Researching Oxidized High-Density Lipoprotein in Obese Adolescents

Dr. Alan Brown:
I'm your host Dr. Alan Brown and I'm at the National Lipid Association's annual scientific sessions and with me is one of the top winners of the National Lipid Associations Young Investigator Competition, Dr. Monica Marin. Dr. Marin is a Pediatric Endocrinology fellow at the University of Oklahoma Health Sciences Center and today we'll be discussing her trail on oxidized HDLI lipoprotein in obese adolescence.

So Dr. Marin, I had a chance to talk with you a little bit before the interview today and I know you're from Oklahoma City but your accent doesn't sound like Oklahoma City. Tell us a little bit about where you're from and how you ended up in Oklahoma.

Dr. Monica Marin:
I'm originally from Romania. I did medical school in Romania, and I came in United States for residency and did my residency in New York City, in Brooklyn, and decided to continue with Pediatric Endocrinology Fellowship and that's how I ended up in Oklahoma City.

Dr. Alan Brown:
Okay, fantastic. So this is a really fascinating and in depth study. Let's start off with how you got the
idea and then tell us a little bit about how you designed the trial.

Dr. Monica Marin:
Sure. So cardiovascular disease usually manifests in adulthood but we know from previous studies that cardiovascular risk factors can be present in childhood. So, _____ (1.25) children is associated with increased inflammation and elevated proatherogenic particles. As a pediatric endocrinologist I thought that it is important to identify a cardiovascular risk factor that are present in childhood.

Dr. Alan Brown:
Okay. So tell us a little bit about what you looked at in your trial and the study design.

Dr. Monica Marin:
So my primary outcome was oxidized HDL but I also looked for other secondary outcomes such as insulin resistance and other markers of oxidative stress, oxidized HDL and myeloperoxidase.

Dr. Alan Brown:
So let me ask you, what are the implications of having high levels of oxidized HDL for our audience who aren’t lipidologists? They know a lot about oxidation of LDL particles and how that’s proatherogenic. What happens when HDL gets oxidized?

Dr. Monica Marin:
So when HDL gets oxidized it becomes potentially atherogenic too. Actually its oxidized _____ (2.39) protein particle from the HDL and these will interfere with the reverse cholesterol transport. So, if your cholesterol influx is affected then oxidized HDL potentially becomes proatherogenic.

Dr. Alan Brown:
Very interesting. Okay, so tell us then a little bit about the study and who is enrolled in the trial and then we’ll talk about the results.

Dr. Monica Marin:
Okay. So I had enrolled 37 subjects in each group and I had two groups. One was normal weight and the other one was the obese group. The normal weight, they had BMI’s between 25 and 75 so they were really lean children, and the obese had BMI above 95 percentile. They had similar ages and similar tanner staging as well there raised distribution was similar.

Dr. Alan Brown:
These are adolescents, right? So, what was the age range of your…

Dr. Monica Marin:
So they were the mid-age was 14.5.
Dr. Alan Brown:
Okay, so young children. So, you measured as you said oxidized HDL, myeloperoxidase. Did you measure any other inflammatory markers?

Dr. Monica Marin:
Didn’t measure inflammatory markers. It just measured oxidized LDL and myeloperoxidase.

Dr. Alan Brown:
If you’re just tuning in you’re listening to ReachMD. I’m Dr. Alan Brown and I’m speaking to Dr. Monica Marin, a Pediatric Endocrinology Fellow and winner of the National Lipid Association’s Young Investigator Competition.

So Dr. Marin, tell us a little bit more then about the results of the trial. So as I understand it, you’ve looked at average age 14-year-olds, normal weight and obese and you measured oxidation of HDL and you eloquently have told us about how that can aggravate atherosclerosis and compete with reverse cholesterol transport. So, tell us what you found.

Dr. Monica Marin:
I found that other oxidative markers, the oxidized LDL was significantly higher in the obese group. The myeloperoxidases was higher in the obese group but not statistically significant, but the oxidized HDL was not different between the two groups even though the HDL cholesterol concentration was lower in the obese group.

Dr. Alan Brown:
So if the total HDL cholesterol was lower and you had equal oxidized HDL, would the implication be that a greater percentage of the HDL was oxidized in the obese children or do I have that wrong?

Dr. Monica Marin:
Actually there was no correlation between the HDL and oxidized HDL so I couldn’t point to anything.

Dr. Alan Brown:
Okay. So what do you think the implications of your findings were in terms of obesity in children? What should we take away from this study?

Dr. Monica Marin:
Definitely obesity in children affects the LDL cholesterol and this is more oxidized in obese children. But this was known before from other studies and it seems that the oxidative stress and inflammation associated with obesity in children doesn’t affect the HDL as much. So the oxidized HDL was not
different between lean and obese children.

Dr. Alan Brown:
So can you tell the audience a little bit about the myeloperoxidase? It wasn’t statistically significant, but for those who I know this is a relatively new marker of atherosclerosis and as long as you looked at it explain to the audience what the implications of myeloperoxidase levels are.

Dr. Monica Marin:
So the myeloperoxidase is released from _____ (6.52) under an inflammatory state and it contributes to production of a reactive oxygen species that are known to cause oxidation of a LDL and there were a few articles that described oxidation of HDL as well under the influence of myeloperoxidase.

Dr. Alan Brown:
Very interesting. So, as you go forward in your career as a Pediatric Endocrinologist and now you’ve done this clinical trial, which we’re grateful you presented to the National Lipid Association, how are you gonna incorporate this enthusiasm for obesity in children into your career?

Dr. Monica Marin:
I’m currently a third year fellow so I’m almost done with my fellowship. I plan to continue one more year with a research year, but in the future I really want to focus on this domain of obesity and beginning of atherosclerosis in childhood and the goal will be to identify markers that can be actually treated in childhood and prevent the progression of atherosclerosis in to adulthood.

Dr. Alan Brown:
That’s certainly _____ (8.16). We are obviously all concerned about the potential for our children’s generation to live less lengthy a life than their parents. This would be the first generation in the history of mankind to have that happen and obesity is the obvious precursor to that. So, I certainly hope that your efforts bear fruit and I appreciate you taking the time to present this interesting study at the National Lipid Association.

Dr. Monica Marin:
Thank you for inviting me.

Dr. Alan Brown:
I’m Dr. Alan Brown and you’ve been listening to Lipid Lumination sponsored by the National Lipid Association on ReachMD. Be sure to visit our website at ReachMD.com/lipids featuring podcasts of this and other series. Thank you all very much for listening.