

MCH EPI Conference

Plenary II –Emerging Environmental Issues in Perinatal and Child Health:

Questions, Quandaries and Quagmires

December 9 – 11, 2009

MARK KLEBANOFF: I want to welcome you all to this morning's plenary session. And we're going to be talking about environmental threats to parents and children's health. I changed that from mothers because many of the environmental threats that people worried about, are worried about also involve male fertility as well as the other reproductive outcomes you're going to hear about.

I also want to thank, although he's -- he's not on the list, it's hard to do anything in this conference over the years without thanking Bill Sappenfield, who helped me -- who along with me co-organized this session and came up with our agenda and invited our speakers. So we certainly owe Bill a debt of gratitude.

Also, with -- by way of introduction note that environment has come up a lot in this meeting, even though it's not something that perhaps many of us in this audience spend a lot of time thinking about or studying. It still was mentioned in Dr. Wilcox's talk yesterday. And even there he noted that he doesn't quite know where it fits or quite know what to do with it in terms of life course events. And it's come up in a couple of other sessions as well. And so clearly this is something

where we're all struggling with and perhaps is fair to say we really don't quite know where it fits or what to make of it.

That's why I titled my brief introduction to this session of more heat than light. And I kind of like watching a good food fight as much as anybody else, although I much prefer it when no one throwing tomatoes at me. But this seems to be an area that really has generated a lot of -- a lot of alarm and often degenerated into some name calling on both sides.

There won't be a lot of talk about Bisphenol A although that's clearly a hot topic but I'll just use this as a brief example. And here is a picture and a report I downloaded off the Web last month from Environment California, and if you'll look at it, that's about probably about a one month old baby and that's probably about one of the cuter babies you'll ever see. And if you look at the title, it implies that we may be poisoning -- we may be poisoning that baby. And stepping aside from the argument that breast feeding might have solved that problem, we still perhaps have a problem. And if you look at -- these are quotes lifted directly from this Environment California report, and they've linked very low doses of BPA to a whole host of things that are clearly things that the average citizen is very worried about, diabetes, cancer, learning problems, things that every parent as well as everyone who's alive today worries about.

The other side is perhaps not above name calling as well. This is a report from a group called STATS which is affiliated with George Mason University, part of the University of Virginia system in Fairfax, and they have perhaps an equally cute baby on their cover, and they have a title entitled science suppressed.

And of course while one side is accusing us of poisoning our children, the other side brings up images of a nefarious conspiracy on the part of a lot of people to foist the myth upon us. I might add if you look at the second quote, I think that Dr. Decant meant frees them of the need to demonstrate the industry's findings are incorrect, but anyway, it's a direct quote. And so clearly there is a lot of name calling going on here.

Where you stand on this issue I would submit to a fair degree depends where you sit, and therefore those of us who are still trying to find their seat find this whole area difficult to sort out. I will say while I kind of point out why people might have their views colored, I'm certainly not accusing anybody of cynically lying to the public and knowingly foisting self-serving untruths. I think life is much more complicated than that.

Certainly if you're looking at industry, any conflicts of interest they might have are obvious. Even if you're a researcher working for industry or even an executive in industry, I think you certainly don't want to believe that your product is causing a

people harm. And undoubtedly in a subconscious way that colors how you view evidence and that colors how you approach the problem.

On the other hand, a cynic would note that whoever makes BPA, and I have to admit I should have looked it up and didn't, but whoever makes it is making money off of it, otherwise they wouldn't continue to make it. And even if you're not a manufacturer, you're just the company that makes baby bottles or tin cans which apparently BPA is a major component of the lining of tin cans and that's a major source for most of us, anyway, even if you're just an end user or end producer, if you had to suddenly change the resin that you make that baby bottle out of, I mean it's going to entail cost and it's going to entail uncertainty. And I would submit that in spite of all the blather that we hear about free enterprise that most business is fundamentally pretty risk averse and would not want to change something that works unless they absolutely needed to.

I would also point out that many of the critics will hide their conflicts. And this is a quote from a source watch that I assessed last month. It goes back to that science suppressed report. That website STATS, which is affiliated with Mason, does not list their funding sources in 2008. In fact, they said that we do not take any money from industry or industry related groups, and in fact as of third of November they still said that. However, there are organizations out there that make it a point to chase things down and as you will follow the money. And they did find out that STATS, even though they presumably are truthful in telling you

they don't take money from industry or industry-related groups, they do take money from the Olin Foundation and the Sarah Scaife, I'm not sure how to pronounce that, Foundation.

For those of you who aren't really up on all of this stuff, Sarah Scaife Foundation is run by Richard Mellon Scaife, who runs a lot of the newspapers in Pittsburgh and was the guy who got the drumbeat of investigation going against the Clintons over Whitewater and basically didn't stop beating the drum until January 20th, 2001. And so even though perhaps STATS is being truthful in saying they don't take industry money, they're not saying that the money they take comes agenda free. And you know, while I'm going to try to a certain degree hide my views, I will go as far as saying that's probably deliberately misleading.

Any way, let's not assume that academic are as pure as the driven snow although I'll repeat for the third time that most of our conflicts are undoubtedly subconscious. You know, people see what they are trained to see, and so that's why I always make fun of people in Homeland Security. They are trained to see terrorists and they will see them even when they aren't there.

Well, those of us in epidemiology are trained to see health threats, and maybe we see health threats that may be exaggerated or may not be there at all. After all, nobody wants to submit that they've spent the last 10 or 15 years researching a trivial problem. And undoubtedly that impacts how we approach things. And I

find many of us in epidemiology, and I'll be impolitic enough to say I see this in my environmental colleagues are susceptible to the Chicken Little syndrome, and it probably reflects their desire to be studying something important.

On the other hand, just as I was cynical with industry, I can be cynical with my researcher colleagues and point out that if we had a totally negative study, we're not going to be in much position to get a follow-on grant proposal funding funded. You could say to your funding agency this time I'll get it right, I really promises and that's not going to carry a lot of weight. So clearly by getting positive findings our stressing the positive in our studies it puts us in better position to submit that follow-on grant proposal to submit our shortcomings or to correct our shortcomings.

Similarly at the collective level when enough positive results show up the public becomes concerned, and ultimately public concern will lead to concern on the part of congress and perhaps that will -- that will end up in increased research funding on the topic. We've certainly seen that for HIV, we've seen that for breast cancer as well. And that's a well tried model at this point.

With that as background, Bill and I discussed what we'd like to present, and we've chosen our speakers today for their ability to step back and look at the whole picture. They're not going to be dismissive of imperfect evidence, they're

not going to take the tact that the tobacco industry used for 40 years and let the perfect be the mortal enemy the good.

On the other hand, our speakers are not given to push the panic button when the evidence is perhaps inconclusive at best and maybe even flimsy at worst.

Our first speaker will be Pauline Mendola, who has had two major careers. Pauline is currently chief of the Infant Child and Women's Health Statistics Branch of the NCHS, and before that she was at the EPA for 10 years and was risen to be branch chief of the epidemiology branch. So as you can imagine, Pauline's background is very strong in both environmental and reproductive perinatal epidemiology.

Pauline will discuss what the concerns are, some of the strengths and what's -- where is the evidence strong, where is it weak and kind of discuss principles of when we should or shouldn't act on emerging concerns.

Our second speaker will be George Rhoads, who is currently professor of epidemiology and associate dean for academic affairs at the School of Public Health at the University of Medicine in New Jersey. George has had three careers and counting. George started in cardiovascular epidemiology. And then when I started at NICHD, George was my branch chief, and so as -- had a very

outstanding career as perinatal epidemiologist and then moved on to become an outstanding environmental epidemiologist as well.

And George will review the limitations of observational research in general and of this troubling environmental research in particular and give a few thoughts for how we perhaps might use a little bit unusual designs to try to overcome them.

And then there's the third speaker will be Kevin Sherin who has also had two careers. Kevin started out as a family -- in academic family practice and focused a lot on antenatal care and then has been a local health officer in three jurisdictions and two states, is currently director of the Orange County Health Department and president of the American Association of Public Health Physicians.

And Kevin, as you can imagine, doesn't have the luxury of standing on the fence and saying gee, I don't know, we just have to do more research. And he has to -- he has a public he's responsible to and has to be able to try to address questions that may not have very good answers or answers at all.

With that as background, I'd like to introduce our first speaker, Pauline Mendola.

[Applause]

PAULINE MENDOLA: Thanks, Mark. It's a pleasure to be here. I've never been to this meeting before, and I'm enjoying it. And I hope that you enjoy this talk.

I'm going to try to give a sort of an overview of some of the issues that are apparent in environmental epidemiology where it transects perinatal reproductive epidemiology and some of the concepts I guess that we have to struggle with when we're trying to decide where the risks are and what should we do.

One of the things that is really a challenge, and I think Mark gave a good introduction to this, is that it's really difficult to separate what gets public attention, what is the hype, if you will, as opposed to things that are real risks. And that's a challenge because at the best of science there are good disagreements among people about the strength of the evidence. But often what gets into the public opinion and in the press is some slice of what the data really say.

So why is this difficult? I mean, if we take a sort of a benign approach to it, then this essential challenge if we assume everyone's trying to do the right thing, is that it's a conflict of competing social values. And at some extent they're all good values, you know, that's sort of the I guess the most benign explanation. But they can't all coexist at the same time. So the disagreements come about when we think about what we're trying to balance in terms of the good.

So we -- you know, we all want healthy mothers and children. That's a really good thing. In this group, I'm sure that would get a good endorsement. But we also want clean water. Well, clean water requires disinfecting water which results in more chemicals in your water.

Affordable fruits and vegetables, we think that's good. We think people should eat a lot of fruits and vegetables, but you know, that may require the use of pesticides.

We want sustainable, cost effective energy, we want low cost goods, all these things. But they require some trade-off in terms of chemicals that end up in the environment and other things that might not be good for us in terms of exposures.

And when it comes to making decisions we also want a certain amount of autonomy and that exists in a free market. So you can see already we have a lot of competing things here.

The other thing that really plays a lot in this field is the area of risk perception. And this is a very small introduction. It's a huge field that tries to analyze how people perceive risk. One of the things -- and so I'm giving you a sort of a short not very sophisticated look at it. But one of the things that's very clear is that people are much more comfortable with risks that they choose. So they can

choose to smoke or to drive drunk. They think that they will still be fine. But they're much less comfortable with risks outside of their control.

So you know that they're worried about living near a contaminated waste site, they're worried about nuclear power plants, they're worried about pollution because they are perceived that things that are outside of my control and they worry me.

And sometimes you have situations where public opinion and risk perception drive policy. And you see things like Mark mentioned, the Bisphenol A and plastic bottles. The evidence Bisphenol A as a human toxicant is pretty weak. You know, there really isn't very much evidence. There's a boatload of animal evidence. But if you heard Dr. Wilcox's talk yesterday, he mentioned that there are a lot of compounds that show risk in animal species but then when we look at human studies it's not there. Maybe we're not looking at the right things or we're not looking as closely as we like.

But clearly that's a case where you know now you see BPA free stamped on all these plastics. The organizations and the industries have responded to public concern that's probably a little bit ahead of the science.

So why are people worried? There are a lot of chemicals in the environment. And it's -- there are more all the time. In the past 60 to 70 years, chemical production

in the United States has increased 20 fold. The number of chemicals registered for commercial use has gone up more than 30 percent in the last 20 years and more than 80 percent of those are not tested for human health effects.

So people have, you know, some legitimate basis for concern. Biomonitoring studies will routinely show that people are exposed to these things. So you can in fact find hundreds of chemicals in people's blood. It's in your body, it's in your kids' bodies. But you know, we don't know how harmful they are.

Most of these things are probably not good for you. You know, if we start from the sort of benchmark that exposure to some of these contaminants is not a good thing but is it really a bad thing? So people a lot smarter than me have been thinking about this for a long time. And one of the things that has come to be sort of a benchmark in this field is the precautionary principle. And the precautionary principle essentially states that if there's a threat of harm to human health or the environment, then we ought to try to do something in a precautionary way, even if the cause and effect is not really fully established, and the people who want to do the thing that's risky ought to bear the burden of proof. So the public shouldn't have to say protect me if they think that needs to happen. So you know, what does that really mean? It's complicated, but it's essentially what it implies is there's a responsibility to intervene. We should try to protect the public when we have some scientific evidence of a plausible risk. And that those protections

should stay in place until in fact those scientific findings are refuted where we have some more robust evidence to suggest that their risk is not there.

In the European Union if people are familiar with any of their environmental health systems, the precautionary system is also a compulsory principle of law. So in terms of the chemicals in our environment in the United States, there are a lot of things that we use commercially that are regulated or banned in Europe because they feel that there's too much of a threat of risk.

So where and when do we protect? So most people would agree with we should have protection from hazard. But there are a lot of disagreements over who should protect and who -- and who should regulate it. Should it be the Environmental Protection Agency, is it be state, should it be up to individuals? And this is one those things that you know you know when you see people complain that the government is not protecting them from something that they've observed to be a risk in their communities or, you know, we ought to be to go something about this risk that on the other hand they turn around and say well, we don't really want the government involved in our everyday life.

So for those of you who work for the federal government you may sometimes feel the push-pull, but it's certainly there. I mean people want to be protected from things like contaminants in baby bottles, they're worried about it, but they also don't want you to overinterfere. And sometimes the evidence is not really clear.

So how to determine a hazard is a real issue. What is the strength of the evidence, and what kind of evidence do you have? You often only have evidence that's imperfect.

Autopsy who pays the cost of the protection? Does it come from the taxpayers, does it come from industry, which then gets passed on to consumers? All of these protections have some cost. And then the question becomes is the cost worth the benefit?

So one of the things that we really need to struggle with is what is the nature of the risk? A lot of the studies in environmental effects and reproductivity and perinatal health, they are often small groups with unique exposures. We see a lot in this literature of an occupational studies and occupational studies certainly provide sentinels where you have people with well-known exposures who are exposed for more intense times and durations than the general population, but also there's a lot of stuff in the literature. You know, 15 women with some outcome compared to another 15 women without this outcome, and it's just odd little sets of data.

But why are they in the literature? Because there's a lot of negative study that are still sitting in people's file cabinets because they can't get them published. So I

think that's another important thing to remember and sort of builds on what Mark was saying earlier.

We also have to think well, how severe are the outcomes in sometimes the outcomes are very serious. They can be death the and permanent disability. Other times they're less serious but common, things like asthma, where, you know, the death rate may be relatively low but it's an incredible public health burden of disease.

Thinking about the likelihood of exposure. Often the most -- a lot of the literature folks on high dose risk association. So the animal literature is almost all high dose. A lot of the occupational literature is relatively high dose. So we don't really know at the levels that are commonly encountered by people, it's often hard to see whether or not the risks are there. And there may very well be subgroups in the population that have specific risks but we have a hard time identifying them. And again this really follows well on what Allen Wilcox was saying yesterday.

And then finally if it's easy to ameliorate the risk, then that's another thing that plays into the decision. We could remove BPA relatively easily from tin cans and baby bottles, and so now a lot of people are doing that. So, you know, that happens because it's easy enough to do.

In terms of formal deciding, how do we decide? There actually is a real process that's used in human health risk assessment which has four steps. You do fairly well with the first step, which is hazard identification. You see a lot of papers in the literature that are more on this, we think this might be worrisome, people need to look at it. So a lot of things to look at. You can go and say, there some potential for harm.

Assessing a dose response's relationship is much more complicated. It's hard, especially in human studies, to be able to see an effectively range of dose, and also to get a good sense of the relationship between exposure and effects.

Exposure assessment, risk characterization. Again, there are studies out there, but they're complicated and there are not that many, especially when you get to real risk characterization where people are deciding this is what the body of evidence suggests.

So what else do we need to think about, as if that is not enough? Certainly variability by maternal and fetal genotype. The dose and timing of exposure. We've been talking a lot about life course in this meeting. Very important. Very environment contaminants, another angle on this, think about intergenerational effects. Some of these exposures are persistent. So we all have some DDT and DDE in our bodies. And we haven't been exposed to that routinely in this environment since the early 1970s. But it will probably take three, four or some people estimate five generations before those levels are below some limit of

detection in Americans because it continues to be persistent in the environment. It's passed from mothers to children, and it continues to be there. So you have those intergenerational effects as well as the developmental impact of exposures at different times in life.

The other thing in this arena we often see variations from typical dose response curves. So low dose responses may activate different enzyme systems than high dose responses. Often the scenarios of exposure for environmental risk are more risky for vulnerable populations. People are probably familiar with the concept of environmental justice. Often people who are poor live in areas that are polluted. They don't have a lot of choice to be able to move to other places. They may eat food that's not as well certainly less organic food. They may eat food that isn't good for them. Certainly subsistence fishermen that are fishing in lakes that are polluted, but that's the only source of protein that they have. They don't often have the choices.

And then the other issue I mentioned a little bit earlier, the risk of exposure. Don't always outweigh the benefits in some places. And there's a huge debate in the literature about DDT as I mentioned as a persistent chemical that people are really trying to get removed from the globe, but in fact it's very effective for malaria control because it kills mosquitoes and the fact that it's persistent, it kills mosquitoes for the whole year. So, you know, we have hundreds of thousands of children dying of malaria every year.

So we may have -- in fact, there was a paper that just came out recently looking at increased urogenital anomalies in kids exposed to DD -- when their mothers are exposed to DDT in malaria areas. So you know there's a little blip uptake in some birth defect, and there certainly may be other health effects, but again, malaria is another thing that really kills a lot of kids.

So what do we think about? We have a lot of outcomes that have been studied. But generally the things that are most looked at in terms of maternal health, things like fertility, pregnancy loss and complications and child health, a lot of interest in asthma and autism and obesity and preterm birth. And there are other outcomes certainly that have been evaluated.

So just to give you sort of a feel for where some of this evidence is. You can see that there's certainly a big literature with respect to birth outcomes. Most of it is sort of equivocal. There are very few things that have strong evidence. Most of the things have some suggestive studies although they're not consistent for all end points.

And then some things that are relatively weak. For example, dioxin. Dioxin is a good example because it's a compound of tremendous public health concern and in animal studies has a lot of very bad effects. In human studies it's not been as harmful.

In terms of child health, there are more things that are strongly associated with poor child health outcomes. Most of these neurodevelopmental outcomes, asthma, cancer. And again, some things with moderate and weak effects.

Fertility just is one example. There's quite a bit of evidence for lead, and some for pesticides actually for people who work in applying and mixing pesticides and then some moderate associations with other persistent organics and solvents. Again, the associations with BPA are pretty weak in humans.

So what do you do about this, trying to avoid exposure where you can. When possible looking at purchasing organic produce. There have been some studies that have looked at biomonitoring data in a family eating a conventional diet and then they switched to organic food for, you know, two or three week period, they biomonitor them and then they switch back and you can clearly see that the levels are lower. A lot of the organic phosphates that are in your food now will be things that will clear pretty quickly.

If you're exposed to some contaminants, you know, try to use personal protective equipment. Integrate a pest management. Generally being aware of things. If you have lead in your home, if you're going to remodel, you know, paying attention to those sort of factors that can mitigate any risks that you have.

And generally following instructions. So if you get a pesticide or some other thing, follow the instructions. But I will say that it doesn't always work. And those of you who saw this tragic case that happened in South Carolina in November know this was a case where there was a young mother with a toddler and an infant who used infant foggers to -- who used pesticide foggers to try to eliminate cockroaches in her home, and her trailer home and she just kept doing it over and over again, even though she followed the instructions, she took the kids out of the house for four hours and then came back, the fact that she used the same product seven or eight times resulted in a buildup of pesticide that caused the death of an infant.

So how do we know when to act? We're much more likely to act when the evidence is strong. And in general the threshold for maternal child health is really lower than for other populations. We want to protect mothers and infants. And I think the population is more willing to accept restrictions when it's about the protection of this vulnerable subgroup.

Also, we're more likely to act when the outcomes are common or severe. We can identify the exposures. This was another important point that was raised yesterday. We know that this stuff is in our bodies. We're not always sure where it's coming from. We can't always tell there's a direct route that we can act on. And if there's the solution or remediation available, especially if it's affordable, then we're much more likely to act.

But it is more I think to remember that there's a balance in all of these things and that if we're trying to act in a way that protects the public health, we have to think about the fact that it could have a negative effect. Right? So we do have people who are especially at the fringes of being able to protect themselves. For example those subsistence Michigan who are fishing in Lake Ontario and getting tons of environmental contaminants from those fish, but they have a male to feed their families? And what are they going to feed their families if they don't get to did that? So we have to think about that there are both sides to the story.

And also I like this because we don't always have a choice when we're trying to protect ourselves. So thank you.

[Applause]

MARK KLEBANOFF: Thank you, Pauline. That was very good, very enlightening in presenting what things that we all ought to be thinking about.

Our next speaker will be George Rhoads who will critically review what the evidence is or the process of gathering evidence is and give his thoughts for how we might do better.

GEORGE RHOADS: Thank you very much, Mark. Thanks to Bill Sappenfield, too, for inviting me to come and give you my opinions. I've gotten to be such a

curmudgeon in my old age that they make me put up this thing that says no one else has to agree with anything I have to say [Laughter] so that certainly goes for you in the audience as well because I will present a somewhat contrarian point of view in the next few minutes.

Evidence based public health holds that we should usually inefficient interventions unless we have solid -- that we should not usually inefficient interventions unless we have solid evidence that the intervention is going to have a net public health benefit.

This implies that the completion of good quality studies that show unequivocal benefits and at least some assessments of any harms that might be caused moreover practicing evidence based public health often requires public health professionals to exercise considerable leadership to try to prepare the community and the legislature that -- and other opinion leaders in the -- that they really should be waiting for evidence and not just going off in all sorts of different directions. And I know that that's a difficult area that may be Dr. Sherin will talk about a little later.

So I think evidence based public health is more difficult than for instance evidence based medicine where it's really largely between the doctor and his patient, although obviously there are other things that weigh in on that.

Some of the challenges that are encountered in environmental epidemiology, one thing that sets environmental concerns apart from most of the rest of our epidemiologic investigations is that often you have a substance and you're looking for a disease rather than having a disease and trying to find what causes it. So you're kind of coming at it from the other side, at least to some extent. And that raises questions that are more numerous than are raised in many other areas of epidemiology.

Nearly all of the observational studies that we do of course are subject to confounding. That's one of the things we teach our students right away. And almost all of the epidemiology done on environmental issues is observational evidence.

Exposure measurements are often poor. Relative risks of interest are usually small. Sometimes it's hard to distinguish a priori hypotheses from exploratory or data dredging if you want to use a more pejorative term from exploratory studies.

And then I think another problem we have is that when these studies get published, often aimed at really trying to communicate findings among specialists in the field, they get picked up by the press and much more is made of them than is really appropriate.

Just to give you an example, our group did a paper on air pollution and small for gestational age earlier this year. It's not an easy topic to address. We took births in New Jersey that were occurred to addresses of moms who lived within 10 kilometers of an air monitoring station. We measured four pollutants and we looked at them in each of the three trimesters of pregnancy, so we had 12 different exposures, if you will. And when we looked at the relative risks, they were actually very modest. Only did we find anything for only two of the pollutants, nitrogen dioxide and particulate matter less than 2.5 whatever the millimicrons -- I've forgotten the unit. And those relative risks were only 1.1, just 10 percent across the interquartile range, which is from the 25th to the 75th percentile.

So when we published this paper, we had three-quarters of a page in the discussion pointing out these limitations. But nevertheless the paper got picked up by the press, and we actually got I think more press coverage than was appropriate for a paper of that sort.

So how might we strengthen the evidence that we're getting on environmental risks? I'm going to talk mainly about three strategies that I think deserve a little more attention than they've been getting. One is that with we do very few randomized prevention trials. You know, when the FDA approves drugs, they don't approve drugs on environmental -- on observational evidence. They really require experimental evidence.

If you're interested in whether BPA and baby bottles causes trouble, it probably would be feasible actually to take women who are going to bottle feed their babies and divide them into two groups and give one group bottles that are free of BPA and let the others do whatever they normally would do. I'm not saying it would be an easy trial to do, but there certainly could be considered to do something like that if this was considered to be a serious question.

We also could use more in the way of quasi experimental studies. And I'll talk about that a little more -- I'll give you an example in a few minutes. And as sort of as part of that and related to that, I think we don't pay as much attention to secular trends in exposures and in diseases as we should. If an exposure causes a disease and the exposure is changing rapidly in our population, then you know, you would expect to see some impact on whatever the health outcome is that you're looking for, and we often kind of ignore that issue.

I think also we should try to employ more variation in study design to better control confounding in the design phase of our studies rather than depending heavily on multi-variant analysis, which is -- you know, the statisticians have made it better and better, but it can't be any better than the measures of the covariance that we put into the model. And that often is quite limited in some of these studies.

So for an example, I wanted to talk about the current controversy about lowering the level of concern for blood lead in children, which as most of you know is 10 micrograms per deciliter and has been set there by CDC since 1992. There have been in the last six years or so a series of studies that have suggested that lead effects on IQ go down to levels of lead that are below that. And not only are the level -- the effects found at those lower levels, but the strength of the association actually appears stronger at the lower levels of lead than it was at the higher levels that we used to be more concerned about.

So I wanted to spend a little time examining this evidence. And of course as I said before, these studies are like all observational studies, are subject to possible confounding. And we're particularly concerned about confounding by socioeconomic status, which confounds many environmental exposures because we all know that we have environmental justice problems in the United States and people of lower socioeconomic group are exposed to more stuff, if you will, than those of us who live in ritzier homes.

So the studies relating blood lead to children could be biased by those kind of differences. And the studies of course have done -- have really gone to great lengths to statistically adjust for the kinds of differences that we're concerned about. But if you think about the home survey, h-o-m-e, which is used for instance to try to assess the quality of the home and adjust for any differences in quality of parental care and so forth between high lead kids and low lead kids, it's

basically an hour assessment in the home. And I submit to you that it's really not possible to completely capture the quality of parenting by visiting the home for an hour.

All right. Well, here is a scatter plot of one of the key papers that got this area going published by Canfield, et al, in the New England Journal about four years ago, maybe five.

And as you can see, these data are from Rochester. There were earlier studies from Rochester that had leads mainly between 10 and 20, but these children the leads are mostly under 10. And when they put a regression line through these data allowing for some changes in slope, the slope appeared substantially steeper in the lower level of the curve, as you can see.

So if you actually look at those coefficients, when they calculate the coefficient by forcing the line to be straight, they're putting it through the whole set of data, you get a coefficient of .35 IQ points per microgram per deciliter of blood lead for the lifetime mean and a very similar value if you use the concurrent blood lead at age three.

If you use only the children whose leads had never exceeded 10 micrograms per deciliter then the coefficient is four times higher no matter which of those two

measures you use and it's indeed a very high coefficient of more than an IQ point per microgram per deciliter.

Now, if you think about what actually has happened to blood lead in the United States, in NHANES 1, which was the late '70s, the average blood lead in the US for children ages one to five was 15 micrograms per deciliter. In 1990, it was 3.6 micrograms per deciliter and today it's under two micrograms per deciliter. So we have removed 85 or 90 percent of the lead from the environments of these children.

And you might think that if these effects -- if there were large IQ effects of lead at these -- across this range that you might see some changes. So applying the coefficients that the Canfield group published though those changes in blood lead, you would expect a modest decline going from 15 down to 10 and then a very sharp decline as we went from 10 down to 3.6, adding up to about 10 IQ points.

And this is -- these data, of course, are nationally representative samples, so the implication is that the entire US population of children's IQs should have jumped 10 IQ points if these studies are correct.

Now, 10 IQ point increase in the mean of IQ would mean that we would expect about a five-fold increase with people of IQs of over 140, and as someone who

teaches lots of graduate students, I am looking for all those new candidates, and I haven't seen them yet. [Laughter].

So here's another set of data. This was a cross-sectional analysis that came out of NHANES 3 where they looked at about 5,000 children between the ages of 6 and 16, selected venous blood lead, got school achievement records, and carried out some psychological assessments at the interview. And the particular thing I wanted to show you are the results of the wide range achievement test which has a reading subtest and an arithmetic subset. And like the data from Rochester what you see is that the coefficients are -- if you just look at all the children and -- where the N is 4853, you got a coefficient of about one rat point per one microgram per deciliter IQ. And it goes up by about 50 percent if you exclude the 300 children and there were only about 300 who had blood leads above 7.5 micrograms per deciliter. So, you know, by this time the vast majority of kids had their leads way under 10. In fact, I guess it was 3.6. I think that's the source of the data I showed you before. Although these are older children than that.

So doing the same exercise, you get an estimate that the reading scores should have improved by about 13.4 points. The standard deviation is 15. So this is nearly a full standard deviation improvement in reading. And you know, you think with that combined with No Child Left Behind that our reading scores would really have zoomed. But unfortunately that actually is not the case.

So here actually are the secular trends in reading scores. These are compiled by the department of education. They run a survey of school children in the United States that is especially designed to track longitudinal change. The top line is the white children, and the lower line are the black children. You can see that as we integrated our schools that there were some improvements up to about 1980 for African-American children which was very heartening.

But I have put two little yellow triangles on here that indicate the times that the children who were exposed to these high and low blood levels were actually being evaluated. So that the top one in the middle is the -- those the time when children who grew up with blood lead levels of about 15 when they were toddlers were taking the test and the one in the lower right shows the time when the kids whose blood lead levels were about 3.6 when they were toddlers were taking the test.

And you can see that if you put a -- imagine a line between those two yellow triangles, you can see that there was a huge decrease in blood lead cross this timeframe, but there's really no response that's visible here in reading scores.

If you look at sort of a broader age range, you see basically the same thing, that children of any age there was really no change except from 1999 to 2004, there was a small increase in kids age 9. All right. Well, we talked a little bit about confounding. I want also just to point out that with lead and IQ and development

there is some possibility of reverse causality. Children with less intellectual potential might have more hand to mouth activity or might persist for longer because we know their development is often slower. And it's well known that kids who actually are mentally retarded are at special risk for lead poisoning.

It would also be possible that parents who minimize lead exposure in their homes might also be parents who read to their children and do other things that would give them a little more intellectual stimulation. So that's another possible reason to think that these studies could -- that the studies could be confounded. And clearly the possibility that the studies of these low level lead things are basically exaggerating the effects is the simplest explanation for why we're not seeing any effect as lead has come down across the nation.

So let's do a thought experiment. Suppose for a moment that we posit an association between slow development and ingestion of exogenous substances. Or if you prefer between poor parenting and the opportunity to ingest contaminated substances. And then let's suppose that we had two groups of children, one with more ingestion and lower IQ potential and the other with less ingestion and higher IQ potential. And let's just for the sake of argument say that the IQ difference between these two groups is three points.

Then in a high lead environment, children with low potential or poor parenting will have subjectively higher blood leads than other children because the extra

ingestion will include a lot of lead. So let's just say it was nine micrograms per deciliter. In the low lead environments, the blood lead difference between the same two groups of children will be slight. Let's say three micrograms per deciliter because the stuff they're ingesting doesn't have as much lead in it.

Then in a high lead environment, we'll have three IQ points associated with a blood lead difference of nine micrograms per deciliter, which is a third of an IQ point for each microgram per deciliter, a rather shallow slope. But in the low lead environment you'll have a full IQ point for each microgram per deciliter, so you're going to get a steep slope.

These numbers are obviously arbitrary, but I think they illustrate the point that as we move kids from high lead environments that used to typify our homes to the lower lead environments that we have today that you would actually expect that with a little confounding that this relationship would steepen. And whereas this steepening is completely predictable from positing confounding it was really obviously a complete surprise to investigators and was not considered at all when they set the levels at 10 micrograms per deciliter.

So just to summarize that, we have in a sense we've real had a nationwide quasi experiment that has shown no evidence of effects predicted by recent observational -- predicted by these recent observational studies for blood lead less than 10 micrograms per deciliter. The observational studies relating blood

lead to environmental outcomes are likely to reflect at least some confounding or reverse causality as the most obvious explanation for this discrepancy.

For these reasons and for several others, I think it -- we probably should not lower the level of concern below 10 micrograms per deciliter until we have better evidence that it actually is going to make a difference. It's going to be a lot of work to start going after a lot more kids and if we're not clear that these really are causal effects down there and in addition to that there are a variety of additional problems such as whether we can really measure leads with our current lab techniques and so forth and so on, it may not be worth going after.

And then finally I think that we should try to look at new observational designs to look at these low level lead effects because the old designs simply relate blood lead to IQ are not going to be able to resolve the kind of confounding we're talking about. So where do we need to go? I think more randomized prevention trials, more quasi experimental designs would be helpful. I think examining the consistency of the conclusions of studies with secular changes in risk factors and disease can help some.

I think we can improve our exposure measurements and tighten up other features of our studies. We need to make clear distinctions between a priori hypotheses and where we're really testing a single hypothesis versus really hypothesis generating studies which I referred to before.

And with those kinds of changes and I think also redesigning our studies a little bit so that we try to take care of confounding more in the design of our studies rather than relying quite so much on multi variant analysis would also be helpful.

Thanks very much.

[Applause]

MARK KLEBANOFF: Thank you. And we'll have time we expect we'll have time at the end for questions, as I'm sure that so far you've heard a lot of things that you would like to ask questions about and may have opinions about. As George said, how do react and exercise leadership on these difficult topics that arouse public concern and may not have clear evidence one way or the other is a very, very frustrating challenge to people who are on the front lines.

And so our third speaker will be Ken Sherin who will talk about how you -- how he approaches this and what some of the problems are and how best to engage the community.

KEVIN SHERIN: Thank you, Mark. I want to thank Mark, and I want to thank Dr. Sappenfield for including this presentation. And I want to give you the flavor of the local health officer who has to deal with the community.

And the title is obviously balancing perceptions with evidence. What did I do? There we go. Now, you have to have a picture of this community. This is the Lake Jewel community in South Apopka, Florida. This is a generally a very poor community. And that mound behind that home is a landfill. So as you might imagine, the environmental health concerns for this community center around that landfill. But there's more. This community also is adjacent to Lake Apopka where for many years there were farm workers who were exposed to pesticides. And there is more. You have studies from the University of Florida on the alligators showing urogenital changes. And there is more. So this is a very heavily studied community. And when I came in to this role a little over five years ago to be the local health officer, I didn't deal with this particular neighborhood but another part of the South Apopka community where there was an open meeting and ACORN and a number of other organizations were there. The farm workers, the newspapers. What are you going to do about this? And just a complete lack of trust for the local health department.

So if anything I can convey to you is this is iterative process, it is a communication process, and it is that risk communication that we talk about in the field of environmental health. And if you've never had a chance to have training on risk communication, I highly recommend it, and I highly recommend training under Vince Covello, in particular.

So we have a poor community, we have environmental justice and social justice that are some of the root issues going on here that color the perceptions. We have the history of the many studies that have been done over the years. We have the drinking water issues where for many years these communities didn't have community water systems. They had wells. And of course this was farm land. This was what we call in Florida muck. Muck is adjacent to these large lakes. And it's very rich, fertile soil. So it heavily farmed and heavily covered with pesticides for many, many years.

And here is another shot with the landfill. And you know what landfills look like. So you don't need to see all this. There's also another industrial source here for air pollutants. Of course there are methane gasses that come off of this landfill. And sulfite smells and what have you. And so you can see why the people are here concerned about what effect is this having on my community.

Then there was a local city commissioner whose granddaughter had an infant death, and all kinds of alarm bells went off. Oh, this was maybe the end of 2008. And so we began to get more voices in this neighborhood, this subunit of South Apopka adjacent to the landfill saying you've got to do something.

So this is Orange County, Florida. Actually the population now is higher than this. It continues to grow despite the economy. It's a high immigration community. Not high economic development community. We have a service economy. You might

have heard of it. It's called the mouse. Lots of hotels, lots of low wage workers, lots of people working in theme parks and so on and so forth. And so our social demographics and our social determinants of health are not the best. And you've heard that in the preceding talks. You know, you heard Pauline touch on some of those aspects.

And here are our statistics. I believe personally they are awful. We need to do more. But, you know, this is a work in progress, and it takes many years to begin to reverse some of these trends. So when I see an infant mortality rate in 2008 that's 9.1 for this county, I know there's a lot of work to be done. But that's the sort of backdrop that the Lake Jewel community plays against. You have to look at the community data as a whole. You can't look at the microcosm of Lake Jewel or the larger microcosm of Apopka or the larger microcosm of Orange County, Florida, you have to look at each one in context.

So we have bad infant mortality rates that are higher than the state of Florida as a whole. And Florida in some respects may be behind other areas of the country in infant mortality of course.

And fetal infant -- fetal death rates, which are higher than the state of Florida as well. And as you might expect, our low birth rates are somewhat worse. And we have more pre-term births. We have more teen pregnancies than you might expect for a population as a whole in the state or elsewhere. And that is the

nature of the social determinants of health playing themselves out with social justice probably being the primary determinant.

Prior studies on this community included the lake -- the Keen Road landfill, which is the one that you're looking at, and ATSDR actually came in here and did a consultation. And the purpose was to evaluate the health threats posed by the Keen Road landfill to this community. And the study was conducted between 2001 and 2006. And the conclusions as you might expect our measurements were not able to show that there were significant issues, however, the city was hooked up to city water and all but three of the near by residences now are on city water. So current exposures for anything with related to wells or drinking water clearly has been mitigated.

Then there was insufficient evidence of a public health threat in these wells. And they continue to do monitoring of these wells. And once in a while you get a hit of, you know, a very low level of lead or a very low level of arsenic or what have you, and it's not enough to raise alarm bells. And the Florida Department of Environmental Protection continues to monitor this in conjunction with the health department.

Well, here's some of that history of Lake Apopka. And you remember the stories of poor folks. Fish. Because that's how they can eat. So they continue to fish in this lake. It's a huge body of water. Maybe one of the larger lakes in Florida. It

crosses over into two counties. And it falls under this water management district. And the study fell under the Florida Department of Agriculture. And it took place between 2004 and 2008 and looked at the fish. And of course we look at the mercury, and of course we look at all the different environmental contaminants that can possibly happen.

And there were some six species of fish which did prompt the Florida Department of Health to issue fish consumption warnings which were then communicated to the community. Subsequent testing determined that annual decrease in pesticide levels in the fish with only one fish remaining on the do not eat list, which is the brown bullhead catfish for those fishermen among you -- I wish I had a picture of the brown bullhead catfish, but I don't. And these were additional studies now with the farm workers project, and Dr. Harman with the University of Florida and Rollins College, which is one of our community's universities. And this in summary is the community that was extensively studied and continues to want action to address real and perceived concerns. And I must say there were even national people who come into this community to make sure there are continued studies.

Well, then, the local health department does our work as the local health department, and of course we work with Florida Department of Health and the CDC, and we have a pay CH project which includes the South Apopka community, since this is our version of the most studied community in our

jurisdiction. And we did 276 health surveys, and we found what, you know, the issues were for these folks and what they wanted. And these were generated from a community meeting.

They wanted more medical and dental clinics. They wanted more medical specialty care. Among other things they felt they had more lupus in their community. Actually there's a higher incidence of lupus in the African-American population. I can tell that you as a family doc. Physician activities and -- physical activities and after school programs which go with many of the communities throughout the United States. More organized sports with low or no fees. And they wanted that stinky dump to be cleaned up so it didn't smell bad. The odor from the Lake Jewel landfill.

The low income community is predominantly African-American, and the closest point of the neighborhood to the landfill is 0.5 miles. In 2000, approximately 2600 people lived within a one mile location of the landfill. 55 percent were black, 40 percent were white, and five percent were other. Community health concerns included the following: And you can read the list. These are not atypical community health concerns from a lot of places, except perhaps when you look at a lupus. But, as I said, lupus is more frequent in the African-American population.

And then when it comes to maternal child health issues we know the drill, we know what to expect, and surely they were interested in infant mortality, they were interested in the still births, fetal deaths, the prematurity and the low birth weight. Now, when we looked at observe versus expected for this in similar communities, indeed you know the observed is high for infant mortality in the Lake Jewel area. But this zip code really extends beyond Lake Jewel. It's the South Apopka area. And yes, it does have a landfill, and yes, it does have nine super act sites, but when you take it against Orange County as a whole, guess what in our infant death rate is about 8.5 for these three years. So you have to look at that and take it in context.

And we just have really bad rates in our county. We have really bad social determinants. And we take that into context. And then we looked at several other comparison communities and you see the numbers there. And so we surely have our supply of super act sites, don't we? Comparable zip codes were picked that had at least twice the number of super act sites as the 2703 zip code.

So how do we go about doing this risk communication? You really have to do it multiple times as a family doc you see the patient back in your office. In public health you have multiple community meetings where you communicate with the community and you hear what their concerns are.

So here's an example of one of those on January 14th. And the attendees included the Lake Jewel community members, the operations manager of the Keen Road landfill, which is under Waste Management, and the health department. And a presentation was given detailing actions to be taken by the health department followed by a question-and-answer session. And not to say that we put it all on ourselves. We work with DEP, we work with the county government, we work with the Waste Management people. You know, we work with the city of Apopka. There's a whole community effort to do as many things as possible to address their concerns.

And the Apopka Farm Workers Association was there. And they were also working with the Lake Jewel community. So here's what you say. Here's how you do this. You listen to the community. And we talk about community based participatory research. The new nomenclature is community driven research. When I go to those sessions at the APHA, I'm hearing this again and again, and I see it with NCS and other things coming along. We want the communities to really have a say in how we design our research and how we go about our methodology. And the questions that we ask and how we do this, we have to act in a fair and objective manner toward all the involved parties, and that includes business as well.

The health department will continue to support the efforts to complete the pay CH project. And we will continue to work with the Apopka Farm Workers Association

and industry to protect and promote the health and well-being of the citizens in this community. And you see the scales of justice in the slide.

We're from the government. We're here to help you. [Laughter]. They don't believe us. This trust that has to be built. And how do you build trust? You build relationships. You keep going back. And I must tell you that the previous epidemiologist in the county when I first came there, they really didn't trust him, unfortunately. And he was really good at this risk communication. So he did both -- he doubled up as a -- he's an MPH epidemiologist and he has subsequently retired. But he doubled up as our PIO. He did the media, he did all that risk communication stuff. They honestly didn't trust him. So there's a lot of issues in this community. But there was a lot of social injustice, if you will.

So remain objective in dealing with all parties, irrespective of the scientific evidence. Still remain sympathetic to community's concerns.

Where did our notion of best evidence come from? Just a side anecdote here. Cochran. Cochran was a librarian in England in the 1950s. He wasn't an epidemiologist. He wasn't a physician. Best evidence. Evidence based medicine, evidence based public health. We can thank Cochran for that. He was a librarian. That's the evidence that we now of to translate into -- a translate our good science into helping the community to understanding these issues and helping

them to continue to express their concerns to us and build trust which helps us in doing our work.

So there are -- and again it's beyond this community based participatory research opportunities, it's community driven research that we really should strive for. And those research opportunities include maternal child health, they include environmental issues. And once in a while we'll find that Erin Brockovich situation if we look carefully and methodically over time and look at trend and data and listen to what people are saying. And we'll find wells that will be hot in Lake Apopka in some of the drinking wells. Once in a while we get a hit.

And there are other health conditions that we can look at. We can enlarge our prism and look with a view that includes what they're telling us that we need to look at. And it may not just be infant deaths and the fetal deaths, it may also include side issues like lupus in this case. Or respiratory problems living next to emissions from that industrial source or the gasses off-gassing of the landfill. And I would thank you, and I would thank my fellow panelists. I think this session came together very nicely.

[Applause].

MARK KLEBANOFF: Thank you to Kevin and to Pauline and to George for laying out some issues and things for us all to think about. We deliberately ended early.

I guess if we go to 10 we have 15 minutes, if we go to 10:15 we have longer. So we'll entertain some questions.

BILL SAPPENFIELD: I'm going to go ahead and start first. I'm Bill Sappenfield. Excellent presentation did, guys.

Maybe you can solve this problem. One of the presentations talked about over 30 percent increase in number of environmental exposures and then we hear that lead probably one of our most studied environmental contaminants we still don't fully understand so that this equation doesn't sound very good for information and evidence based and sound decision making. Advice?

MARK KLEBANOFF: Why don't we let Pauline and George give alternate views of that.

PAULINE MENDOLA: It is an interesting conundrum, if you will. And part of the problem is that if you look at just looking at lead, certainly it has gone down in the environment over time. And we see that reflective in the population levels because the major sources are going down.

So I guess you could look at it a few ways. One is that there's no reason necessarily to think that the dose operates in a linear fashion. So the difference between one and two may not be the difference between 15 and 16 micrograms

per deciliter if you will. So some people would certainly argue that the low dose effects that are seen may in fact be real but that the curve sort of operates differently at different levels.

Again, it's a challenge. It's hard to know when you would expect to see these end points because there's so many other things that are playing there. I mean all the things that George was talking about reading scores for example. Those are very multi-factorial so there's a lot going in there besides the lead effects. You do see more biomonitoring stuff related to lessening exposure but how to measure that in terms of effect is a lot more diffuse.

GEORGE RHOADS, MD: You know, lead is a toxin and I there's nothing that I said that would want me to make things go backward. The issue I was trying to just point out is that there's a tendency to kind of keep going forward. And we -- I think maybe you want to take a second look before you decide to just keep pressing this issue further and further.

I guess the reason I sort of come in on the evidence based side of this debate is that there are so many exposures, and they're so difficult to resolve that it seems to me we really need to focus on the things that we think are likely to be the most important where there really is some solid evidence. And, you know, one of the really tough issues I think is the business about community driven research or community based participatory research. And on the one hand, and getting the best studies to try to answer underlying questions on the other. You know, if I'm

at a -- if I go to my doctor and he's seeing, you know, 25 cases of whatever it is I have, I'd rather have him rely on the literature which has examined much larger numbers of cases to decide what's best for me than to rely only on his own experience, which I think in public health we sort of like to rely on only your own community.

So you do need community input, absolutely. And the reason I said I think that to do evidence based public health is so difficult is that there's two sides to it. One is to really listen to the community, and the other is to try to educate the community about what the best research actually does show. And that's tough.

UNIDENTIFIED SPEAKER: I'm Tom [Inaudible] from Milwaukee. This is a question for Dr. Sherin who relates to the concept of community-driven research. In your example in Orange County were you able to provide opportunities for community members to participate in the design and conduct of research on their priority issues?

KEVIN SHERIN, MD: During the last five years, and I don't know if this mic is on or not, but during the last five years, yes, I can say there was some of that that was attempted. And how successful we were with it would really be a perception of the community rather than us. I mean, when we have researchers going in there -- the whole purpose of pay CH, for example, is the community helps to design what it is they want done. So we're just -- the health department locally is

doing the pay CH model. That's the model that we use. So the framework is there for us to look at what they are interested in looking at.

Now, as far as the other researchers coming in from the University of Central Florida, the University of Florida, USF, the alphabet soup of our universities in this state and now University of Miami with NCS. I can't say that we've really achieved, quote, community driven research. But as far as the local health department and what we've been attempting to do, yes. And as far as the University of Florida and what they did with the lakes and the fish, yes. The community had raised up those concerns. And they had input. Did they really get to the driven level? I don't know. You'd have to ask the community.

UNIDENTIFIED SPEAKER: Good morning. My name is Jim [Inaudible]. I'm from the University of Florida. Just a question for any member of the panel. It has to do with the non effect and response of people in the communities that feel that as sort of a floor on progress. I was thinking of issues like the lack of increase in the reading scores despite huge investments in education. Potentially a superintendent in a state could say well, we should be witnessing increases as a result of our investments, potentially what's holding us back is the level are these environmental influences.

How do we respond to findings of no effect?

GEORGE RHOADS: I'll respond to the -- respond to findings of no effect? You tell them that. And you tell them that it's an iterative process that you can continue to look at data over time. I mean, we're going to continue to collect data on our infant deaths and on our fetal deaths and on our low birth weight and our preterm deliveries. And we're going to be able to assess that data into the future. And so you don't let it end the way it -- our results were negative. As you can see this particular community it's been involved in a lot of studies. So we -- when we have negative hits on the wells, we put that out in front of them. And we say that there's no evidence of leeching from the landfill. They know that. And then we haven't found direct links between the toxins in the landfill and the community health concerns. We share that. But I to think that it's a iterative process where you go back again and again and you listen to what they're saying and you tell them what you do know. And you tell them what the evidence shows. You tell them the science. And they may not believe you, but you keep doing it.

PAULINE MENDOLA: And I'll just add a little bit. From a methodological point of view, this is the challenge that we have in epidemiology. We deal with real people in real communities with real life exposures. They don't behave like laboratory animals in an experimental study. Most of the things we are interested in are not amenable to things like clinical trials. So we can't adjust or control for everything. And so you always have the problem of unknown, unmeasured confounders.

In a situation that you describe where you make a tremendous community investment in trying to improve an indicator like child reading and it doesn't happen. It's very frustrating and difficult to try to look at what else is going on, what else is changing and it's really hard to prove that, you know, you didn't see that expected benefit because of some other detriment that kind of came in at the same time.

It's certainly possible that it could happen. But it's really, really challenging to measure effectively. The only thing that I would suggest in terms of methods, sometimes community based studies are able to see that. If you have two or three communities that are making the same investment attempting to improve child reading and some succeed and some don't, then you have an opportunity to see what else is going on in those different states or counties or something, to see if you can tease out something like an environmental effect. Environmental effects are often subtle. I mean as people have mentioned our ability to measure them above the noise of sort of everyday life is limited. And so you see effects of 10 percent, 20 percent, 30 percent difference. If that's in fact really true, that's a tremendous public health burden because most people are exposed. But hard to tease from the error that goes with all the measurements. It's a problem. I agree.

MARK KLEBANOFF: And perhaps in getting back to the life course discussions we had yesterday we've sort of now accepted that it perhaps is and unrealistic demand on our prenatal care providers to reverse preterm delivery rates in the

setting that someone has lived 20, 25, 30 years before they ever became pregnant and maybe it's an unreasonable demand on our educators to say that our schools alone can reverse the life course -- the life course impact that children and their parents have had before they ever show up at school. Better schools are probably necessary to solve this problem undoubtedly, but perhaps they're necessary but not sufficient.

UNIDENTIFIED SPEAKER: Yes, hi, my name is Lauren Smith. I'm from the Massachusetts Department of Public Health. And I had a question for Dr. Rhoads that might connect with yesterday's discussion, which is if we think about the fact my understanding is that lead in a -- never really leaves the body unless you can sort of actively chelate it but most people don't have that. If you have -- could you have the NT generational transfer of lead exposures effects as a cause for the fact you haven't -- we haven't been able to see the kind of increases in reading scores or other measures yet? In other words, since the 1970s that's when the lead started to decrease. Perhaps we'll need to see more than one generation of people because of a timing of when infants in utero were -- or fetuses in utero were exposed to lead in critical periods, and maybe we haven't seen it sort of wash out enough. Do we have to wait until we can see the really positive effects of not having lead in the environment when we have pregnant women who don't have lead any more that leeches into the placenta and the fetus?

GEORGE RHOADS: That's an interesting idea. And I think that there are some risk factors for which we were concerned about. Dr. Klebanoff, I know, did some research on moms who themselves had preterm births and then the length of pregnancy and their offspring, and there seemed to be some carryover.

I think with lead, almost all the body lead that a toddler has, the vast majority of it is lead that that child in fact ingests. It doesn't actually come from what came from pregnancy or what he got from his mom. So it's a little harder for me to see why there would be an intergenerational effect, except insofar if mom was -- if her functioning was somehow compromised maybe the way she brings up her child could have some sort of effect. But it doesn't strike me it does.

We know -- I mean in Newark I only have anecdotal studies but one of the folks who took care of lead exposed children back in the '70s in Newark worked with me on the big randomized trial of chelation that she did, and she said when they followed up the kids of those lead exposed kids she worked with -- and in fact, they did not seem to have -- their risk maybe was a little bit higher but it wasn't hugely higher for being lead exposed in the next generation.

UNIDENTIFIED SPEAKER: I'm Jane [Inaudible]. And I want to personally thank Dr. Sappenfield for this session. He humors me when I send him articles about environmental health issues and maternal child health issues.

For those of you who have never used pace environmental health and are working with communities, it is an excellent tool for guiding the work that you need to do at the community level. And I would urge you to learn more about it. It was developed by CDC in ash to, so it's and excellent tool to help you work with the communities.

And then to follow up with Dr. Rob, I think one of the things that frustrates us at the Department of Health is the [Inaudible] work with our partners at DOP because the policy issues [Inaudible] which don't allow us to connect health [Inaudible]. So if we could move to be able to look at some of these [Inaudible] outcomes and school health performance at the state level it would really help us a lot.

UNIDENTIFIED SPEAKER: I could comment on that briefly. There are communities that have been able to do that. There was some work done at Chicago. And there usually are correlations between lead levels in children and their school performance in a across sectional way. But I think the problem is again the sort of the confounding issue as to whether the kids who have lower level school performance, whether it's necessarily due to the lead, not that lead certainly in high levels does contribute, but at these very low levels it may not contribute as much as it used to.

UNIDENTIFIED SPEAKER: I would also add that there's an issue that's an institutional barrier that's not unique to the state of Florida. And that is the FERPA legislation, the federal legislation that protects school health data. So there's really a barrier to sharing data with epidemiology, with public health from schools throughout the United States concerning health. It's protected at another level because it's also classed as education data. And that would include the reading scores and the things that we would maybe want to look at.

UNIDENTIFIED SPEAKER: This question is for Kevin Sherin. In your experience working with this community that's very motivated regarding environmental health issues, have you found that this has facilitated interactions with the community on other health issues and if so, could you share a couple of examples?

KEVIN SHERIN: Yeah. Well, when I first came, you know, there was a real need for specialty care for the medical services to this community. This was one of the communities that had a problem with access. And there was a grant from the Florida Department of Health that had enabled the federally quality health center in the area to add another site. That's an example.

But subsequently, we've done a lot of things with pay CH. You know, we're working with a number of issues, whether it's safe routes to school, whether it's lighting, safety in the community. Obviously the study with the fish. You know,

there are real clear examples of where the community has had input and things have happened and been followed up on.

So there's a very systematic programmed approach that keeps going back and doing it again with the community empowered to a greater extent than they once were. And I might also add that there's also a more of a continuity in the community than might have once existed. With a migrant farm worker population people were always on the move. With the community as it is now, it doesn't have that transient farm worker population. These people have become embedded in the community. So there are long term relationships that build up with city government, with county government, and with the different agencies that are there that support the community and the health and the education and the safety issues.

UNIDENTIFIED SPEAKER: I had a couple of quick questions. We're winding down. But Dr. Sherin, in your ongoing communications with your community trying to explain what you're able to do and what you cannot, even to the point you've got now looking back, is there anything you wish you could have done better, and how might you have done it better?

KEVIN SHERIN: Absolutely. I wish we had started pay CH sooner. But honestly we didn't -- we didn't get that going until maybe three years ago. But when you look at it over a five and a half year time that I've been there, I think pay CH was

right where we wanted to be in earlier in the process. So I can't overemphasize its usefulness as a tool for local public health. And it works obviously in our community very well. And there are other examples in the state of Florida. And I've heard good things about it nationally too. So I would say put pay CH out there sooner.

And also, get the right people at the table. Like I told you when I first went there, they didn't trust the particular personality that was representing both the communications and the epi side of the house, the science and the evidence based. And so sometimes you need to change up the people at the table so you can build that trust back. But this is a community where that trust was fractured for many, many years with the powers that be. And a lack of trust for agricultural, a lack of trust for business. And, you know, there were some bad things done over time.

UNIDENTIFIED SPEAKER: I think perhaps we can have a bit longer break than was scheduled. I see people are looking back. Are there any further questions? We can certainly stay around and people would like to talk, but I see people are going out. So perhaps we could end now.

[Applause]