurs to me I've been stuck in the fifties and sixties, and it occurs to me I see a lot of actual continuity and repetition here—a lot of what we're hearing about, this new particularly the endocrine disruptors argument, but the whole scientific—the whole kind of [unclear] of scientific—science and politics as well and one of these is the—I mean of course this is the era of *Silent Spring*, right but that's only the tip of the iceberg [unclear] what's going on in that period I don't think it lays the foundation for where we are today with some of these things, so just some points of comparison to try and draw some parallels, but also the contrasts are really interesting too. In Hilton novelty was there in the sixties and it's kind of extraordinary that Luther Kerry, the surgeon general who published this [unclear] smoking reports also talking in apocalyptic terms about synthetic [unclear]—the chronic exposures—that's a big concern—the uncertainty of these exposures—the evidence for parts from [unclear] out there for DDT and [unclear] parts per million in the mid-sixties turns up in the Antarctic and so forth—low doses as well as [unclear].

The differences, though, those are fascinating. I think how the public presence of talk about these risks in the nineteen sixties. It's right here—pollution is the number one public issue where people mentioned in a poll in 1970—just extraordinary they pass the baton [unclear] and, you know, how did that happen—I've spent like fifty books—popular quasi-type of books on pollution between *Silent Spring* in 1970 and this is something that we see here in the presentations we've gone through but it's really more segregated [unclear] in the environmental media, and you don't see it on the *New York Times* [unclear] page in the

newspaper. So I don't quite understand [unclear], but it's something to consider. The other thing is the object of concern—I mean cancer in that period obviously was the big topic of concern—chronic disease—they were just figuring out how to track the causation in long-term exposures linking them to chronic diseases. Smoking is the obvious example and asbestos, so studies were just coming alive and I think that triggered a lot of the anxieties about chronic diseases—we carried out actually the Department of Justice [unclear] but the concern here about the endocrine disruptors and sort of gender blurring [unclear]—it occurs to me this is the moment in which that becomes visible—there's all sorts of other things going on in the other history [unclear] that makes this beast more visible. But it seems to me looking at this panel as a whole that this is the kind of board old fashioned focused on the environmental justice and cancer and diseases that kill you and—and gender blurring may not quite translate the same way [unclear] but just sort of speculation.

- BA: I think one of my concerns is that if there are where we're seeing second-generation effects from endocrine disruptors and the kinds of things they were talking about in the earlier panel, but my concern would be if history—if recent history played out in that particular milieu, what we're going to see is the same kind of action where it's science got an existence. Oh, we'll do the study, you know, and the—the state or the regulators and the corporations get together and they concoct the study of endocrine disruption and the problems—and low and behold, you can construct a study that shows the abs—and once the science is done, and by Jim by the state or by the government then it's sort of put away; it's put on the shelf. So, my concern is that we'll see a repetition of what's been done with cancer in endocrine disruption and other kinds of environmental health issues, because it's worked for them in the past to do nothing like creating these studies, so why wouldn't it work for a future [unclear].
- NL: I want to jump in and add a comment that I think we all know, but it's good just to bring out that the scientific community is no more unitary than any other community, and we have enormous numbers of scientists working on endocrine disruption who are doing all sort of very sophisticated work very much in tune with communities. But there are definitely also studies funded by industry—studies funded by government—that do their best to show a lack of response so it's complicated.
- SF: Can I just throw something out as well? I think Barbara's comment highlights a concern for me, which is there always seems to be this push, and I see this a lot in the biomonitoring literature, that there's this push to know more. What's great about biomonitoring is it allows us to know all this information about what's in our bodies but what are we going to do with it? What's that going to get us? I personally see biomonitoring at least in the way it's evolving now as a distraction from the old kinds of hazards and the old kinds of—you know, we know we a lot—Chris was just saying—we knew a lot of stuff back in the sixties and so what, you know, we still know a lot.

MAUDIENGE: Barb, I think I mentioned this to you before but you—you're absolutely right on point about the [unclear] blastoma's and [unclear] lymphoma's clusters—I think that scares the hell out of industry and the health department [unclear] and about Cancer Alley years—and I think I told you this, but years ago I had somebody—a medical person with the state who was telling me that it isn't Cancer Alley, it's that people there—there's not a higher cancer rate in Louisiana, it's that people are slow to get medical services and that there are more cancer deaths, but than there should be but not higher rates and I said, "Oh, [it's known] now as Cancer Death Alley [unclear]. We can put that on the front page of the paper tomorrow morning and I'm sure people will feel better."

FAUDIENGE: Eileen McGerty—I spent [unclear] about how much, you know, [unclear], that we knew [unclear] this kind of actually [unclear] earlier [unclear] when TOSCO was first passed the idea was that actually to look at the [unclear] the legislation for TOSCO was that we have all of these chemicals and we don't know what's happening. We have to stop making these chemicals until we know what's happening—because we had indications vinyl chloride information was available [unclear] variety of other individual chemicals. So, what's interesting is how that emphasis like [unclear] said the initial intention and the scientists in nineteen seventies when they wrote the first [unclear] report how it completely shifted as the regulations tended to sort of insulate them and to a large extent [unclear] those industries [unclear] on those relationships come into play, but also to a certain extent about how to do the science for this.

In some ways, the regulators—when they were setting up this regulatory structure—really didn't even know how to go about doing this, because it was such a complex problem, as you know, and then the other thing I wanted to say was also [pause] how I'm interested and I would like to hear [unclear] about how ambivalence that environmental [unclear] regarding the use of [unclear] the whole sort of uncertainty aspects of it and how it can actually sometimes backfire, because of these issues that we're talking about in terms of a strategy of trying to get a certain change or regulation or whatever is stopping that from happening that we actually kind of move away from being [unclear]. For example, this is [unclear] that actually have a lot of information on their website about why you wouldn't want to do this kind of science—not saying that you shouldn't do it but there are problems with it so you better use that as a strategy.

ME: [Unclear] Yeah, Linda—sorry.

LN: Well, in response to the public presence issue which Chris first raised and you reiterated, I'm beginning to think that maybe the worst thing that ever happened was the creation of the EPA—which I say somewhat facetiously, but only somewhat, in that I think it actually helped create a sense of complacency because people feel that the government is taking care of them. We want to believe that the science and the regulatory process is so complex that it's very difficult to understand unless you devote your life to it to understanding it. And so then there also are also the distractions of consumer culture, which I'm not going to downplay, but nonetheless I think the creation of the EPA and the sense that the government was addressing the problem has given us in this era a sense of

complacency that I think Carson's generation didn't have because there was nothing like that. They needed to create it and so I think maybe that was a terrible thing and then [laughs] and—yeah—and I don't know if I'm the one to address the science in Susan's activism question so much, but I do think the regulatory capture model is too simple for describing what's happened with the EPA.

Having once worked at the EPA, I can't accede to that model, because there's a lot of people in there who are very well intentioned and really are not captured and really want to do the right thing. Nonetheless, the agency's very mission, as well as its implementation and enforcement strategy, have been shaped by a court system in which industry has won time and time again. So—it's not that the agency itself is captured; it's more—the argument I really like is Ted Porter's "trust the numbers," which basically says, you know, science and technical approaches to problems—scientific, numeric approaches to problems, quantitative approaches to problems, are not something that bureaucrats and experts impose on the public; they're something bureaucrats and experts do, not because they have power, but because they don't have power and they're actually trying to wade through sort of a complex political and social situation. And they're trying to find ways to gain power, and they see these numeric quantitative methods as their best hope for that, because they're respected in the arenas in which they deal. I think we could use that argument to understand how regulatory structures have evolved in this country.

ME: I've adopted John's three-finger trick and I've got to stop at three—John, you're actually first on the list—Gregg and then Alan—we'll come around—it's actually handy—it works.

JL: Yeah, I know—you have to remember that last week.

ME: I want to stop it at three.

JL: I grew up in New Orleans. I want to make two short comments speaking from a scientific perspective about how to close the gap between community action and basic science, because this comes up over and over again. The first relates to the problem with language and we actually are working at our center with people in the humanities and the arts to create what we call a language of [unclear] for scientists to talk about what they're doing in a way that makes sense to real people. And I think that's been a huge disconnect always and I think that Tyrone's presentation is a good example of how a scientist is doing cutting edge, really good work in explaining to people, so everybody gets the same idea and have the same sense of passion, and yet so subjective that it's also passionate and I think that's a critical issue we have to work on. The second, above all what Linda said about the germ theory—I think one of the problems with the environment and convincing people to do something about the environment is the very fact that we're still sort of medieval in our theories, and we don't have a germ theory for the environment—we don't know anything about genetics for the environment.

If you think about it, the other piece besides the germ theory was [unclear] in the forties, fifties, sixties—especially seventies—the idea of chromosomes—

genetics and now everything's explained in terms of human disease by germs or by genes, and that if you go back historically to medieval times with the bubonic plague what did everybody do—what did the public health system do whenever people were dying of the plague—they would fall on their knees and pray, you know, dear God please take them and not me or they would burn your house, but before we knew about germs, we had no rational way to think what to do—to say what to do or to add any preventative measures.

After we understood germs and now that we understand genes, we can say convectively here's what'll happen if you have a chromosome twenty-one q abnormality: your baby will be born and she'll have—as my niece was—a crossed blood vessels—it will be blue and we can do such and such right at that time—I think the big thing for us in environmental programs is to bring science to some point where we actually have a germ theory of the environment, so we can do things that are predictable—we can tell people what actions they should take. I think that's really an issue that we've got to struggle with against your historical perspective if that makes sense or not, but that's where I see a big issue for us [unclear].

ME: If I can just jump in first—I'm currently working on an article that traces the history of the Barry Commoner's Center for Biology of Natural Systems, which was created in the nineteen sixties as directly engaging in this public information or development of scientific information that was accessible to concerned lay audiences. The idea was to provide expert information in a vernacular manner that could be understood by people without any technical expertise. This expands the number of people who can participate in environmental problems and reduces the authority of science and scientists in the decision-making process, but the science is actually very useful – we don't want to throw it out but we do need to make it useful and accessible for people who might want to use it on a public participatory level. And this is taking place in the nineteen sixties, and constitutes a quantum shift in the history of scientific practice, which, Chris, I guess goes back to your point that a lot of these issues are recurring or continuing . . . .

LN: Well, I just want to say quickly so we can let some others speak, it's a staple of medical history and public health history at this point that the onset of germ theory at the end of nineteenth century really didn't change preventive public health strategies for a lot of diseases. Malaria was a huge concern and even with a disease like typhoid, the importance of germ theory to controlling it is debatable, I would say. With malaria people actually knew what measures to take long before they understood how it was transmitted and, so my point is not that germ theory hasn't created tremendous advances, but that we may need other kinds of models in addition to those from classic germ theory—I mean germ theory's been very good for certain diseases and certainly was critical to the development of antibiotics and all of the benefits that have flowed from that, but in the end we may need to have a few models—I like to think of it sometimes as like the wave versus the particle theory of light. Maybe one model's not enough and maybe we need to expand our thinking to allow more than one.

ME: Scott, do you want to jump in?

SF: Yeah, just a quick comment—about John’s comment, which is that if we needed a new germ theory of the environment that is a theory of the environment that explains much, as much as germ theory does, we also need a national institute of environment. That comes back to the political and institutional questions of why we don’t know—why we don’t have these alternative models now. The political processes that create the conditions for knowledge production have been channeled in certain directions and not others, and one of those not other areas has been environment.

ME: Gregg.

MAUDIENGE: Yeah, Gregg Mitman – I want to respond to comments that Linda and Nancy made about regulatory agencies and scientists...I wonder if we’re at a moment when the kind of regulatory agencies embodies the science that we look to in terms of mobilizing action, you know, the political arena are having [unclear] and that there’s a shift taking place [unclear] and I say that because just a few months ago the CDC announced this new [unclear] initiative in which they’re rethinking the way in which they’re evaluating risk [unclear] in risky places. And yet I just came back from doing a talk at Columbia, where I was talking with folks doing work on [unclear] doing a lot of [unclear] based [unclear] research with West [unclear] Environmental Action Coalition on asthma and environment and they told me that all their work now has the funding for that has been gutted by the National Institutes for Environmental Health Sciences, because they’re only focusing on work on microgenetics and toxogenomics.

So hearing about where the environmental regulatory bodies which you would think would be the ones that would be supportive in terms of the kinds of science [unclear] be done mobilizing that or actually not and more traditional medicine and public health scares are moving into these arenas. At Madison we’ve now become an integrated school of medicine and public health where revamping the med school curriculum, where the vision is that all docs being trained will come out with a public health perspective and at least 60% with an MDH. And so I wonder if we’re at a moment when the kind of critique against medicine and medical approaches is actually no longer being a service to us, and we need to draw those communities in again.

MAUDIENGE: We keep going back to the subject of uncertainty and I think one thing that you see it as a basic ingredient of this is that scientists need a statistical methods and [unclear] starts beginning with the Null Hypothesis, so unless you can prove a statistically significant relationship you end up with no findings, and this is what the industry wanted to use because it’s useful. What if in certain preference towards—you look more scientific if you are cautious against being conclusive rather that cautious against [unclear] and of course [unclear] that being—especially in the early part of the twentieth century [unclear] familiar later—but that being said, there’s no necessity about public health decisions being made just because science is constituted a certain way. That is, if you go through a process and you end up in the position where your conclusion is uncertainty, that doesn’t mean that the proper solution is to do nothing but there are certain incidents in my work as people who know what I do—I work at Tetra Tech and [unclear] decision

of the surgeon general [unclear] is the nineteen twenties was just such an instance so as a result is what comes to a decision rule on how to treat on certainty, they reached a point where they said well we don't have any conclusions and they [unclear] scientific—unscientific they go any further than the evidence allows them and then there's the policy aspect which is [unclear] decision [unclear] if you have uncertainty then the proper thing is to do nothing, so what solutions to a lot of the things we discussed today can come up with [unclear] different sort of decision rules in which reaching a point of uncertainty is not simply a block to any sort of regulatory action. We need [unclear] way of getting from scientific uncertainty to some sort of provisional action that you can take, a no loss type of argument [unclear].

NL: Well, just to say very briefly, that's the debate over precaution of course. At what point do we apply the precautionary principle? What are the cautions with that? Looking at drug regulations since 1938 and then 1975, precaution has been required in drug regulation, although obviously it's been very controversial and complex. But there are certain cautions needed with precaution. I served on an NRC committee recently over the Klamath Basin. Federal agencies—including the Fish and Wildlife Service—were arguing for precaution. But what we were seeing with the models was that their application of precaution might actually hasten extinction—that it wasn't as simple as intuition suggested. I'm sometimes very uneasy hearing people say, "well, let's not work with science, let's just do what we know is true", because often in the communities we've looked at, this intuition is wrong. It was wrong with the fish, and I think we need a more nuanced way of working with communities and science and not just give up on science.

ME: Sure—is there someone—Scott.

SF: Real quick—I think we also need to make a distinction between the kind of science that gets done, where science happens and when science is coming out of industry there's this—I use this quote by Sam Epstein in my book where he says something like "the questions that industry toxicologists pose are narrowly framed, narrowly considered and narrowly answered," and they pose problems in a way that's going to give them the results that if it's not that the results that they get that they don't want but it—won't hurt them. Scientists working in other kinds of settings don't necessarily have those same kinds of constraints placed on them so—

BA: It's so hard to get science done answering the questions that the citizens want answered and, my new case study is in Italy and one of the reasons I chose that as a case study is there's more medical doctors per capita than any country in the world—hordes of them—it's a middle class profession like a school teacher or something [unclear]—they come to your house when you're sick. There's lots of doctors so because they don't have an AMA putting a stranglehold on how many doctors, and it's a bit controlling and it becomes a precious profession with huge salaries there's a whole tradition of the radical medical researcher and they go around and they collect data radical medical research institutes. A lot of really interesting work and some of the interesting work on petrochemical exposure has

actually come out of Italy, because the institution of medicine looks quite different.

ME: Sarah—do you want to—

FAUDIENCE: I'll say a couple [unclear].

ME: —have the last word—yeah.

FA: Just to—maybe on a couple themes that I don't think that we necessarily need the ticket—I don't think you're saying Linda the ticket for just the germ theory framework but I think obviously what happens with how you use that concept is indicative of what you get out of the knowledge, so the concern of course now is that if we can link specific diseases to specific gene types to specific chemical that you've been—this has already happened—but then you say those workers in the third stream aren't allowed to work in a certain area so you have the right to work kind of issue. But that knowledge doesn't necessarily have to lead to that outcome just like germ theory led to like we're saying a lot of great outcomes that have—it's better informed the relationship between the body and the environment. But if it has also led to the kind of the commercialization of caught it but then treat it more like a commercialization of our gene [unclear] is a decision that gets made in the political sphere, as opposed to the knowledge necessarily leads as down this path.

ME: We are out of time—we need to continue this on the tour bus—John has a website he wanted to mention.

JL: I just wanted to mention that we have a website that covers a lot of this stuff—it's called e.hormone.Tulane.edu, and a lot of the papers we talked about are there. One of Tyrone's presentations is there—links to other places are there—other people—so if you want to go to this e.hormone.Tulane.edu that's the website.

ME: Thank you very much for coming—thank you to the panelists....

NL: And we'll see you on the bus at 12:15.

[Tape off]

[End of Session #2]