New perspectives challenge the idea that saturated fats cause heart disease

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In science, sometimes a new perspective can turn our interpretation of the data upside-down, and necessitate a paradigm shift.

There has been, and continues to be, fierce disagreements in nutrition science as to what constitutes a healthy diet. A key controversy is the role of saturated fats in health and disease. Saturated fats are known to increase blood cholesterol levels, and increased blood cholesterol is often observed in people who develop cardiovascular disease.

It has been thought for more than half a century that saturated fats in the diet promote heart disease by increasing blood cholesterol. However, a new model explains why this so-called 'diet-heart hypothesis,' which has had a major influence on dietary guidelines, may be wrong.

In a new article published today in the *American Journal of Clinical Nutrition*, three scientists have raised a question that challenges the diet-heart-hypothesis: Why do saturated fats increase blood cholesterol, and why should this be dangerous? After all, saturated fats occur naturally in a wide variety of foods, including breast milk.

"Cholesterol is a critically important molecule for all cells in the body," explains associate professor Marit Zinöcker, the lead author at Bjørknes University College, Oslo, Norway. "A cell is surrounded by a fluid membrane that controls cell function, and the cells depend on the ability to incorporate a certain amount of cholesterol molecules, so that their membranes don't become too stiff or too fluid."

"The basis of the model is that when saturated fats replace polyunsaturated fats in the diet, less cholesterol is needed in the cell

membranes," she explains. The opposite is true when eating more polyunsaturated fatty acids, which include omega-3 and omega-6 fatty acids. "This is because polyunsaturated fats from the diet enter our cell membranes and make them more fluid. The cells adjust the fluidity of their membranes by incorporating cholesterol recruited from the bloodstream. According to the model presented by the researchers, this can explain why blood cholesterol levels decrease when we eat more polyunsaturated fats.

The authors have named the model the Homeoviscous Adaptation to Dietary Lipids (HADL) model.

"Cells need to adjust their membrane fluidity according to changes in their environment, such as the access to different types of fat", says co-author Simon N. Dankel, researcher at the Department of Clinical Science, University of Bergen, Norway.

"This phenomenon is called homeoviscous adaptation, and has been described in both microorganisms, vertebrates and in human skin cells. We argue that this is a critical principle in human physiology. Our cells are normally capable of adjusting their cholesterol content according to changes in dietary fats."

"Nutrition research often focuses on what changes in the body, but the question of why something, such as the blood cholesterol, changes, is of equal importance", says co-author Karianne Svendsen, postdoctoral fellow at the Department of Nutrition, University of Oslo, and Oslo University Hospital, Norway.

This is where the new HADL model comes into play, providing an explanation based on adaptive human physiology. "From the perspective of the HADL model, we find logical explanations for why cells need to change their cholesterol content, and thereby the blood cholesterol, when

fats in the diet change," says Zinöcker.

"We know that the causes of atherosclerosis and heart disease are multifactorial. With this model we propose to disconnect the blood-cholesterol raising effect of diet from the elevated blood cholesterol that is causally linked to heart disease", says Svendsen.

In the paper, other reasons for elevated LDL-cholesterol in people with cardiovascular disease are discussed, such as low-grade inflammation and insulin resistance. This indicates that elevated blood cholesterol caused by metabolic disruptions must be uncoupled from elevated blood cholesterol caused by a major change in intake of dietary saturated fatty acids. It also questions the benefit of lowering blood cholesterol by adding polyunsaturated fatty acids to the diet, and not addressing the root cause.

"There is at best weak evidence that a high intake of saturated fat causes heart disease," says Dankel. "The overall data are inconsistent and unconvincing, not to mention the lack of a logical biological and evolutionary explanation."

"Also, people with metabolic disorders often do not show the expected changes in blood cholesterol when changing their fat intake, suggesting loss of the normal response."

"The research and reasoning that the HADL model is based on indicates that the effect of dietary fats on blood cholesterol is not a pathogenic response, but rather a completely normal and even healthy adaptation to changes in diet." Zinöcker concludes.

The authors state that although the model is based on existing knowledge of cellular mechanisms, the model still needs to be verified. The authors therefore urge researchers to discuss the HADL model using

#HADLmodel and to test the model.

More information: Marit Kolby Zinöcker et al, The homeoviscous adaptation to dietary lipids (HADL) model explains controversies over saturated fat, cholesterol, and cardiovascular disease risk, *The American Journal of Clinical Nutrition* (2020). DOI: 10.1093/ajcn/nqaa322

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