DANNY LENNON: So here we are Prof. Leanne Redman thank you so much for joining me on the podcast.

LEANNE REDMAN: I'm so excited. Let's do this.

DANNY LENNON: Yeah I've got a lot that I wanted to ask you about and you've published in a number of different areas that I find fascinating and I suppose one of these this was intersections of a Venn diagram of some of that work is talking about this intergenerational transmission of obesity, how that correlates with maternal diets and so on but I just thought maybe to introduce people to the topic before we get into any specifics can you maybe explain that term intergenerational transmission of obesity and then how that relates to your wider work and focus?

LEANNE REDMAN: Yeah. So this is is a really I love this area of study because you know the big idea here is that obesity has really early origins in the life of a person and that in fact we inherit our some kind of risk for obesity and other chronic diseases in utero and we inherit actually more risk from genetic origins which of course would be from our parents. So it's this idea right that we obtain disease risk from our two parents and even as far back as our grandparents now we know that they have some level of responsibility in this cascade as well but layered on top of that are the exposures during pregnancy as well which it brings up the

maternal diet that you just mentioned invoking even a greater risk for people from the time that they're born. So it's interesting right because if you think about the the classic obesity maps that we often see and these are like heat maps where you'll see the map of a country, so I'm in the United States I will look at the map of the United States and over the course of time it shows using a different intensity of colors that there's an increase in the amount of obesity that's reported on a state-by-state basis over time and we know that some of that is due obviously in part to changes to the environment in which we live. We all have enjoyed modern technology and the advancements that has brought to our lifestyle making things being able to go much more quickly and more efficiently but what that technologies have also done has led to more processed foods which I know you've had other speakers here talking about that before but it's led to more processed foods and it's also led to a workforce that's less physically active. And so therefore more sedentary lifestyles. So we know that part of the obesity epidemic is due to our physical environment but it can't explain it in entirely. And so then investigators have spent quite a lot of time looking at obesity transmission in rodent models and in other animal models where they will feed a male animal or a female animal a high-fat diet prior to conception to induce obesity but then they may sort of control those diets throughout pregnancy and just by having obesity prior to conception in a maternal species will increase the risk of that offspring being born with excess body fat and then if you follow the weight change of those offspring throughout the course of their life they are more likely to develop obesity as adults and the associated co-morbidities.

DANNY LENNON: Fantastic I think that really sets the stage for much of what I wanted to get into and of course obesity is a huge part of that but one of the things that I found really fascinating from reading some of your published work was this discussion around we know that the maternal obesity increases risk for some of these adverse outcomes that you mentioned but there is a large degree of variance it seems that it's not a 100% susceptibility. And in fact there are people that would be classified as living with obesity that then we don't necessarily seem the same adverse outcomes as

in other people and so some of what you published had mentioned that this variance in the impact on say offspring adiposity in the future is not necessarily just completely down to obesity per se but maybe that prolonged exposure to things like glucose or triglycerides in the bloodstream. Can you maybe just mention some of that and how that fits into this this piece?

LEANNE REDMAN: Yeah obesity is fascinating because what we're talking about is somebody that has a higher body size than somebody else or that has higher adiposity or fat mass but when you think about that from a practical standpoint what is it in our weight that could signal genes or proteins or functions within cells to invoke some kind of disease risk like there is really no nothing in weight itself like weight as a measure, mass that can do that. So that's why we have to look at the types of of mass. So that's where we get to fat mass but then even more granular than that we we start looking at things within in circulation. So we know that two people can have the same BMI and I'm sure you've had people on here to debate that before where you might have two people that are both overweight or both have obesity but one person achieves obesity by having 50% body fat and the other person might achieve obesity by only having 35% body fat because BMI is just a representation of weight to height. So we have to look beyond obesity. So we know that this we call it the maternal metabolic milieu and you can think about that as all of the different factors in the maternal circulation that are present during intrauterine development as being precursors for developing adipose tissue for the fetus during gestation but then that also infers the amount of adiposity that the baby would have at birth. So there are two primary substrates in the maternal diet and within the maternal milieu that we know are very important for developing adipose tissue in utero and those substrates are glucose and also triglycerides. There are others. We know leptin is very important and of course there are growth factors like your IGF proteins for instance and obviously growth hormone. There are other hormones that are equally responsible for fetal growth but these two maternal substrates glucose and triglycerides are very important. Think about the example of gestational diabetes. So

gestational diabetes is where the mother develops diabetes in pregnancy and she's never had diabetes before. So all women have insulin resistance in pregnancy. Some women are unable to control the insulin resistance so they develop diabetes. These women have the highest circulating glucose during pregnancy of all women in pregnancy and those babies are well known to be born larger than babies that were not exposed to the high maternal glucose as a result of the gestational diabetes in pregnancy. So not all maternal obesity "is equal" and so you know as we are starting to embark on this really exciting field of precision nutrition and precision prevention and precision treatment or medicine of disease. We too are starting to think about precision nutrition and precision prevention within the context of pregnancy and one of the things that we're focused on here within maternal obesity is trying to understand these other metabolic risk factors for obesity risk in offspring. So they are these maternal growth factors, so glucose and triglycerides we also know that blood pressure or hypertension in the mother is also important but on the other end of the scale in lower adiposity for the babies but we're thinking about these other factors as potential determinants, if you will, for greater obesity risk in babies. So obesity is not one size fits all and we want to make sure that we can have conversations with mothers and start asking questions of the science that dives much more deeply than just the simple classification of obesity.

DANNY LENNON: Yeah that's fantastic because I think sometimes people can go to either end of that extreme of using that as something well well now BMI no longer matters for example or then go to the fire extreme of if someone has obesity then that is going to be the same risk regardless of all the other markers and as you view eloquently described here that we can use that BMI cutoff and that classification of obesity as a really good way to screen a lot of people who may be at higher risk and then there is probably some degree of refining of that risk through looking at further markers like blood glucose, blood triglycerides, etc. right?

LEANNE REDMAN: Yeah it's really important and we have a really exciting publication that's impressed at the moment and we'll

present it in November at ObesityWeek which is the Obesity Society Meeting and that paper shows that if we are to classify women with obesity on the basis of these other metabolic risk factors. So we use the metabolic syndrome classification which takes into account waist circumference, glucose, total cholesterol, LDL cholesterol, yeah LDL cholesterol and triglycerides and we classify those women into having no risk factors, only obesity but no other risk factors so those women are metabolically healthy, if you will, and then we had women with two or more of these metabolic risk factors in addition to obesity and those are the metabolically unhealthy mothers. What's really interesting about those two groups of women first of all is this that the amount of weight that they gained in pregnancy was identical. The amount of fat mass that the women gained in pregnancy was identical but what differed between the two groups of women were the excursions in these metabolic risk factors across pregnancy. So you've got one group that started out with high levels. They were metabolically unhealthy and they stayed at high levels all throughout their pregnancy whereas the women who were metabolically healthy at the start those levels of glucose, triglycerides, cholesterol; whatever those all increased gradually across the course of pregnancy which is very normal. That is the normal course of what happens in pregnancy. So then you have two different situations of fetal exposure. One group of fetuses which to the mothers with metabolically unhealthy phenotypes those babies are exposed to high levels of these growths promoting substrates for the majority of the pregnancy whereas the other the healthy group it's a gradual exposure throughout the course of gestation. Those two groups of babies differed at birth by not birth weight but the amount of adiposity it was almost double, almost double.

DANNY LENNON: Wow! That's incredible. So it's not necessarily the peak of some of those markers or what someone ends the pregnancy on. It's this cumulative integrative exposure over the whole time course of the pregnancy that seems to matter.

LEANNE REDMAN: Yes. So you think about it like the area under the curve. So we're calling it the area under the pregnancy curve and it's just, it's very simple. In my opinion it's

the length of the exposure. So normally in fetal development the baby it accrues adipose tissue in the latter part of pregnancy. We all know that that's when the mothers start to gain the most weight but if you're starting to promote fetal adiposity much earlier makes sense that by the time that they're born that they're going to have more than their counterpart who had the normal exposure to those substrates strategist station.

DANNY LENNON: Right, yeah. That's fascinating. So if we think about some of those possible adverse outcomes that can occur when we have metabolically unhealthy obesity during the pregnancy one of the areas that that you've published around is preeclampsia and particularly it was a one of the papers had an eye-catching title of obesity complements preeclampsia. So I suppose before exploring that can you maybe just, for people listening who are unaware of, can you explain what preeclampsia is and then what you kind of mean by this obesity complements preeclampsia?

LEANNE REDMAN: Obesity doesn't only pose a risk for the baby. This is an important point. Obesity in pregnancy also poses a risk for the mom and we really touched on the risk for gestational diabetes. Mothers who enter a pregnancy with obesity are more likely to develop gestational diabetes than their counterparts who might have normal weight and the same thing is true for conditions that are associated with hypertension or high blood pressure in pregnancy. So women retain fluid during pregnancy. Fluid retention is just a normal physiological response to pregnancy. The blood volume actually expands by more than one liter. So that's a normal physiological response but in some women they retain more fluid than we can understand and explain and that contributes to an exponential rise in blood pressure and it starts to occur from sort of mid-gestation onwards and this is why blood pressure is a routine measurement at every prenatal visit that a woman has. So preeclampsia is like the worst possible blood pressure condition in pregnancy. It's very dangerous for the mother. So at this point the kidneys aren't functioning properly. You will see proteins spilling over and you can measure it in the urine. So protein urea is a characteristic of women with preeclampsia, very high blood pressure. So

systolic blood pressure greater than 130 is the trigger for more intense monitoring but for mothers who develop preeclampsia their systolic blood pressure can be 160 and sometimes over 200. The only cure for preeclampsia is to deliver the placenta. So we know that there is a placental origin to preeclampsia and we know that it occurs at the time of implantation. So when you study the placenta of these women who give birth to babies following preeclampsia, the placenta often it disintegrates in your hand and it falls apart. We know very early in gestation is a critical period for the angiogenesis in the placenta. So that is the development of all the capillary networks that are in the placenta. So if you think about it like the muscle. So we know that when we exercise a muscle bed we increase the capillary density in that muscle bed so we have better exchange of oxygen and carbon dioxide as well as we can bring fuels there and get waste products out. It's very much the same function in the placenta. So the placenta starts out very small and then as the pregnancy progresses and the baby grows the placenta grows and so does this network of vascularization. So in preeclampsia the vascular pool and the vascular bed is very poor. There is fewer numbers of capillaries, there is tons of blood clots and so nutrient exchange is very poor so often these babies are born small for gestational age as I mentioned earlier like they tend to have less adiposity because of that. So they have a lot of complications with their fetal growth. So it's very hard to study because I just said the origins are around the time of placentation around the time of implantation when the placenta is starting to be developed they are very-very hard to study in in people because we get the placenta at birth but at that time the placenta is like 30, 34 weeks old and so you know kind of the ship has sailed on the genetic origins and the markers that have set the stage for the poor vascularization. So I collaborate with a veterinarian here in Louisiana. Her name is Jennifer Sones and she has a mouse it's the BPH5 mouse the blood pressure high five mouse and this mouse has high blood pressure and after they started to breed these mice they learned that these mice just at random would start to develop hypertension in pregnancy and this preeclamptic phenotype. So this was really exciting because now we have an animal model that which we can collect tissues and study

things at the time of conception and three and four and five days after conception and so we started really focusing on the pool or the depot of adipose tissue that exists around the reproductive organs. So women have a small pool of adipose tissue obviously in their visceral, in visceral fat. So this is the fat that encases our organs in the abdominal cavity. I will say that rodents do have more adipose tissue around their reproductive organs but humans definitely have it in close enough proximity and so we've been studying the inflammatory profile of the adipose tissue as well as what we call the implantation sites in these mice to see if there is factors in the adipose tissue, inflammatory factors such as complement like we talked about at the top of this question and to study those in relation to the preeclampsia in these mice. And so we do know that the mice that have more adipose tissue around the reproductive organs, so a worsened inflammatory milieu. So inflammatory cytokines such as interleukin, interleukin 1, 6 ioBeta as well as TNF alpha for example as n-complement factors, have a worsened blood pressure profile throughout the pregnancy more and more preeclampsia and the pups are always born smaller, there are smaller litter sizes and all those things. So that's the con the obesity complements preeclampsia but it's more getting at the complement factors which are part of the immune system that we know comes when we have this systemic inflammatory state that obesity offers.

DANNY LENNON: Fascinating. Such cool work. So given that we've discussed some of these issues around metabolic unhealthy obesity that there's in that particular scenario that we've got a much higher risk of adverse outcomes and generally when we look at obesity there is going to be this correlation to risk. So when it comes then down to understanding for example weight changes across a pregnancy what we typically see versus what might be advised and so on what or suppose first if we look at the weight gain that we typically see in a pregnancy, how does that typically stack up versus what our current guidelines would be for during the course of a pregnancy if we have average figures like that?

LEANNE REDMAN: Now we're starting to think about how do we prevent some of these things. So obviously we have non-modifiable risk factors. So we just said before that we inherit some of obesity risk from our parents. So some of that we don't know yet how to change. So as a woman who enters pregnancy if she has obesity some of the risk that she's going to pass on to her offspring is going to be the result of her appearance and obviously part of her DNA but what we're starting to think about now what are the modifiable risk factors and so since weight gain and gains in fat mass are associated with adverse outcomes outside of the context of pregnancy. We think that optimizing weight management in pregnancy is as equally as important as outside of pregnancy for lowering someone's risk for metabolic consequences. So that idea is definitely supported in epidemiological studies. So if we were to take a cohort of women entering pregnancy, doesn't matter what the country is, whether it's the U.S., Australia, Ireland any other country in Europe and we would just take this cohort, simply measure their BMI at the time of conception and a whole host of maternal and fetal outcomes at the end of pregnancy we would see in those studies that there is an increased risk for maternal adverse outcomes. We just said a couple gestational diabetes, preeclampsia, pre-term birth, non-elective cesarean section, large gestational age; those maternal risk factors are higher in the mothers with maternal obesity and of course we have similar adverse outcomes in the babies from those moms too. So those babies are more likely large blah blah we just said that. So then you think okay well the obvious thing then is to start focusing on weight and weight management. So there are international I'm putting quotation marks here around my my fingers so there's international recommendations now on the amount of weight that is considered optimal for a woman to gain during her pregnancy and this is not that new I mean the first was in 1990 and they were revised in 2009 and the revision in 2009 accounted for the different BMI classifications now. So as you might expect the amount of weight that a woman can gain is more liberal if she enters pregnancy as normal weight and they're more restrictive as she enters pregnancy as a heavier person. So for a woman who is normal weight the recommendation for her is 25 to 35 pounds for the pregnancy. Then it goes down from there. If she's

overweight it's 15 to 25 pounds of weight gain in the pregnancy and then if she has obesity the weight gain recommendation is only 11 to 20 pounds. That's pretty restrictive. So if you think about that 11 to 20 pounds if the baby weighs 8 pounds on average which is likely then it only leaves three pounds for the expansion of fluid, we talked about that being a normal physiological response not to mention the placenta, not to increase the natural size in the mammary glands or the breast tissue. So it doesn't really leave any weight gain at all for mum. So we have these weight gain recommendations and the hypothesis is that if we can attenuate the amount of weight that a mother gains during pregnancy we alleviate some of the burden on these maternal and fetal outcomes. In particular we can reduce the burden on offspring obesity risk. So a large proportion of the work that my lab has been undertaking for the past decade has been trying to design different programs to optimize weight gain for mothers in pregnancy and to also have to see downstream benefits on these maternal and fetal outcomes. So in the general uh population two-thirds of women exceed these weight gain guidelines if you don't intervene at all. And it doesn't matter which BMI group that you fall into. So we have to ask ourselves well why is that and part of it, it has a two-pronged or multiple-pronged reason I think. The first is that it's really difficult to talk about weight still in general and it's increasingly difficult to talk about weight during the context of obstetrics. So in some countries the UK is one women don't get weighed at their visit. Women don't want to be weighed at their visits and women don't want to talk, have been talked to about their weight either at their visits. So there's this whole thing about weight and obesity stigma and those sorts of things. That makes it very difficult. The other is that we have a lot of cultural norms around weight gain in pregnancy. So often you can think when someone becomes pregnant and you see them eating something that they've got to eat a little bit extra because now they're feeding somebody else and so that's the poster that I have behind me which says change the culture, eat for you not for two because it's this idea that now you're pregnant you have this license to eat for two people and so we have stigma challenges around just talking about weight how to counsel somebody on the weight management and

then how to get over these cultural norms is that it's okay to eat for two. So we've got our work cut out for us here and we can talk if you want uh about how those studies have fared up into this point and what we're thinking now.

DANNY LENNON: Yeah think it's so interesting particularly when you mention this cultural point because I think a lot of people when they first hear about those typical recommended rates of weight gain and especially if they translate that into what type of caloric excess that might end up being over the norm, it's much smaller than they would have perceived and again that's probably for cultural reasons. There is probably other things going on I guess within the course of a pregnancy where there is obviously a lot of stress. There is things like cravings, etc. that may change the dynamics practically about what someone could do but if we were talking purely physiologically is there any basis for and I can't remember where I would have heard this kind of discuss but in a case where you have someone with an extremely high BMI for example. Is there any physiological reason why they couldn't aim for fat mass loss through the course of a pregnancy or does that have contraindications? Do we have any data that might shed any light on that particular question?

LEANNE REDMAN: Yeah. It's fascinating right and it's so important to start thinking more specifically not just about weight like we talked about before but what is it about weight that we're actually trying to target and since most of our metabolic risk factors come from adipose tissue or fat, you think that that's really what these interventions and messages should be focused on but they're not quite there yet in terms of a widespread narrative and conversation but it's definitely the way that my lab has pivoted and what we're thinking about now. So we did some great observational cohort study and this was funded by the NIH and it was it's been to this point the only one of its kind and so I didn't want to intervene. I wanted to just understand the physiology of weight gain in pregnant mothers with obesity with no intervention. So I got 75 women. We enrolled them as early as we could which was around 10 weeks practically. You find out someone's pregnant around six seven weeks then they got to go for a

doctor's visit and we got them to the research center and we were able to perform very-very cool nutritional and metabolic phenotyping on those mothers. So we did doubly labeled water studies on them to understand their energy requirements. They spent the night in the metabolic chamber to assess their energy expenditure. We made the best measures that we could of their body composition changes across pregnancy and we tried to understand the physiology between mothers that had excess weight gain, the recommended weight gain, and then the mothers who according to these guidelines didn't gain enough weight. And it's fascinating. So in a population where there's no intervention the heaviest women they gain the least amount of weight on their own and so I'm talking about a woman with morbid obesity. So maybe with a BMI above 50 and so this is a woman that would weigh about 150 kilograms, 150. So it makes sense from an evolutionary standpoint if you think about it because the normal weight women have to gain more per the guidelines compared to the women with obesity and now within the context of obesity we've got women that gain some and women that gain none at all and in fact we have women that lose. So why is that and I think there's some sensing in the body fat as an energy source for the pregnancy. The body knows that this woman doesn't need to accrue any more energy stored as fat to be able to contribute to normal fetal growth for the child. And so it started to make us think well if they've got enough adipose tissue to be able to dedicate to fetal growth then why aren't we designing interventions that are much more restrictive in terms of food intake to try to promote fat mass loss in pregnancy because if we're not targeting the adipose tissue which is what's responsible for this pro-inflammatory milieu of preeclampsia, the pro-metabolic milieu of the growth promoting factors for the baby then we're not going to move the needle on obesity risk in the offspring. And so that's our current thinking and we have a brand new trial now NIH funded and it's a small trial and that's intentional because it's a feeding trial and so we are studying 100 women, 50 will receive the feeding trial, 50 will be in the control group. These are women with grade 2 and grade 3 obesity. So that's a BMI of 35 to 50. We are going to prescribe or we are prescribing diets that only provide 75% of their caloric needs. So it's a 25%

energy deficit from the beginning of the second trimester. So around 13 weeks throughout the course of their pregnancy. And so we hypothesize that this degree of energy restriction will mobilize the mother's fat mass during pregnancy. The fetus will grow normally but by the time she delivers that eight pound fetus as well as the placenta and loses some of the fluid that she's retained throughout the course of her pregnancy she will on average be five kilograms lighter than when she enrolled in the program at about 13 weeks pregnancy. So those kinds of studies are moving the needle. So it's pretty innovative in thinking it's not what necessarily people feel comfortable with. People think it's really important. So we're doing it with this controlled feeding paradigm to ensure that the mothers are getting a healthy diet while they're being calorie restricted. We're monitoring the fetal growth every four weeks to make sure that there are no problems and so I'll have to come back in about three years and let you know what we find. We've got eight women in the trial right now.

DANNY LENNON: That's so awesome and again like you say it's really getting at the core of these questions that oftentimes are probably not explored because it is uncomfortable to some degree to even talk about caloric restriction during a pregnancy but as you've outlined particularly if we think of those cases where BMI is extremely high then there is more than enough substrate round to fuel that fetal growth but also the changes that we typically see in things like blood glucose, triglycerides; that accompanies significant fat mass loss is clear and so yeah it seems like that should be good ground to see does this intervention have impacts on the offspring.

LEANNE REDMAN: Yeah and you hear all the time pregnancy often described as a teachable moment. It's a time in a person's life when they're more likely to make behavioral changes than any other time like for example prenatal vitamins are recommended for people during pregnancy and they get prescribed to them and they adhere to them really well whereas other times in their life they might take a multivitamin or whatever here and there but they don't stick to it at all. So we're trying to also capitalize

on the moment and thinking that the person now who has obesity and that is pregnant unless we try to teach them the skills and the tools that they need to optimize their health long term if we miss that moment now then we may miss the moment not only for that person for the rest of their life but also for the newborn child.

DANNY LENNON: You mentioned the timing of it was from week 13 on I believe you said. Can you maybe just explain the thinking behind that methodology?

LEANNE REDMAN: Yeah so part of it is a practical standpoint. So when we start thinking about precision prevention in pregnancy we really don't have an opportunity to intervene until we know the person's pregnant, until we can have an ultrasound which usually occurs around 10, 11 weeks to confirm that everything's okay with the pregnancy and then you go from there. That's sort of when prenatal care starts. So whenever we want to intervene in pregnancy we've got to start as early to that time point as possible. So then from a weight management standpoint. We approach weight management interventions for about a six month period of time 24 weeks- sh so then that allows us to get in sort of a normal weight management program throughout the course of the gestation. So it works out perfectly from that standpoint. But any of the the listeners who are interested in this topic or know something about this topic as well are going to probably read my mind for the next sentence and that is we're starting to think that we're not intervening early enough. There is evidence to show that some of the epigenetic programming that occurs in the organs and tissues of the fetus is as a result of programming to the gametes themselves, to the ova and to the spermatozoa itself and of course that is way before 13 weeks of pregnancy. So there is also several groups now starting to think about how to intervene in couples prior to pregnancy with these similar approaches whether it be weight management, smoking cessation, whatever the behavior change may be because they're starting to think that the vulnerable period for fetal development. Obviously it's the course of gestation but it's also very early at the time of conception.

DANNY LENNON: These are such interesting questions to think about and I could keep you here for hours talking about this subject but with time in mind before I did wrap up I wanted to talk with you a bit about the obesity campaign from you guys at Pennington which you sent me across details of and it was, it was pretty impressive to see. It was so novel. I hadn't seen something like it. Can you maybe introduce that to people about exactly what that is?

LEANNE REDMAN: Yes. So I work at Pennington Biomedical Research Center and I know you've had Eric Ravussin on here before and maybe some others from here. We are a nutrition institute dedicated to not only nutrition research but I would say primarily to the study of obesity and its comorbidities and we think that it's extremely an urgent topic of modern times and that it's hard to speak about a pandemic now as we are all facing and dealing with the pandemic of COVID but right before the covert pandemic we were, the World Obesity Federation was about to come out and describe obesity as a pandemic and so it it needs much more attention. It's not getting the attention it deserves and so we decided to establish a campaign and it's an awareness campaign to bring attention to obesity as a disease and to raise awareness to consumers, to people, to consumers, to clinicians, to funders and people basically worldwide. So it was a two year or more long process and it's interesting because we engaged the consultation of very large PR and marketing firms who typically work with food companies to sell bad food and I don't want to throw out any brands but you could probably pick a couple of fast food chains if you wanted or soda brands if you wanted. The top ones that come to mind are ones that worked with these companies in the past. So we went to them and said we need to bring attention to this. You've been able to convince people to buy all these things which are bad for them. So now we need to throw it the other way. That was very interesting. So that's where we started. So we created this campaign obesity and it's a play on words o-b-e-c-i-t-y and of course it's spelled o-e-s-i-t-y obesity and so we created a world, we created a city obecity come and visit obecity a visit obecity.org is the website and when you visit obesity.org you're going to enter in the city where fast food restaurants are outnumbering, I forget the

statistics on there now and I should know them on the off the top of my head but you've got all kinds of very graphic advertisements. They say where pizza is a vegetable. It's where you can have them have a muffin for breakfast but it's also known as dessert like so they just play on all of these different facts that exist within our normal society that people don't associate with obesity. So it's really important because in 2013 the American Medical Association which was endorsed by many other organizations classified obesity as a disease, defined it as a disease but it's still not recognized as a disease. When we think about cancer there is a lot more empathy for a person who is dealing with cancer because cancer is viewed to be not the person's fault. They are the victim when they develop cancer but when we think about obesity it's still thought to be the person's fault and so when somebody has obesity that they've basically dealt their own hand that's the view. And that might be true but obesity as a state of someone's metabolic health is totally different from the weight gain that gets them there. Yes obviously we're talking about an energy imbalance problem. Yes obviously in order to develop obesity at some point or another there was a long-term imbalance between calories in and calories out but how much of that is the person's fault. We have socio-determinants of health that's what you'll also learn when you visit obesity.org. You'll hear about the food desserts right and the lack of access to healthy foods and the lack of access to fresh fruits and vegetables and the lack of access to safe places to go walking which is for a large majority of people in the United States and around the world. So part of the campaign is to advertise on obesity. And then part of the campaign which is one of the reasons why I like talking about the intergenerational transmission of obesity is to introduce people to two of the residents there and we've created two animated characters at the moment; a teenage girl and a teenage boy. So we have Charlie and Ezra and there are very short like one minute long animated videos where you can learn about Charlie and Ezra and you see the environment in which they live. You see where they go to school and you realize at that point that the cards are stacked against them and so if this is the generation that's being born today how is obesity their fault. That's the world in which they've been born.

DANNY LENNON: Yeah that's the thing that struck me of how good a job it does to illustrate those very facts of that this is not just a health or a nutrition problem, this is a societal problem and we have these various socioeconomic determinants that you've mentioned whether that's from socioeconomics or that disparity in health that tends to accompany disparities in wealth and you mentioned some of those statistics between poor neighborhoods versus wealthier neighborhoods and then the role of industry and it's not a coincidence that you have this like density of fast food restaurants in some of these areas and then the more people become aware of that we can see that why we say that obesity is clearly out of someone's hands in the vast majority of cases because of all these determining factors.

LEANNE REDMAN: Right. So we have to come together. We've got to acknowledge that there's weight stigma. This obesity is not a cosmetic issue. We're not trying to encourage people to reduce their body size and to reduce their adiposity so that they look better. It's not about that. It's about health and we have to try to optimize health at every size and so we have to stop the blame and so in order to stop the blame we have to get the message out there and advocate for obesity as a disease. We have to be able to reach people with obesity to ensure that they know that we see them, we hear them, we understand. It's a complex issue. We're trying to understand it more and to get better treatment options and prevention options and all those things and then we have to appeal to the public. When we were developing the campaign and we realized we also needed to have a vehicle to try to fundraise to get more money back into obesity research but from like just the general population, the different philanthropy groups they rank the amount of money that people donate each year to different causes and of course cancer's way up there and all of that. Obesity was not even ranked as a cause and yet it's one of the most prevalent health problems of modern times.

DANNY LENNON: Yeah. And you kind of see the same parallel in the major need for public policy reform in some of these areas but without pressure from a broader public it's

very easy for policy makers to neglect that area more than should be I guess.

LEANNE REDMAN: Yeah and so I was sort of hoping that one of the silver linings with the COVID-19 pandemic is an increased awareness of obesity since many more of the individuals who have become infected and had worse outcomes and even more worsened mortality have been individuals with obesity. I was hoping that that would be one of the silver linings is that it's just going to get a lot more attention as to its seriousness and the pandemic's not over obviously so hopefully by the time it's over and we can sit back as scientists and the research community and start to really collate all the facts and share those with the public that we can elevate obesity to the extent that it needs in order to move the needle and improve the health for everyone.

DANNY LENNON: Yeah. And I guess one of the the cruel ironies is that because of the pandemic much of the service provision to people living with obesity has been disrupted to some degree as well but with that Prof. Redman, I'm very mindful of your time this has been absolutely fascinating. So before I get to my very final question can you let people know where they can maybe find you on social media, where they can find a word about your lab on the internet or any other links you might want to send them towards and then obviously remind them of that visit obesity link as well?

LEANNE REDMAN: I'm going to give you a shout out to first of all my Twitter feed. I guess that's easy that I'm just Dr. Leanne Redman on Twitter very simple straightforward. I'm also on LinkedIn. They can search for me there. At the Pennington Biomedical Research Center Website which is pbrc.edu. We have faculty pages on there and I can be found there and of course check out the obesity campaign website visit obecity which is obecity.org.

DANNY LENNON: Fantastic and for everyone listening I will link to all of that in the show notes so you can go and click through and check that out and with that that brings us to the final question I always end the podcast on which can be completely separate from today's topic if you wish but it's simply if you could advise people to do one

thing each day that might have a positive impact on any area of their life what might that one thing be?

LEANNE REDMAN: It's funny because i deal with health and lifestyle and all these things every day but that's not going to be my my answer. I think more than ever now we have to learn to listen and I think we have to be kind. We've lost some sense of empathy as a human race and we're so quick to jump to conclusions maybe upon our own biases but I think that there's still a general lack of understanding and the more we can learn to listen, be kind, and show empathy that's how we can come together as a human race.

DANNY LENNON: Very wise and certainly something I agree with and I don't know if there's ever been a time where we need that advice more than now when you look around most societies at the moment but with that Prof. Redman let me say thank you so much for taking the time to talk to me today. I've really really enjoyed this but more on that I've also really enjoyed your work and reading that over a period of time. So thank you for doing this. I really-really appreciate it.

LEANNE REDMAN: Thank you. I'm giving you my virtual fist bump.

DANNY LENNON: Nice. There we go awesome.