

Transcript Details

This is a transcript of a continuing medical education (CME) activity. Additional media formats for the activity and full activity details (including sponsor and supporter, disclosures, and instructions for claiming credit) are available by visiting:

<https://reachmd.com/programs/cme/remnant-cholesterol-the-missing-link-in-ascvd-risk/33226/>

Released: 05/30/2025

Valid until: 05/30/2026

Time needed to complete: 48m

ReachMD

www.reachmd.com

info@reachmd.com

(866) 423-7849

Remnant Cholesterol: The Missing Link in ASCVD risk

Announcer:

Welcome to CME on ReachMD. This activity is provided by Medcon International. This episode is part of our MinuteCE curriculum.

Prior to beginning the activity, please be sure to review the faculty and commercial support disclosure statements as well as the learning objectives.

Dr. Ray:

Hello, this is CME on PACE-CME and ReachMD. I'm Kausik Ray and joining me is Professor Børge Nordestgaard. Welcome Børge.

Dr. Nordestgaard:

Thank you so much. Nice to see you.

Dr. Ray:

Lovely to see you. And one of the things I want to discuss with you today in this episode is about remnant cholesterol. Can you start by explaining what remnant cholesterol is and its relationship to triglycerides, and how it increases the risk of atherosclerotic cardiovascular disease?

Dr. Nordestgaard:

Definitely. So, first of all, let's try to make it very clear. The remnant cholesterol are found in exactly the same particles as the triglycerides, but most MDs tend to focus on the triglycerides because that's the value they get from the laboratory. But these particles have cholesterol also, and it is the cholesterol content that drives atherosclerosis and end up in the atherosclerotic plaque. There's no triglycerides in there.

However, the triglycerides could do a part because triglycerides will also, in the arterial intima be degraded into free fatty acids, monoglycerides, and they will be toxic to tissues and thereby, drive local inflammation. So, maybe they're part of more making of vulnerable plaque. And then if you have atherosclerosis, getting a myocardial infarction, or it could even be a stroke.

So, hopefully people understand more and more it is the cholesterol content, the remnant cholesterol, that drives the atherosclerosis, whereas the triglycerides maybe is more what drives plaque rupture or even pancreatitis.

And we have a new thing coming about, namely APOC3 inhibition. That is a novel therapeutic approach. There is some data now, showing it not only reduces triglycerides but also reduces remnant cholesterol up to 50%, and that is way more than we have seen before in other drug types.

Dr. Ray:

So, that's been a great discussion, and it's obviously time to wrap up. So, the key take-home, one thing that you would want the audience to know, what would that be?

Dr. Nordestgaard:

So, it's the cholesterol in the triglyceride-rich lipoproteins called remnant cholesterol, those are the ones that are the main cause of

atherosclerosis and thereby, atherosclerotic cardiovascular disease, myocardial infarction and stroke, not the triglyceride content.

Dr. Ray:

Great summary. So, from my side, the key take-home is that we normally think about LDL cholesterol but when we have patients with these elevated triglycerides, they have an additional residual atherogenic burden. Thank you so much. I've really enjoyed speaking with you today, and that's all we have time for today.

Announcer:

You have been listening to CME on ReachMD. This activity is provided by Medcon International and is part of our MinuteCE curriculum.

To receive your free CME credit, or to download this activity, go to ReachMD.com/CME. Thank you for listening.