



## Conference Call Transcript

2004 Bureau of Chronic Disease Prevention Pediatric Obesity and Type II Diabetes Cardiovascular Disease Audio Teleconference  
June 23, 2004 12:30 p.m. ET

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### PRESENTATION

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Operator

Good morning and welcome, ladies and gentlemen, to the Pediatric Obesity and Type II Diabetes Cardiovascular Disease Audio Teleconference. At this time I would like to inform you that this conference is being recorded and that all participants are in a listen only mode. At the request of the company, we will open the conference up for questions and answers after the presentation. I will now turn the conference over to Tisha Keller. Please go ahead, ma'am.

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Tisha Keller

Thank you. Good afternoon. Continuing with our series of audio teleconferences, the Florida Department of Health Bureau of Chronic Disease Prevention and the Diabetes Prevention and Control Program welcome Dr. Michael Goran and Dr. Francine Kaufman. Together we'll discuss the topic of pediatric obesity and type II diabetes cardiovascular disease.

Dr. Michael Goran is currently a professor of Preventive Medicine and Physiology and Biophysics and also the Associate Director for the Institute for Prevention Research at the University of Southern California in Los Angeles. He earned his Ph.D. in Metabolism from the University of Manchester UK and held post-doctorate appointments at the Shriners' Burn Institute in Galveston and the University of Vermont Department of Medicine. Previous positions include Professor and Director of the Division of Physiology and Metabolism at the University of Alabama at Birmingham and Research Assistant Professor at the Department of Medicine at the University of Vermont. Dr. Goran has over 170 peer-reviewed publications concentrating on a wide range of pediatric metabolic issues. Among his many awards are the Krechmer [sp] Award for Pediatric Nutrition Research at the American Society for Clinical Nutrition, the Lilly Award for scientific achievement and he was a 2002 Interdisciplinary Research Fellow at the University of Southern





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California.

Dr. Francine Kaufman is currently at the Children's Hospital, Los Angeles, Division of Endocrinology and Metabolism. She earned her medical degree from the Chicago Medical School and served both her internship and residency in the Pediatrics Department of the Children's Hospital of Los Angeles. Following her residency, she was awarded a fellowship in the Endocrinology and Metabolism Department at the hospital. Dr. Kaufman is also Professor of Pediatrics at the University of Southern California. In addition, she serves as a faculty mentor in the Children's Hospital internship program and served as the President of the American Diabetes Association for 2002 - 2003. Her curriculum vitae outlines the over 35 grants and contracts she's received for her research, posts on the editorial boards of six diabetes research journals and many peer reviewed publications and intellectual properties. Dr. Kaufman's numerous awards include a commendation from the California State Senate for the Soda Ban in the LI Unified School District and the Woman of Valor Award from the American Diabetes Association and Children's Hospital, Los Angeles. In addition, she was a medical expert for the LI Unified School District's banning of soda sales in school movement and was entered into the Congressional Record of the 108th Congress for service in the field of diabetes. Earlier this year, Dr. Kaufman was appointed by Congress as a local legend with the American Woman's Medical Association.

We are honored and delighted to have Dr. Goran and Dr. Kaufman with us today. I'm now going to give you some important CE information related to today's call. Continuing Education credit has been approved for the following health care professionals: nurses, certified health educators and dietitians. Big Bend Area Health Education Center has approved this program for 1.5 contact hours, Nursing Provider Number SBN 2654. Swanee River AHEC [sp] has been approved as a multiple event provider of continuing education contact hours through the National Commission for Health Education Credentialing, Incorporated - Multiple Event Provider Number SL 0078. This program is approved for 1.5 hours of CHES [sp] credit. These are not national providers, so nurses and health educators in states other than Florida must request

approval from the professional boards in their state. All CE credits will be reported to Florida's new CE Broker Monitoring System.

Nurses who want to receive CE credit and health educators who want to receive CHES credits must complete the appropriate CEU paperwork with the correct license number and a legible name and address. The Commission on Dietetic Registration has approved this program for nationwide dietetic continuing education credits, CPEU Level 3, 1.0 Major Sessions, Topic Code CL0000.

Dietetic practitioners who are not in the PDP process should sign and return the CDR prior approval CPE reporting form. If there were not RD's or DTR's in attendance please return the reporting forms and indicate no RD's, DTR's attended at the top. Dietetic practitioners who are under the professional development portfolio process should not sign the CDR Prior Approval Reporting Form. These individuals should sign in on the RD and DTP PDP Education Program sign-in with the Florida Diabetes Prevention and Control [unintelligible] logo at the top. In addition, these individuals should record this activity on their Step Forward Learning Activities log. Please read the flier attached to the CDR forms for more information.

Attendance certificates will automatically be provided for dietitians. Nurses and certified health educators will receive CEU certificates approximately four weeks from receipt of complete, legible paperwork. Paperwork received after June 28th deadline or paperwork with incorrect names or license numbers will not be eligible for CEU credit. Nurses and certified health educator specialists should sign in on the participant roster, complete the AHEC [sp] registration form and the two-page impact survey. In addition, certified health educator specialists should sign in on the CHES sign-in sheet to receive proper CEU credits.

All participants should complete the two-page impact survey for this program, sign in on the purchase event roster and complete the post test for this program. [unintelligible] site administrators should [unintelligible] and completed forms to Tisha Cruz Keller, Florida Department of Health, 4052 [unintelligible] Cypress Way, Bin A-18, Tallahassee, Florida 32399 or fax to 850-245-4391 by June 28th.



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It is not necessary to fax and mail your forms. CA credit will not be issued to participants who have not signed in, provided their license number and a legible address and completed the required forms by that date. If you have questions, we will have a time for clarification at the end of this program. I'll now turn the call over to Dr. Goran. Go ahead, sir.

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Michael Goran

Hello?

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Tisha Keller

Go ahead, Dr. Goran.

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Michael Go ran

Hello?

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Woman

[unintelligible] for a while.

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Michael Goran

Can people hear me?

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Tisha Keller

Yes we can hear you.

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Michael Goran

Well good morning. It's a pleasure to be talking to you today. Thank you very much for the invitation. I'm going to talk through the slides that you have in

front of you. I think you have the PowerPoint handout. I'll refer to the slides in numerical order.

So the first data slide shows the rising prevalence of obesity in children over the last 15 - 20 years. I'd like to say a little word about the definition of overweight and obesity in children first because that's always somewhat confusing. I don't have the growth charts on here but there are now body mass index percentile growth charts for children which shows the relationship between body mass index and age in children. And the current definition that is widely used now is a body mass index percentile above the 85th percentile is classified -- the technical definition is risk of overweight or sometimes overweight. And a body mass index above the 95th percentile is considered overweight. Those are the CDC and NIH-approved definitions of obesity. And the reason that at percentile is used instead of just a simple body mass index like is used in adults is that the body mass index changes naturally with growth in children.

And on this slide here you can see the prevalence of a body mass index above the 85th percentile. And that is relative to a standard population from the late 1960s. So that is used as a reference population. So in other words, if there was no change in the level of overweight in the population, the prevalence would always be 15 percent.

Now you can see here in 1986 we start off in all groups: Caucasian, African-American and Hispanic children with a prevalence of 20 percent. And this steadily increases over time, a little bit of a dip after 1992 for some reason and then a rise particularly among African-American and Hispanic children. 1998 data showing 35 percent above the 85th percentile. Now more recent data that has come out either in paper or abstract from shows even higher levels of the prevalence of overweight, again especially among minority groups: African-American, Hispanic and Native American children.

And the next slide gives a little pictorial diagram of one explanation, one of my favorite explanations of the evolution of obesity showing how we've evolved from what I call the hunger/gatherer to the clicker and deliverer. And this explains the rapid increase in obesity that has occurred in the last 20 years not due to a change in the gene pool, which could not have





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changed, but due to some interaction of our genetic makeup and the environment that we live in.

And this is probably the rapid increase in the prevalence of obesity probably being explained by an interaction between a normal physiology, a normal genetic makeup in an obesity-promoting environment, that is the society has society has evolved to provided a greater abundance of food and a lower requirement for physical activity whereas from an evolutionary perspective we were designed to be hunger/gatherers, to go out and expends lots of energy in physical activity in obtaining food whereas we can obtain food rather quickly and efficiently probably at a lower, much lower nutritional quality.

In other words, this is an evolutionary adaptation because the other point to consider is that actually metabolically, humans were designed to store energy, to store fat for survival. So there is a survival benefit to fat acquisition. Now the only reason this becomes problematic is because fat acquisition leads to negative health outcomes not in all individuals but in susceptible individuals and that's probably where the genetic factors come into play again because there is clearly some genetic susceptibility to the increased risk of being overweight.

The next slide shows just a couple of examples of how this reversal of fortune has also affected children. So some examples there are probably obvious to most of us by now in terms of physical activity and eating behaviors changing to more cramming in schools, lack of safe play, more television, etc. and the more use of convenience feeding behaviors also in children.

The next slide just demonstrates clearly that we can consume calories a heck of a lot quicker than we can expend them. So the quote/quote active meal at the top of \$4, that's the happy part. The sad part is that it's 1,250 calories and that becomes even sadder when you consider how much physical activity you have to do to burn off those calories. And in the bottom it shows you different types of activities. For example, on the far right if you're walking at a general walking rate you actually have to walk for almost four hours to burn off those calories. And even if you're an avid jogger at 8 minutes per mile, you have to jog for about an hour to expend those calories.

So we have created for ourselves this environment in which we can consume large amounts of low nutrition calories, coupled with an environment in which we do not require or need or have access to greater amounts of physical activity.

But it is also important to recognize in the next slide just a very simple statement. We often-- I often have people telling me that obesity is not a complicated problem, it's just because we eat too much or because we're too physically inactive. While on the one hand, that may be a very simplistic approach, in reality it's not necessarily one or the other, but a breakdown in the homeostatic regulation to balance the energy that we consume with the energy that we expend. And this is defined or explained more clearly when you consider that in any given year over a 12 month period the average adult actually consumes 1 million calories. And they also expend 1 million calories if they don't gain weight. And that regulation of those 1 million calories being finally controlled to energy expenditure is the end product of a very complicated series of homeostatic mechanisms that regulate what we call energy balance. And that involves central regulation, signals coming from fat stores to the brain, controlling appetite, controlling physical activity to finally regulate that. But when that regulation breaks down for the reasons we don't know and that's the issue, that that regulation breaks down, there is acquisition of energy. And as I said, evolutionarily we were actually designed to accumulate excess calories that are coming into the system.

Now obesity of course is not just an issue of how much we weigh or how we look but the reality is, as I said, in some individuals there are negative health impacts. And on the next slide, it's just a list of some of those conditions that have been associated with obesity. At the top of the list, and the focus of today's conversation, is cardiovascular disease and type II diabetes but there are others listed there which contribute, at least in adults -- the figure there is 300,000 deaths per year but that slide needs to be updated to 400,000 deaths per year. That was the most recent estimate of the toll in death per year attributed to obesity and its related conditions. And this now is the second-leading cause of preventable death next to tobacco.





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Slide number 8 just mentions briefly -- and I'll talk about them -- reasons why body fat has negative health outcomes. So as I stated earlier, actually there is many benefits of body fat accumulation, one of which of course it's the only major form of energy reserves that we have access to in the body to keep us going. It also has important functions in insulation and organ protection. But we do know that there are negative health aspects of obesity in some individuals.

So the important thing to try to understand -- and this is the scientifically important question -- is why is fat so bad for health. And these are three theories: the portal theory, the ectopic fat theory and fat as an endocrine organ. And what these have in common is the fact that fat is not all equal, depending on where it's stored in the body. The portal theory talks about fat acquisition in the abdominal cavity, sometimes called intra-abdominal fat or visceral fat. This is fat stores around the organs. And this is thought to be harmful because it drains its products including high levels of free fatty acids with direct exposure to the liver through the hepatic [sp] portal vein. So there's an anatomical connection between this fat and the liver, the main metabolic organ of the body. And it's thought that based on products from fat directly exposed to the liver leads to a whole cascade of metabolic events that lead to health risks. And I'll talk a little bit later about those specific factors are.

The second theory is ectopic fat and that talks about fat buildup in organs in which you don't necessarily expect fat to be, the major culprits now being muscle fat and liver fat. So and you can see, if you look with careful imaging techniques, you can see fat acquisition in different tissues of the body, not just in adipose tissue under the skin.

And then the third theory is that we now know that fat is not just a globular fat under the skin. It's actually a very active organ. Metabolically, it releases various signaling molecules, the first of which that was discovered was Leptin [sp] just over 10 years ago, which is now known to signal the hypothalamus and the appetite regulating systems to act as a signal between fat stores and the central nervous system. And now we know fat also releases a whole host of other hormones and cytokine [sp] factors that probably interact, interface with metabolic regulation.

The next slide, which is slide number 9, shows you a cross-sectional image of adipose tissue at the level of the abdomen. At the top of the image you can see the embolicus [sp]. The white ring around the body, that's subcutaneous fat or fat under the skin. That's the kind of fat that you can measure with a skin fold caliper and you can obviously see it in the mirror when you look at yourself in the morning. But the fat that you can't see that's not under the skin is the visceral fat around the organs. And those are the white islands of fat within the abdominal cavity surrounding the organs that I mentioned earlier.

Slide number 10 shows again some epidemiological data of the prevalence of diabetes, which is probably the most measurable indication of the negative health effects of obesity. And you can see state by state in the US going back to 1990 the rise in the prevalence of diabetes. If you look at the slide for 2001, you can see actually California on the left with a prevalence above 10 percent. And Florida is in orange and actually that is not showing in the legend, that is a prevalence of diabetes above 12 percent. So Florida is actually leading the nation, along with some of the other southern states, in the prevalence of diabetes. And that's probably due to the higher prevalence among blacks and Hispanics, which are high in the southern states. And of course the higher numbers in California are probably due to the high numbers of Hispanic population in this state.

That's in adults. The focus of today's talk is in children and the next slide, number 11, just shows you some quick epidemiological numbers on the statistics of type II diabetes in children and adolescents. And we do know that the major risk factors are being overweight, having a family history and being African-American, Hispanic or Native American. Based on clinical observations that have compared the diagnosis of type II diabetes versus type I diabetes, it has been estimated that there is 20-fold increase in the incidence of type II diabetes in the last 20 years. So more and more cases of diabetes are being diagnosed by pediatricians are due to type II, not type I diabetes. And I'll talk in a minute about what that means physiologically.

But we also know that a large number of overweight minority children have what is called pre-diabetes. Pre-diabetes is an elevation in either fasting or post-





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challenge glucose that is high enough to be considered pre-diabetes but not high enough to be considered type II diabetes. That's typically a two-hour glucose above 140 but less than 200, which would be the definition. If it was above 200, that would be type II diabetes. But it's also becoming readily apparent that most overweight children already have other risk factors in addition, including high blood pressure or high cholesterol.

The next slide just shows you an example of the types of extreme cases and Dr. Kaufman will talk more about some of these clinical situations. This just shows you an unfortunate situation of a 352 pound 14-year-old child who volunteered. This was not a clinical test, this was a child who was otherwise healthy and volunteered for a research study and turns out to be type II diabetic as well as having hypercholesterolemia and hypertriglyceridemia [sp]. And I show this because it's a good example of children who are out there who are otherwise healthy who probably need to be screened more carefully for underlying conditions that they or their families may not be aware of that if we can catch those conditions early and get them under control, we could avoid the long term effects of having diabetes or heart disease for a long time.

The next slide is rather complicated but when I give the lecture I like to build it up more slowly so I'll try to talk you through it. But it talks about the importance of insulin resistance at the center of this diagram. Being the main physiological component of type II diabetes, of course the main difference between type II and type I diabetes is that in type II diabetes there is plenty of insulin being produced, in fact sometimes too much insulin. And that's because the peripheral tissues upon those it acts like muscle and liver become resistant to the effects of insulin. And if you become resistant to insulin, your pancreas has to produce more insulin to get the job done of clearing glucose from the circulation.

Numerous factors shown at the top affect insulin resistance, like physical activity, dietary factors, obesity-- the different fat tissues on the left. Those are the modifiable factors and then the non-manufacturing factors like at Misty [sp] and genetic factors. Those factors also more him more insulin-resistant. So if you are African-American or Hispanic, you are more insulin resistant, regardless of

your diet, regardless of your body fat content.

And then the other factor in children that's important is puberty. So puberty itself contributes to greater insulin resistance. The more insulin resistant you are, the most you have to challenge your beta cells to pump out enough insulin to compensate to get the job done of clearing glucose so you can maintain glycemia at the expense of high insulin. The theory is that in susceptible individuals who are unable to compensate, and that's the arrows on the right hand side going down, failure to compensate at the level of a beta cell means you cannot produce insulin for that insulin resistance and then type II diabetes emerges.

Moving onto the next slide this shows you the continuum of insulin resistance that exists in the population of children. And I'll talk you through this slide. On the Y axis, the vertical axis, we see the amount of insulin secretion by the pancreas relative on the X axis to the degree of insulin sensitivity of the tissues. On the far right-hand side of that graph, you can see a healthy weighed pre-pubertal let's say Caucasian child. Tissues are very insulin sensitive. There's not much insulin resistance whatsoever. You can see at that level the relationship between secretion and sensitivity follows this hyperbolic pattern. So if you're very insulin sensitive, you don't need much secretion.

But as you gain weight, as you drop your insulin sensitivity going down to 8, you can see the rise in insulin secretion that is needed is very poor. This hyperbolic relationship becomes very important as you move to the left going up the hyperbola if you consider, for example, the Hispanic or African-American overweight pubertal child who has an internal sensitivity of less than 2 is now challenging the beta cell. And if that cell becomes more insulin resistant, if insulin sensitivity falls by a small amount you can see you get a huge increase in secretory demands.

This is an attempt to explain why the combination of risk factors like ethnicity, overweight and puberty become very metabolically dangerous because of the increasing challenge to the beta cell to secrete enough insulin to sustain that level of resistance.

In the next slide we see some data from some of our studies in overweight, Hispanic children which shows





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the prevalence of impaired glucose tolerance, or pre-diabetes, by severity of the obesity status. And you can see there that across levels of obesity about 30 percent of these Hispanic children already have impaired glucose tolerance or pre-diabetes. But interestingly, the severity or the prevalence of impaired glucose tolerance was not necessarily influenced by the severity of obesity.

So the bar on the far right, for example, those are children who are above the 99th percentile for DMI. So those are children who are probably above 250 pounds. So the prevalence of impaired glucose tolerance in those children was no dramatically different --- in fact it was very similar to overweight children who might have prevalence-- who had a DMI at the 35th percentile.

In the next slide, which is slide number 16, I'm showing you another risk factor that I haven't mentioned and that is pregnancy factors. So in fact, any mutual exposure to gestational diabetes increased the risk of diabetes in children quite dramatically. And that is shows in their slide here and which we saw in this study and which the prevalence of pre-diabetes 2s twice as high in children who are exposed in utero because their mothers had GDM during their own pregnancy. So this is another point to remember that control of maternal glucose and insulin may have significant impact on a newborn [unintelligible]

We're going to skip that next slide because it's a little complicated, to the next slide, which is slide number 18, which talks now about what's called the metabolic syndrome, another focus of today's talk. The metabolic syndrome is defined clearly in adults as clustering of metabolic risk factors. Those risk factors are shown there high blood pressure, high fat, high glucose, low HDL, high triglyceride. If you have three or more of those risk factors, you have what's called the metabolic syndrome. And in adults the cut points for those things are very clearly defined. For example high central fast is defined in women as a waist of conference above 100 centimeters.

So if you have three or more of those risk factors, you have what's called the metabolic syndrome. A metabolic syndrome is a very significant risk factor for development of type II diabetes and heart disease and, in fact, mortality from heart disease. If an adult

male has the metabolic syndrome, you have a four times higher risk of dying from heart disease at some point in their life. Now among adults about 20 to 30 percent of adults have the metabolic syndrome. It's particularly higher, again, among overweight adults and Hispanic adults, one in four have the metabolic syndrome. More and more data is beginning to emerge that shows that overweight children also have these risk factors that we need to be screening for more carefully.

The next slide, which is number 19, shows in a group of 150 overweight Hispanic children, the percentage of those children that have the different risk factors. Now for those risk factors, there age-specific and ethnic-specific cut points which can be used. This graph shows that 60 percent of these children has a high waist circumference which is not that surprising because they are all overweight to begin with. But more surprisingly is that 60 percent have low HDL cholesterol. Almost 30 percent have high triglyceride levels. Almost 30 percent have high glucose and almost 20 percent have high systolic blood pressure.

And if you look on the next slide, number 20, you can see the clustering. This slide shows the percentage of children with a number of features of the metabolic syndrome. On the far right of this slide, it shows that almost 30 percent have three or more features of the metabolic syndrome and 40 percent have two or more features of the metabolic syndrome-- sorry, two features. In fact, 90 percent of these children have at least one risk factor.

And this is thought to be driven, and this shown on the next slide, this is thought to be driven -- this goes back to insulin resistance -- you can see here on this next slide, this is a level of insulin sensitivity by a number of features. The low insulin sensitivity, again, means you're very insulin resistant. And you can see that, a the number of features increases, insulin sensitivity decreases or insulin resistance increases. So the more insulin resistant you are, the more features of the metabolic syndrome you have. This has been shown pretty clearly in adults and now we have data in children showing that the mechanism is probably the same, that insulin resistance plays a very central role not only in driving risk for diabetes like I mentioned earlier because it puts stress on the beta cell, but also for some reason contributes to these greater risk factors for cardiovascular disease.





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So that's some of the underlying pathophysiology of the condition. I'd like to talk for a minute about some of the good news that we have on how we can use that information to try and prevent obesity and type II diabetes. And this will be emphasized more in the next talk in which we'll focus again much more on the management.

But slide number 23 just kind of gives a quick list of some of the factors that we know are thought to be protective or preventive. At the top of the list is breast feeding. This is not in terms of any order but this is one important public health approach because there is some evidence, the jury's still out and the evidence is mixed, but there is some good evidence to support that duration and extent of breast feeding protects not only from high body weight later in life, but reduces risk factors like blood pressure later in life.

In terms of physical activity, we do know that increased television viewing is associated with physical activity and we do have interventions in which it has been shown that reduced television viewing reduces obesity. So we do know that in children we have an advantage of being able to teach healthy eating early in life, which hopefully will have lifelong benefits. We do know that family-based approaches are more effective which lead to more sustainable effects. And as I mentioned earlier, screening is important because oftentimes these conditions can go unnoticed. So we have to screen more carefully for some of these risk factors. And we do know in adults that lifestyle intervention can be effective at preventing diabetes. Unfortunately, we don't have very good information on the types of lifestyle intervention that can be effective for preventing disease but we can guess at what some of those might be.

Another factor to recognize in children is that a focus on body weight reduction, or BMI reduction may not be that helpful, in fact could be harmful. Slide number 24 just gives a couple of reasons why a focus on body mass index may not be that helpful at least in children. And the slide after that, slide number 25, shows another more important reason which is that dieting in children, focusing on body weight as an outcome actually leads to greater weight gain. And that's a very interesting study. In almost 7,000

children it showed that the frequency of dieting was associated with greater weight gain: not less, not similar but greater weight gain over three-year periods. The focusing in dieting was not only not helpful, it was more harmful for long term weight gain. This is the reason why a focus on body weight exclusively is not going to be that helpful and why we need to focus more on some of these risk factors by designing interventions, designing population approaches or individual management approaches that focus on some of these risk factors. I'm not saying we should ignore body weight, but we need to focus on getting some of these risk factors under control, especially in some of the more overweight children who are coming into the clinic.

Slide number 27, I'm going to start to speed up because I think my time is running out here, talks about some of the nutrition approaches. And I want to make a point here that nutrition approaches are [unintelligible] studies. We don't have that much information. What we do know is that large school-based studies which are very cumbersome, very expensive, very politically charged right now; we do know that, yes, we can change nutrition in schools. We can get healthier food on the food lines like increased fruit and vegetables and reduced fat items. But as yet those studies have shown very little effect on outcomes, including BMI and other metabolic outcomes, and have shown that it is very, very difficult to extend those effects beyond the school. And this comes back to the importance of the family involvement, on the community involvement, factors going well beyond the school involvement being involved in this syndrome.

The next slide just talks about the importance of macronutrients. There's a lot of discussion right now. Obviously this issue of low carb diets, glycemic index becoming and they're making the population revolutionizing some of the way we think about foods which ignores really the real issue, which is not necessarily the amount of carbohydrate, the amount of protein,. But it's important to think about the types of fats and the types of carbohydrates being more important not only in terms of weight regulation but in terms of risk regulation. For fat, we need to think about not just low fat foods, but exchanging saturated fat and trans-fatty acids out of the diet with more healthier plant-based foods into the diet. And for carbohydrates, getting the simple,





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unprocessed carbohydrates of the diet and replacing them with process-- excuse me, unprocessed or high grain, high fiber, low glycemic index carbohydrates.

And in fact, the next slide shows a study from last year which compared two dietary approaches in 14 obese adolescence. And the dotted line, a traditional 12-month intervention focused on low fat, step two type diet showing a change in BMI which in fact tended to increase in those children versus a diet which focused on reducing the glycemic load, which basically is talking about more complex carbohydrates that have a lower glucose release. That type of dietary approach led to a reduction in body mass index. I'm not necessarily endorsing that approach, I'm just saying that other alternative strategies need to be focused at that talk about the types and quality of dietary carbohydrates and fat.

Another study, just quickly shown there, is another example of a very simple intervention among Native American youth which basically just promoted increased water use, providing healthy drinking water in schools. Some of the sodas in vending machines with water, this is a study of native American youth showing an improvement in 30 minute insulin, which is an index of insulin secretion after three years.

Very quickly, physical activity obviously important. That's clearly a message we hear every day: we have to be more physically active, we have to get children to be more active. The reality is it's very difficult to get overweight children to be more physically active, using conventional aerobic type activities. So we have to think about what types of physical activity may be useful for particularly overweight children.

The next slide, number 32, shows the data from the TV trial I mentioned earlier, just showing -- this is a six-month intervention trial which reduced TV viewing, you can see in the left panel, from 16 hours a week, which is the average consumption of television viewing down to almost 8 hours per week. On the light panel you can see it's a very subtle effect, but the rise in body mass index in the treatment arm was lower than the control arm.

But another important aspect is to think about the types of physical activity which may be useful, particularly in overweight children. And we are focused on resistance training as another modality

which may be a from of exercise that very overweight children can actually perform and succeed at and feel good at. And this now is a from of exercise that has been endorsed with appropriate supervision, has been endorsed by several large organizations.

And slide number 34 just gives you some data from an ongoing study that we have in which 16 weeks of oversight boys, that's twice per week for 16 weeks, actually does not change body weight. We don't even have to talk about body weight as an outcome but in fact you can see there so far in this data, all of the children who have gone through the intervention, have had a dramatic improvement in their insulin sensitivity. That's about a 50 percent improvement in the solid lines and a deterioration in the control children. This is important, if you think back to the stuff I mentioned earlier, if we can improve insulin resistance, increase insulin sensitivity we can have multiple effects that lowering the stress in the beta cell to secrete insulin. We can improve the risk of the metabolic syndrome and reduce the risk factors for the metabolic syndromes but not even talking about ways to changing the diet. That's just one example, I'm not saying that's what we need to be doing in isolation but need to consider that as a feature of interventions.

And just to wrap up, I want to just mention what we're up against from a public health standpoint and a policy standpoint in terms of the incredible media focus, incredible media stresses on promoting obesity through television viewing. This is a report from early this year which is very good reading, giving some incredible statistics like children watch 40,000 TV ads per year. That's not just because they watch TV, that's because TV is a lot more ads per television hour. So double the amount of TV ads and you can see there that TV ads are primarily for unhealthy foods like candy, cereal and fast food.

And the next slide just shows you financially what we're up against in terms of the advertising budget. For McDonalds it's \$665 million a year. even for a single food item like Pringles, it's \$30 million a year, still 10 times the budget of NCI Five a Day Health Campaign. That's just one annual year for the whole study. So there has to be more emphasis not just on families and individuals but on industry to cater toward this problem.



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And the next slide, which is the almost last slide, just gives some examples of how big food is beginning to at least come around and make small steps in trying to recognize that there is some responsibility from big food industries to try and control the types of portion sizes, the types of foods that are offered, offering healthier choices. So think we need to do a better job of trying to get industry to make healthier choices available and promote them in our communities in their environment so that we can create the next food evolution, shown in the last slide, which is to transition from a happy meal on the left to a happy, healthy meal on the right.

So that kind of brings me to the end of what I had to say and I'm not sure if we're taking questions now. We're at the end where I think I'm turning it over to my colleague, Dr. Kaufman.

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Francine Kaufman

Thank you, Michael. I can't quite have that great accent Michael has. I have a Chicago twang, so you'll have to bear with me. And I'm going to go through my slides, which I'm just going to mainly focus on the diagnosis and the treatment of type II diabetes. If I have a little bit of time at the end, I think some of my health promotion messages are a little different than Michael's. And mine are a plea for all of us to become advocates in this arena in which perhaps we can make some changes for our patients and actually for ourselves and for families and people in general in this country and across probably the globe.

So the first slide just kind of shows the natural history of type II diabetes. And this is a slide really meant to show that natural history in adults. And the big question we have to kind of ask ourselves at this point is this the exact same kind of event and series of events that we're going to see in children or is there maybe some temporal differences, and obviously there are. This is not a very indolent disease with a long lead period if it's occurring in 12- and 14-year-olds. So there's certainly a much shorter time period in which we have this genetic susceptibility impacted on by these environment factors around nutrition and physical activity that

lead to overweight causing insulin resistance. So that period of insulin resistance must not be very long, and as Michael suggested, markedly impacted on by the event of puberty. So we're seeing a lot of these children presenting during the pubertal age range.

And we know these children have high insulin levels. A significant number of them have as well a dyslipidemia [sp] and a high blood pressure that puts them at risk perhaps for early development of cardiovascular disease. And when is early development? Is it when these children in their late 20's or early 30's or 40's? We don't know but certainly one could be concerned that 20 years down the line when these individuals are now in their mid-30's that we're going to be looking at a cohort of people growing up with significant morbidity and if not mortality.

Then there's this period of IGT, or pre-diabetes, and how long does this last and how critical is this? Is it transitioning into frank onset of diabetes? And then the time period after diabetes in which there is ongoing hyperglycemia and a rather unfortunate poor control of this disease process leading to microcirculatory abnormalities or retinopathy, nephropathy and neuropathy and macro circulatory issues around cardiovascular disease risk. When are these children going to have these complications that could lead to disability and death. And that's what's unknown and that's what certainly is very compelling in making us not look at this as a little bit of diabetes but rather in fact a very significant disease in children and something that really needs to be addressed.

So the next slide shows this rather significant increase in incident cases of type II diabetes and I think my colleagues and I get together at a lot of meetings and bemoan the fact that in the '80s and early '90s when we were beginning our careers, although some of us weren't necessarily beginning at that time, there was very little type II diabetes in our clinics. And then sometime in the mid-90's, we started to see more and more new onset cases being type II diabetes in our overall diabetes pediatric population. And now, depending on where you are, we're at the perhaps 25 percent range in my own clinic in Los Angeles. So of our 300 new onsets last year, 25 percent of those children under the age of 18 had type II diabetes, where in 1992 there were closer to 200 children and very few had type II diabetes.





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Some of my colleagues in urban areas in inner cities have more like 40 percent of the patients they're seeing with new onset diabetes being type II diabetes.

The next slide shows that these-- some the characteristics of these children, again the overall age is during puberty. There have been now pre-pubertal children and more and more of them being reported with type II diabetes. There is a supposed four-year-old native child with type II diabetes. And I always kind that the race is on: who can find the youngest one. Although we will continue to find most of them at the time of puberty. There are more girls than boys that present. They are all with a BMI greater than the 85th percentile and most of them belong to ethnic racial minorities. There is a strong family history in first and second degree relatives who have type II diabetes. I mean the typical family we will see is they will come in with a pubertal child. Mom will say she had gestational diabetes or a little bit of the diabetes. And grandma is accompanying them and in a wheelchair perhaps already has had a stroke or an amputation.

And then a lot of these children, depending on which series, have acanthosis niagrancans [sp] which is this classic darkening and thickening of the skin around the neck and in the flexural [sp] areas. And a lot of us are using this as kind of a hallmark of risk in overweight children to begin with, but this certainly not something uncommon at the time of presentation of diabetes.

Then how do these patients present and the next slide is they are symptomatic. I mean we are not finding these kids by going out and doing a lot of screening programs. And in those programs that have been done looking for children either in the overall childhood population or even at-risk populations, we don't see a lot of silent type II diabetes. Most of these children present symptomatic and at that point in time have usually a relatively high blood glucose level and a high hemoglobin A1C. Most of them have lost some weight as a result of their glycoceria [sp] and a fair number of ketosis and if not, mild diabetic ketoacidosis [sp]. And I think some of us are appreciating that some of this diabetic ketoacidosis may not even be that mild, with pH's at the 7.2 - 7.3 range, low bicarbonates less than 15 and a rather dehydrated and ill child.

I think the concept that a child at the presentation of type II diabetes isn't very sick is not true. And in fact, Arlen Rosenbloom [sp] in his group reported about eight deaths of type II diabetes that they had heard around the country in, I think it was about a year or two year time period, at presentation. And that would actually probably be more deaths at presentation than in the type I population for an equivalent number of kids. So this is not necessarily a benign disease. This is not something that you can just put a child on a ward and worry about them later. These children need intensive level care and need meticulous attention, particularly if they have ketoacidosis with it and very high blood sugars and hyperosmolar [sp] dehydration. Very meticulous attention paid to their metabolic abnormalities and nee really need meticulous attention by somebody who understands diabetes. A lot of these children, oh it's just type II diabetes, we don't really need to get the endocrine team involved. And again, there is nothing farther from the truth.

The children, theoretically by definition should not have islet cell autoimmunity. That should be reserved for our type I population, but as you can see here, some of them do, and maybe it's 15 percent, maybe it's 20 percent, it's a little unclear. Does this mean that they're really not pure type 2's? Are they maybe some element of type 1 plus type II? I think we're going to have to figure that out and it's unclear to most of us but there is an element that some will have autoimmunity, but characteristically they should not. That should be reserved to the type I population. This could be assessed by getting islet cell antibodies, either anti-GAD [sp] or ICA itself or some of the other biochemical antibodies. They should have presentation of C-Peptide. Once you've resolved their initial metabolic abnormalities and indicating that they don't have complete destruction of the beta cell myth.

There is a picture of acanthosis [sp]. And this can be somewhat graded and obviously is of extreme interest. I think the typical thing I hear from a family when the child comes in and I start looking at the neck, the mom or dad will look at me and say, "Well you know, doc, we've been scrubbing that neck with a Brillo pad for the last year and we just can't get that dirt out. And when you actually tell them that what it's due to and what it's indicating, they actually feel a little relieved that their child is just not a hygiene





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issue but that actually this is something that they could not have helped and is a result of the underlying insulin resistance.

Now let's get on to the treatment. I mean again, I think we are compelled with the potential of what this diagnosis means at this age group to meticulously manage their glucose, their blood pressure and their other cardiovascular risks, their lipid levels. And I think I have, along with a lot of my pediatric colleagues, started to focus for the first time on really the abc's of type II diabetes in children and not just the abc's of type II diabetes in adults. And we have to really pay attention to all of this.

There is a little bit of data emerging that was presented at the ADA and the pediatric research meetings that chart reviews in some of our pediatric clinics maybe up to 40 percent of the kids have abnormal blood pressures that nobody's doing anything about. And certainly we know there's a lot of abnormality of lipid. We're still kind of debating what the lipid LDL level of treatment should be but I think we should start realizing that this is more than just about sugar. This is about sugar, blood pressure, dyslipidemia, obviously other issues around overweight and some of the other issues of liver disorder, sleep apnea, all of these things need to be addressed in these children. And we can't just focus on sugar alone.

So the overall treatment protocol should involve a multi-disciplinary team with a rather broad look at the child and the family together. We should set glycemic targets and they should be to attempt to normalize glucose if we can and realizing that some of this is in teenage years and perhaps have A1C target as less than 7 as being ideal but not say, "Well you know, boy they're teenagers, there are a lot of issues in this family, a lot of other diabetes in other family members and they're not doing well so we can't expect the kind to do very well." I mean that would be an unjust approach for the child over the long term.

Diabetes education involves the patient and the family. You know I think the typical 16-year-old girl doesn't want her mother anywhere near her and I think we have to figure out very unique ways and innovative ways to get this family working together around diabetes. And this involves the entire family

and we need to stress this. The role of improving lifestyle will be critical for this child. We need to address what kind of food issues are in the family. Obviously we need, as advocates, to address what kind of food issues are out in the environment for these children, particularly in school and at the same time their ability to have access to good physical activity.

I'm going to stress a little bit what is the pharmacotherapy that we have and what are some these regimens and how are we going to assess efficacy, effectiveness and outcomes in this patient population. So the next slide is again the treatment goals to look at an A1C hopefully less than 7 percent, to certainly attempt to eliminate symptoms of hyperglycemia and then to reduce microvascular complications over the long term, to attempt to get to a reasonable body weight through-- and then if we can to maintain it. And then of course, this big issue of improving cardiovascular risk factors as well. And then of course setting this in a teenage child for the most part and looking at all the other physical and emotional issue and helping them achieve well-being with reducing risk for what's out there in the environment: drugs, sex and alcohol, all those other things that pediatric providers need to focus on.

So here would be our glycemic targets. We would like to near normalize glucose. There's a normal range for fasting post-prandial bedtime and A1C, of course, what would be our goals are to have it higher than that because we can't seem to achieve better or normal values with the therapies that are available to us. And then of course, the goal and when we would suggest action, we don't want someone to have too low of blood sugar or too high. And so we need to look at changing regimens if we are not achieving our goals in those kind of ranges. Certainly to whatever they're on, if they're unable to achieve an A1C less than 8, we would not want to keep the patient on the same regimen they're on, we want to move them onto a more effective regimen and work perhaps trying out different regimens to get them to these targets.

The next slide again stresses the importance of the whole family being involved in diabetes management with this child. So this is a study done in African-American families. So group one had direct family supervision and group two had no direct supervision and you can see the difference with those A1C's at





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the end of the study period. You know, children who are teenagers are extremely still dependent on their parents, no matter what they may say and what they may want to feel they're able to do, most of them have enough trouble getting their homework done and their rooms clean and all the other avoidance of risky behaviors. And they can't be left alone to manage their diabetes. And we have to work with whoever is a significant member is. A lot of times it's grandma, a lot of times it's an aunt or uncle or an older sibling to really get effective management and we can't leave this to the child alone.

Then the next slide is kind of dealing with what do we really know about lifestyle alteration in children and how will we achieve this., where will this be done. Well we certainly know that lifestyle and the environment are risk factors for overweight and type II diabetes. And there is a consensus that if we could modify this, it would be primarily goal for obviously all of us. It might even lead to a remission of the diabetes. But where are we going to do this? I mean how are we going to deliver an intensive lifestyle to a lot our patients who are socio-economically not able to pay for personal trainers or gym memberships or camps that will take your kind all over the place and get them physically active, in environments in which there's not a lot of resources perhaps in their neighborhoods and in their communities and in schools in which we're not getting the kind of lifestyle that we need to have these children exposed to. We certainly know that the results in adults are difficult to achieve and we don't know if they'll be more effective or less effective in kids. It's labor intensive and expensive so we're a little unclear where we'll be able to achieve these but a lot of us are looking into hopefully improving the entire environment in which these children live so that some of these lifestyle modifications will be easier to put in place both for prevention and then for treatment.

The next slide just shows you some statistics from my own center in 2002. And you can see that these are our type II patients. We had 100 that we were actively following, 100 who had really been with us so that we could establish that they were really our patients. And this is after the first year of having type II diabetes. You could see we had very few that were in the low age range and actually almost all of those were 9- and 10-year-olds and their A1C's were

not too bad. The next age range of 11 to 16, we start to get these A1C's that are not optimal. These A1C's are actually no different than what we have in our type I population. So the concept that this is either better or necessarily a lot worse than our type I population doesn't really look like it from our overall data. The question is, what happens after we watch this cohort for a lot longer, in that we haven't had them for the kind long periods of time that we have type I. and there's emerging evidence that after the three and four year time period that the A1C gets a lot worse for most of these patients and a divergence between what we're seeing in our type I and our type II, and that their A1C's are actually worse than the type I population. So I think we can't again view this as with only type II diabetes, they don't need much attention, they don't need much medication. These children really do and you can see that even with that and our intensive approach, we're not able to do a whole lot better than we can with our type I patients.

The next one is just all the different kinds of drugs we could use and where they focus their activity, what do we know about these drugs in children? We have no data on the Celphona Ureas [sp] or Repaglanide [sp]. We certainly have lots of data on exogenous insulin. We know insulin works very well. We know pediatric endocrinologists and diabetes pediatric care providers have the most experience with insulin. And a lot more of our children are on insulin compared to adults with type II diabetes as a result of that and as a result of its known efficacy. The only drug really approved in pediatrics at the present time is Metformin [sp]. It has both efficacy and safety in the pediatric population and as a result, is a first line oral agent in type II diabetes.

Eckerbose [sp] sounds really good but when you tell a child that they're going to have a lot of flatulence and perhaps stomach aches, I always say that the average adolescent boy has no problem passing gas but it's all got to be on his terms so a lot of them don't really prefer to use that. although I'm not sure we've given it a really good, good trial in children with type II diabetes.

And then the TZD's in the next area, we are gaining. There has been a type II pediatric trial with Avandia. There results are out to the FDA and some issues





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around when it will be formally approved. But the TZD's are certainly getting more and more use in the pediatric population.

And the next slide just shows again all these different agents and where they act. And then I think if we go to the next one, we can kind of see that. What we don't know is this kind of progressive decline of beta cell function that occurs in adults. Are we going to see the same thing in pediatrics? Is it going to be even faster, which would mean we would want to be maybe more aggressive and not think of monotherapy but maybe dual therapy earlier than later.

So the next one kind of shows an overall schema that's been developed so a type II child comes in. Could we try diet and exercise alone? Certainly if they're not sick and their A1C's are not that high, we might be able to try that for a period of, I don't know, three months if you want to as long as you're watching them closely and supporting them to be sure they're not getting sicker. If there's no better-- in other words if you're not able to achieve a A1C less than 7 in a fasting plasma glucose lesson, 120 by the end of three months or perhaps even earlier, then you'd have to go onto therapy. And this is for a child that's coming in not sick at all in the beginning. And then you could try Metformin [sp] alone. If you can achieve those targets with Metformin alone, you can keep them on that. If not, think about dual therapy. What would be the add-on? Is it really a [unintelligible] urea TZD, I don't think we really know for sure. And then if you're not able to do it with dual oral therapy, to consider insulin or I suppose you could consider insulin as a second like if you're unable to achieve target with Metformin alone.

The next one kind of just truncates it a little bit and that's-- I use this in a talk outside the US but you wanted to say what's the only difference here is if you're not symptomatic from the beginning and you have a high glucose. And whether you want to say 250 or 300 milligrams per deciliter, you need to start insulin at the beginning along with attempting lifestyle modifications. And then you can go onto weaning insulin by adding Metformin to that regimen, try to get off the insulin and then be on Metformin alone at some period in time. And we're all kind of working on these algorithms. I don't think we've actually set them in stone and hopefully at

some point we'll have some evidence behind what is the most efficacious and safe in these populations. And one way we'll get there-- I just want to mention the studies to treat or prevent type II diabetes in use. And the Today Trial, which is the treatment part, we have just started. There's 12 centers involved across the country. And this is to compare the efficacy of three treatments, Metformin alone, Metformin plus intensive lifestyle and Metformin plus TZD actually Avandia in incident cases who have had the diagnosis of type II diabetes for less than two years.

The goal of this will be to enroll 750 children with 250 in each one of these arms over a treatment period of three to five years, with treatment failure defined as an A1C above age that cannot be controlled in these arms. And then there will be an add-on period of Glargean [sp] alone. And if that can't control blood sugar, then to go onto any insulin regimen somebody wants. We'll be looking at a number of outcome measures beyond glycemic control but measures of insulin sensitivity in secretions, body composition, fitness and physical activity, nutrition, early complications both microvascular and cardiovascular risk, quality of life and cost. And hopefully in seven years or so-- I guess well maybe five years or so we'll be able to give you some answers.

I just want to briefly touch on can we differentiate type I from type II diabetes and again say that this is a study from [unintelligible] and this is pretty much what all of us are seeing, comparing a cohort who really look like they have type II diabetes compared to a cohort that really look like they have type I diabetes and we can look at the type II have those kind of issues. And then again, the type II do have DKA. Most of it's more mild. The type II do have preservation of c-peptide but so do type I patents for a while, particularly in the first year as we go to diagnose them much earlier and attempt to preserve c-peptide so that their overall management is a lot more effective as well. And then just showing that the level of antibodies that she found in her mainly Hispanic cohort of type II patents, and again are all these pure type IIs or do they have an element of type I? Do they have something like double diabetes and we need to kind of work this out over time.

So what are the barriers to the accurate classification is, as there's more and more overweight in children,





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we're going to see more and more real type I's come in overweight as well and pick them up earlier and don't give them a month period of kakexia [sp] before they're diagnosed. Then they won't be wasted and they may still be overweight. 15 percent of the minority population have a positive family history of type II at baseline. And even the ones that come in with type I has a strong family history of type II and does that mean they're at risk for double diabetes?

Three times increase in family history of type II, so this is translated into three times family history of type II in our patients with type I, this overlap of the c-peptide measurement and onset later on, and then this high percentage of those with type II in adolescence who actually have ketosis at onset.

And let me just briefly touch on the co-morbidities. So just to show you that there is a difference in the co-morbidities between the type I and type II population, this is data from my center showing that the type IIs have much more elevation of blood pressure than the type I population does and then are we effectively looking at these blood pressures every time we see these kids in clinic? Are we doing what is really appropriate as far as managing them, which would be to treat them at a blood pressure above the 95th percentile, not even the 97th and just try to get it under the 90 with treatment.

And then just to show you, this is kind of some of the overall significant poor outcomes that these children have. This is a report by Heather Dean, looking at first nation youth, youth that were diagnosed before the age of 17 and the follows between 18 and 33 years for kind of a mean observation between 10 and 15 years. And they had poor glycemic control at that time, a 9 percent mortality, 6 percent were on dialysis, there's a 38 percent pregnancy loss. And these are pretty significant issues when you're looking at a population now that's maybe in their late '20s and early '30s.

And then the same kind of thing looking a PEMA use, that at diagnosis they have high cholesterol, hypertension and microalbuminuria [sp], and then after 10 years a significant risk to their kidneys. And you can see what their mean A1C value, which was extremely high, that we are not able to do a good job of controlling both the glucose itself, the risk for

microcirculatory complications and the risk for macrovascular complications.

So I think maybe I'll stop here. We don't have very much time. I think Mike talked a little bit about screening. And I know there's a couple of questions about screening. Maybe it will easier to just answer them as questions. And then I suppose my only other plea before I conclude would be that to really make an impact for these children who are already diagnosed with type II diabetes or are trying to get a handle on what we're now calling an epidemic of the onset of type II diabetes in youth, we have to look to improve the overall environment for children and families in this country.

And then to do that we need advocates at every level and there is nothing more powerful than health care providers as advocates. If you get involved in your local school, start looking at what's going on in your local school nutrition and activity and try to make a difference in your local school, you will have made a significant impact for all of those children that movement gets kind of carried up and you can end up with things like the LA Unified soda ban and now the LA Unified ban on junk food. And already hopefully a significant improvement in what will happen in every state across this country in at least making school a safe environment for children to go to every day as a health promotion environment rather than an environment where their health is deteriorated.

So I think I'll stop now and I guess we will take questions.

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QUESTION AND ANSWER

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Operator

Thank you. The question and answer session will begin at this time. If you are using a speakerphone, please pick up the handset before pressing any numbers. Should you have a question, please press star, one on your pushbutton telephone. If you wish to withdraw your question, please press star, two. Your questions will be taken in the order they are received. Please stand by for your first question.





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Tisha Keller

Operator, we have a question here that was e-mailed before the teleconference, so I'll start with that one. The question is, one of my pediatricians has been screening children using elevated insulin levels and c-peptides as criteria as advised by a pediatric endocrinologist at a conference he attended. Another pediatrician asked me for some researched information about using these tests as a diagnostic tool. Is there any documentation or do you all have any diagnostic tools for screening in children?

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Francine Kaufman-

You want me to answer that, Mike?

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Michael Goran

Yeah.

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Francine Kaufman

OK well screening for what? You know that's certainly not going to pick up anybody with diabetes. So diabetes is -- the diagnosis is made on glucose values, so either fasting above 126, ambient glucose above 200. So that's really the only way to make that diagnosis. Whatever the insulin level, it doesn't matter if you have abnormal glucose levels. I think there's a lot of people now out there screening for insulin resistance or metabolic syndrome or pre-diabetes. And what are we doing with all these patients that we're finding, I guess is the better question. I think inside of research studies these are very, very useful tools if you want to start to look at children with insulin resistance, children with metabolic syndrome. And you can't just use those alone. If you want to use that in the clinical arena, we're not exactly sure that any cutoff point is. It certainly makes a difference, the pubertal stage of the child. And if you want to say, "Well I'm going to tell everybody about a value of 20 micro units per ml. Or 30 micro units per ml. That they have high insulin.

OK I mean you could tell people that. I don't think anybody really knows what that means. You could probably look at an overweight child on physical inspection if they have particularly a high waist circumference, if they have acanthosis, if they have a particular distribution indicating visceral adiposity [sp] you could probably tell them they have a high insulin level or that they're at risk for metabolic disturbances as a result of being overweight. I don't know what the insulin value itself is going to do. I don't know if you can actually follow and insulin value itself in the clinical arena and tell somebody they either got better or worse. So I don't know what you do with that.

I guess the biggest fear is that you are going to take that value and now put that child on Metformin. We don't really have any good clinical data that Metformin alone is going to make any difference. And that Metformin has got efficacy or safety in that child. So I would hope that we're not out there indiscriminately using Metformin to treat these children. These children need to be counseled, they need to be evaluated for diabetes or pre-diabetes. Again, that's done with glucose values. I'm not even sure what we're going to do with the kind with pre-diabetes to be sure. But the kind with diabetes needs to be found just getting insulin values on overweight children, I just don't think that we have any concept what to convey to that family. And we certainly don't want to start treating all these children with Metformin. You want to add anything, Mike?

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Michael Goran

No think that's pretty good.

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Operator

We do have a question coming from Cynthia Wint from Charlotte County Health. Please state your question. Cynthia, your line is live.

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Cynthia Wint - Charlotte County Health

Can you hear me?





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Michael Goran

Yes.

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Cynthia Wint - Charlotte County Health

Yes you can hear me OK. I wanted to know whether the children with the acanthosis nigricans [sp] whether it's found in children with type II and type I diabetes? And could you please kind of elaborate on the acanthosis nigricans because I think I understand it clearly but I'm not sure whether I do and I just wanted a little more elaboration on the-- how it's presented.

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Michael Goran

Fran?

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Francine Kaufman

OK, well you know, you find it on inspection. So you mainly look at the neck as the place to start.

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Cynthia Wint - Charlotte County Health

Is that the only area?

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Francine Kaufman

No, no you can look at under the arms and mainly in the flexural areas of the skin so the armpits. Some people will have it-- you know if they've got a lot of rolls of adiposity [sp] they may even have it between these rolls. A lot of girls will have it under the breast tissue. And it's darkened, it's thickened. So sometimes there's even little skin tags. And what this is indicating is insulin resistance. And we're seeing it in a lot of kids. Some of the kids-- I mean we've been sent kids, you know some of them have eczema, some of them have other kind of skin issues. We

picked up actually somebody with Cushing's Disease with it, where high ACTH changes the skin color as well. So not all of this thickening equals acanthosis nigricans. It just shows you that that patient has high insulin levels and insulin resistance. So you'll see it in the overweight insulin resistant child, you'll see it in a child with type II diabetes. If you don't see it, it doesn't mean that that child doesn't have type II diabetes. We theoretically would not see a child with type I diabetes because they're insulin deficient. So they're not going to really show that. We are-- I mean and this is kind of complicated. We've seen it in a couple of kids who were originally diagnosed with type I and then they're Latino kids, they've been treated for a long time. They go through puberty, gain a lot of weight, start to show evidence of insulin resistance and maybe an element of type II and do these kids really have double diabetes or what do they have. So it's not absolutely excluded in type I but it's pretty rare. It's pretty rare, it's certainly rare at onset. It would have to be way later. So it's really a sign of type II or insulin resistance before somebody develops type II. And sometime it fades when the kid loses a lot of weight and insulin resistance improves.

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Cynthia Wint - Charlotte County Health

Oh thank you.

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Tisha Keller

We have a second question that was e-mailed in prior to the conference. I believe this is coming from Dr. Goran's article. Could you please explain the statement "fat-derived metabolic products may contribute to insulin resistance"?

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Michael Goran

Yes, I refer to that in my talk a little bit when I talked about the theories relating fat to increased health risk. And so this talks about fat as an endocrine organ that releases hormones and signaling molecules. One of those -- at least several of those probably influence





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intermediary metabolism in some say. Adiponectin [sp] is one, Resistin [sp] is another, TGF-beta [sp] is another. Different mechanisms that contribute or cause insulin resistance on other tissues. Does this answer the question?

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Tisha Keller

Yes, thank you.

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Operator

Our next question comes from Barbara Stillwater from State of Alaska. Please state your question.

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Dr. Roger Golub

This is actually Dr. Roger Golub [sp] meeting with Barbara Stillwater. I was really curious about the-- following up on the issue of screening. And a lot of places, including the American Academy, is advocating screening of kids with overweight and additional risk factors which I am involved with in the clinical setting that I work in. But I'm really concerned a number of things, including what do we do with it, which was addressed briefly. But it also seems that some of the kids who are no hyperinsulinemic [sp] may actually be at higher risk because they lack compensatory mechanisms. So I'm concerned, are we possibly doing not just a no good, but a disservice by using those screening programs to help us think that we're actually addressing the problem at all?

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Francine Kaufman

I'd love to answer that. I agree with you. I mean I think, first of all, those recommendations were made in 2002 by an expert panel. And what the American Diabetes Association does is they don't have data and they feel that we need to come up with some consensus, I mean where are we going with this, what do the experts feel? They get a panel, a room filled

with experts and they don't let them out until they come to some kind of consensus. So we, having been president of the ADA when this was getting conceived, or president-elect, we had no idea. And people were asking us for a statement so we got a bunch of experts together and their best guess was, well we don't know if there's a lot of kids out there with type II that we're missing, like we think there are with type II who are adults. And it would be tremendous disservice to have them languish. So I think, not knowing, that was the recommendation. Now a lot of us have been out there screening and there really isn't much diabetes found. I mean the most you find, even in a very overweight population at high risk is maybe 2 percent of the kids might have type II diabetes. So don't know if we should really be spending a lot of time screening kids for type II diabetes. What we should be spending a lot of time with is looking at overweight children with risk factors and trying to get them some kind of better environment and into maybe some lifestyle counseling programs and developing lifestyle programs rather than just trying to get a whole bunch of risk factors together and start screening them and when nothing happens, we drop the ball. So I would rather-- and I don't even know if we need to do a lot of insulin values on these kids or a lot of even blood sugar values. I mean there's enough. If you're in research, we still need to gather data but in the real clinical arena, if you see a kid and you do an exam and they're asymptomatic from a diabetes standpoint: their blood pressure is OK and you might want to get a fasting glucose and a fasting lipids, those are OK. We need to get that kind in the lifestyle program. The lipids aren't OK. I mean we need to evaluate whether or not at some point this kids going to meet criteria for management of dyslipidemia. If the fasting blood sugar is not OK, do we want to do oral glucose tolerance tests on all of them and find IGT? What would we do different if we found somebody with IGT, other than try to get them to lose weight. Maybe it would be a more powerful message. I don't know. And this is what we have to still sort out but I don't think we're going to find a lot of kids with diabetes and I think we need to really focus on what we can do from a lifestyle standpoint and lifestyle counseling rather than waste resources on screening a whole bunch of kids, going out into all these school and screening kids when we're not going to find them?



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Michael Goran

Can I add something to that because I partially agree with that but I think on the one hand you are saying to screen and on the other hand you're not. Because I think the issue sending a powerful message of identifying a risk factor is a very important issue and one that, I mean you know, in adults is very common to have frequent screenings for some of these issues that you mentioned like blood pressure and lipids.

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Francine Kaufman

Well let me add, Michael, maybe it didn't articulate it well. I don't think community screening gets us very far because then you end up with a kind that may have a high lipid level and whatever you're going to do and there's really no follow-up. I mean you tell them to go to a doctor and they don't have one. Screening inside the health care environment makes sense to me at the time of a health care visit, just like we're recommending for adults. I mean the ADA has not advocated opening up malls to have screening things because nothing happens after you tell somebody, "Boy, your blood sugar is high." And then of course a lot of those screenings are done with finger sticks that aren't very reliable. And I would say, sure if you want to send high risk kids by weight, ethnicity into the health care setting, have the health care provider look at the other risk factors and maybe screen them not just for diabetes but for lipids, blood pressure and diabetes, and use some of that to help counsel, that would be a better idea. What I think we should do out in the environment is start to make our message heard out in the environment about environmental lifestyle change.

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Dr. Roger Golub

I think that's an important distinction so I think I fully agree with that. so you're saying, "Not yet ready for a large scale community screening but certainly we need to be doing a better job within the health care system that's screening for some of these things.

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Francine Kaufman

And dealing within the health care system or dealing with it somewhere. I mean I still see a lot of kids screened and their pediatrician told this and not given any-- OK now what do it do or just given a drug. I mean here, take Metformin. We have no idea what's going to happen with these kids on Metformin.

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Michael Goran

Does this answer [inaudible] from Alaska?

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Dr. Roger Golub

Quite a bit. it actually answers my question: is everybody else as confused as I am because we're doing this kind of clinical screening with risk factors and I think we're finding and everyone else is finding that the interventions are totally ineffective. And I'm worried about it also being counter-productive in the absence of effective public health and community interventions that may be getting slighted at the same time.

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Michael Goran

That's probably because we don't-- we know a lot about interventions for reducing body weight, even though they might not be that effective. But we don't really know too much about interventions that might address those secondary risk factors.

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Dr. Roger Golub

Well anyway, your discussion is very helpful, thank you.





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Michael Goran

You're welcome.

itself is just really hard to follow. If somebody's got an abnormal glucose, you should follow the abnormal glucose level, particularly be sure it's not progressing to diabetes.

Tisha Keller

We have another question that was e-mailed. This one comes from Albany, New York. It says, "I have a pre-pubescent Caucasian [unintelligible] boy who has some impaired glucose tolerance, dyslipidemia and CRP 9.0. I'm working with him on lifestyle changes. What means of assessing progress should we be using? Apparently we are looking at weight changes and changes in eating and exercise habits. His mom asked me about rechecking his fasting insulin level, or is that useless, given that he will be entering puberty? At what point would we recheck if that is a reasonable thing to do?"

Michael Goran

Yeah I would agree. I would say follow the glucose but I think the other issue is the intervention that is being applied, I would say take the focus off body weight, take the focus off the eating plan, otherwise we're going to lead to some risk of eating disorders. Find some very simple dietary exchanges that are healthy to the diet, find some simple activities that this child enjoyed and can succeed at and can do readily. And keep track of the glucose periodically more than the body weight. The idea is -- forget what age you said this child was, but presumably they're still growing. So the idea is to have them grow into their ideal body weight so that weight loss itself may not be that important. And more importantly, to make sure it doesn't progress to diabetes.

Francine Kaufman

It sounded like she said that the child had IGT, is that true?

Tisha Keller

But since this child is pre-pubescent, would you recommend testing his glucose every three months or what would you recommend or recheck after he's already finished puberty?

Tisha Keller

Yes.

Francine Kaufman-

OK well I mean if a child has IGT, so you've done a two-hour oral glucose tolerance test, I presume, or has an impaired fasting glucose. I mean you might want to follow just glucose levels. I'm not sure I agree that I don't know what any of us are doing with all these insulin levels. I mean is 30 worse than 40? I mean there's so many-- because 30 maybe actually means that now the beta cells are really dysfunctional. I just think insulin levels are tough in the clinical setting to know what to do with. They're very good if you want to look at large populations and did we shift means and things like that or cutoff points. But in the real clinical arena, an insulin level

Francine Kaufman

Oh I don't think you need to recheck. You certainly don't need to do an ODTT every three months. And a fasting-- you know it just depends on what those values were I uses initially for them to say that this child had IGT. I mean the presumption is they were doing two hours. I mean I would say maybe at the end of a year of working with the child and the family. I mean obviously this is kind of a kid inside a whole family structure that got the kind there I the first place, you kind of have to deal with the whole-- I know that's what Michael is talking bout, the whole family structure and what they choose for food and





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activities and things like that. But I would say at least probably six months or a year.

Michael Goran

Yes.

Tisha Keller

OK thank you.

Operator

Our next question comes from Anne Faith from Hernando County Health. Please state your question. Anne your line is live.

Anne Faith - Hernando County Health

Thank you, I'm sorry. We were just wondering-- actually someone already answered the question about the -- I can never pronounce this, the mark around the neck.

Francine Kaufman

[unintelligible]

Anne Faith - Hernando County Health

Yes, what causes that but that question has already been answered. So thank you very much. This was very, very good.

Michael Goran

You're welcome.



Operator

Our next question comes from Carol Bullock from Medical Nutrition Therapy. Please state your question.

Carol Bullock - Medical Nutrition Therapy

I was just wanted to know that you all are looking for intervention. Of course me being a dietician, many insurance companies are not reimbursing for nutrition education. Medicaid just recently approved for diabetes self-management but I don't know if you all are kind of pushing towards the Congress and people that make the-- approve reimbursement for medical nutrition therapy for weight management or any other such as even having a dietician in every county anyway in the school system in the country.

Francine Kaufman

We're pushing. Trust me, we're all pushing. You know there's a lot of issues around the reimbursement, the validity of a diagnosis and all these other things. I think actually we're making some headway in California with some of the managed care, including our entitlement program, the Medicare-- MediCal managed care system of them realizing they pay for it now or they pay for it later. And prevention is still cheaper than treatment. So they are allowing some referrals to lifestyle programs for kids.

Carol Bullock - Medical Nutrition Therapy

Yeah I appreciate that certainly they have done some intervention with seniors but yet the children in the school system, etc., I know that we here in Florida have tried to promote medical nutrition therapy or nutrition intervention, even teaching the teachers that are teaching some nutrition in [unintelligible] education classes because there is so much



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misinformation out there that again, having a dietician or nutrition expert, if you will, that has the qualifications to guide people in terms of making positive food healthy choices not just against diabetes but multiple illnesses as well.

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Francine Kaufman

No, that would be wonderful. And obviously school nurses are critical as well in being able to disseminate information. But I'll be real honest with you. And I hate to suggest it, none of us have any free time, but the real thing is beyond what you could do in that professional role to also think of what you could do as a volunteer. And I've volunteered on our local school issues. My kids are all out of school and done but it carries so much weight when you go and you talk about using candy as rewards by the teachers and all these machines and why are we having another bake sale. And you could start to set some standards with your local school. I mean this all eventually has to get done locally as well. And we passed-- I love going up to Sacramento, all the laws that have been passed around school. I mean none of them get done. There's books filled with laws and regulations and mandates and you go to the local school and they don't even turn off the vending machines and lunch time and that's absolutely federal mandate and a state mandate. So no matter what we do, we still have to focus on what does really mean. And I would tell you, I don't know where everybody works who's out there. Look where you work too. I mean my hospital-- we run an obesity program here, you walk in my hospital, we got a McDonald's, we got a gift shop filled with candy. We got a cafeteria, the new food they're now serving is fried chicken, a new fried chicken stand. You can't find the stairwell. We need to kind of walk the walk ourselves and clean up our own environments if we want people that enter our healthcare systems and think we're serious about this.

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Carol Bullock - Medical Nutrition Therapy

I can appreciate that very much. Thank you.

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Operator

Our next question comes from Susie Gibbons from Children's Medical Services. Please state your question.

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Susie Gibbons - Children's Medical Services

Hello? I was wondering if you could answer two questions actually for me. I was interested in knowing what the percentage of children are that take Medformin and have nausea and vomiting to the point where they go to the emergency room from it in tolerating it. And also in your articles you had mentioned the Tanner Stage and its effect on insulin resistance in children. And I was curious if you could highlight those two areas for me.

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Francine Kaufman

I tell you what, I'll deal with the Medformin, Mike you can have the Tanner Stage.

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Michael Goran

Good idea.

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Francine Kaufman

OK. It's probably about 10 to 15 percent of children who seem to have real GI disturbance from Medformin. Whether they end up in an emergency room, I mean I don't know if I can answer you that. It's not usually vomiting it's usually diarrhea and abdominal distress. And they just won't tolerate it. You go back down on the dose a little bit, they get a little bit better. It's not an efficacious dose so you have to go back up again and they get those symptoms and you just have to change medication. But if you had a child really vomiting, I wouldn't think it's the Medformin, I'd think it's something else. And you know that when there's somebody who's got a current illness or going to undergo





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surgery or gets dehydrated, Medformin becomes a little bit of a risk, so you have to take somebody off those drugs when they're sick like that. And there have been some isolated deaths around-- with kids on Metformin. You know, is it due to Metformin, some other things, it becomes unclear. But Medformin isn't like water. This stuff has side-effects.

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Susie Gibbons - Children's Medical Services

OK thank you very much.

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Michael Goran

And with regards to the maturation phases, at the midpoint of puberty children become naturally more insulin resistant, not sure why. Probably some mechanism that promoted growth. However they [unintelligible] resistance as I mentioned, does challenge the data cell. And the more insulin resistant you are to begin with, the challenge to the data cell becomes exponential. That's why puberty and the insulin resistance is probably becoming more impactful because the starting point of insulin resistance has become more severe, especially in those minority children. Typically, the normal pattern is by the end of maturation, levels of insulin resistance return to where they were beforehand. We're beginning to see -- actually we haven't published this yet but we're beginning to see that this in fact may be more blunt edge or may not be as noticeable for very overweight children who are already extremely insulin resistant to begin with. Does that answer your question?

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Susie Gibbons - Children's Medical Services

Yes that does. Thank you so much. I've enjoyed this program.

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Francine Kaufman

You know puberty is a time of resistance, that's all I can tell you.

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Susie Gibbons - Children's Medical Services

OK I'll remember that.

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Tisha Keller

Operator, I think we need to wrap up the call. I want to remind nurses, dieticians and certified health educator specialists who would like CEU credit for this program, you need to mail or fax all completed forms and rosters to me by June 28th. The fax number again is 850-245-4391. I want to thank Dr. Goran and Dr. Kaufman. We appreciate so much you giving your time and expertise. These insights will be very useful for improving diabetes care nationwide.

We are now going to have a short time for your questions regarding the CEU forms if you need clarification.

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Operator

Thank you. We will now take your questions about continuing education credits, CEU's now. If you are using a speaker phone, please pick up the handset before pressing any numbers. Should you have a question, please press star 1 on your pushbutton telephone. If you wish to withdraw your question, please press star 2. Our first question comes from Jan Waters from Diabetes Health Management. Please state your question.

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Jan Waters - Diabetes Health Management

Could you just clarify the fax number?

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Tisha Keller





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Yes, 850-245-4391

Jan Waters - Diabetes Health Management

And is there a post test or is it just the survey?

Tisha Keller

There is a post test. It is on our web site and you can download it from there.

Jan Waters - Diabetes Health Management

And what's the web site?

Tisha Keller

[www.doh.state.fl.us](http://www.doh.state.fl.us). When you get to that home page, choose Diabetes Prevention and Control from the drop-down menu at the top. And that will take you to our home page and you can follow the links to the teleconference page that way.

Jan Waters - Diabetes Health Management

Can you repeat that? it's [www.doh.state.fl.us](http://www.doh.state.fl.us). And under diabetes prevention and control does it have post test?

Tisha Keller

It's under the audio teleconference. It'll be the same place that you registered for the teleconference. That same page, there's a new box on it with the post test and that kind of thing.

Jan Waters - Diabetes Health Management

OK thank you.

Operator

Our next question comes from Bobby Long with Caro Mont Diabetes Center. Please state your question.

Bobby Long - Caro Mont Diabetes Center

Yes I just want to clarify which items you need RD's and the CDE's to send in for continuing education units. I'm looking at your web site under Materials for CEU credits, I assume CDR portfolio, the RD survey. Is there anything else that we need to send in?

Tisha Keller

It depends on if your dieticians are on Portfolio or not.

Bobby Long - Caro Mont Diabetes Center

We are.

Tisha Keller

OK if you are, then you need to download the from that is on the portfolio, you press the Diabetes Prevention and Control logo on the top. That's the from you need to fill out. You also need to do the post test and the two-page impact survey. We do not have CEU for CDE's at this time. The first CHES, certified health educators we do but for this call we don't have CDE. So if they're duly certified they can get CHES credit.





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Bobby Long - Caro Mont Diabetes Center

Alright thank you so much.

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Operator

Our next question comes from Nymara Devitis from Blue Cross. Please state your question.

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Nymara Devitis - Blue Cross

Oh hello. I was just wondering which form would-- I'm a registered nurse. Which from do I use for the CEU because I really didn't see anything that pertained to that.

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Tisha Keller

OK you're going to use the Florida AHEG [sp] Network impact survey. That's the registration that effectively registers your for CEU credit. Also the post test and the two-page impact survey and registration form.

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Nymara Devitis - Blue Cross

The two-page impact survey and registration form.

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Tisha Keller

And the post test, yes.

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Nymara Devitis - Blue Cross

And the post test, ok great. Thank you very much.

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Operator

Our next question comes from Scarlette Hutchison from University of Alaska, please state your question.

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Scarlette Hutchison - University of Alaska

Yes I was just wondering as far as getting the CEU credits for out of state, do I still fax in the information to you or do I just apply directly to my board?

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Tisha Keller

You will need to fax it in and then our CEU provider will issue you a CEU certificate and once you get that in your hand, they'll mail it to you, then you apply to your board for that credit.

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Scarlette Hutchison from University of Alaska

Thank you very much.

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Operator

Our next question comes from Margaret Ward from St. Lucy County Health Department. Please state your question.

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Margaret Ward - St. Lucy County Health Department

I am at the web site for the teleconference and I do not see a link anywhere for a post test. If you're familiar with the web site, can you direct me to the post test on here?

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Tisha Keller





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Margaret, I don't have it up in front of me but I can e-mail that to you.

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Margaret Ward - St. Lucy County Health Department  
OK.

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Tisha Keller

It would be Margaret Ward?

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Margaret Ward - St. Lucy County Health Department  
Yes at doh.state.fl.us.

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Tisha Keller

OK I will e-mail that to you and if anyone else needs it, just shoot me an e-mail. I'm the one that sent you your registration confirmation and I can send you the e-mail, the post test that way.

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Margaret Ward - St. Lucy County Health Department  
Thank you very much.

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Operator

Our next question comes from Topaz Wallace from Healthcare. Please state your question.

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Topaz Wallace - Healthcare

I just was looking for the post test as well.

Tisha Keller

OK if you'll just send me an e-mail, I will send it to you. It was on the e-mail that had the call-in number on it. It was attached to that e-mail. Look for it there and if you can't find it and you can't find it on the web site then send me an e-mail and I'll e-mail it back to you.

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Topaz Wallace - Healthcare

Thanks.

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Operator

Once again, ladies and gentlemen, as a reminder, should you have a question please press star, one at this time. If there are no further questions I will now turn the conference back to Tisha Keller.

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Tisha Keller

Thank you. I want to thank everyone for participating in today's call. I once again want to thank Dr. Goran and Dr. Kaufman. And everyone have a good day. I'll turn the call back over to the operator for a conclusion. Thank you.

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Operator

Tisha. They actually disconnected.

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Tisha Keller

Oh, OK thank you.

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Operator

This concludes our conference for today. Thank you





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all for participating and have a nice day. All parties  
may now disconnect.

END

