Twitter Thread by Marit Kolby Zinöcker





Did we get dietary saturated fats all wrong? The #HADLmodel provides a new understanding and an opportunity to get it right. THREAD

Increased dietary saturated fatty acids lead to increased cholesterol in lipoproteins, but we don't know why. Enter the #HADLmodel, which explains changes in lipoprotein cholesterol as adaptive homeostatic adjustments that ensure optimal cell membrane fluidity and cell function.

We propose that circulating lipoproteins enable appropriate redistribution of cholesterol molecules between specific cells and tissues, to accomodate changes in dietary fatty acid supply, due to our omnivore nature and variable intake of fatty acids. #HADLmodel

Our #HADLmodel implies that circulating levels of LDL change for protective, not for pathological reasons; an SFA-induced raise in LDL cholesterol in healthy individuals is a normal response, while a lack of this needed response may reflect a deeper pathology in lipid handling.

Circulating lipoproteins may change for pathological reasons, when regulatory mechanisms become disrupted by pathogenic processes related e.g. to inflammatory processes. Diverging lipoprotein responses in healthy versus metabolically unhealthy individuals support this view.

Low grade inflammation can interfere with several fine-tuned signaling pathways necessary for homeostasis, including proper lipid handling. Altered circulating cholesterol levels may here reflect underlying pathogenic processes, unrelated to saturated fat intake. #HADLmodel

Dietary factors causing chronic low-grade inflammation, driven by diet-microbiome interactions, should receive more attention. The role of saturated fats in pathogenesis may be misconceived and greatly exaggerated. #HADLmodel

Is the #HADLmodel impossible? Is there more evidence to support the model? What else do we need to test in high-quality studies? Keep the discussion going - fair and factual. We need to improve the conversation on dietary fats. #publichealth #dietaryguidelines

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