OPEN ACCESS Freely available online

ISSN: 2155-9589
Journal of Membrane Science & Technology

Perspective

Oxidized Low-Density Lipoproteins (oxLDLs) and their Risk Factors in Cardiovascular Diseases

Irena Gooch*

Department of Medicine, University of Illinois at Chicago, Chicago, USA

DESCRIPTION

One of the main risk factors for the development of Coronary Artery Disease (CAD) and plaque formation is thought to be alterations of Oxidized Low-Density Lipoprotein (oxLDL). Likewise, both in humans and in animal models of atherosclerosis, excessive levels of oxLDL are linked to an increased risk of CAD and correlate with plasma hypercholesterolemia. It is also well established that exposure to oxLDL causes a variety of pro-inflammatory and pro-atherogenic consequences, although the underlying processes are still up for debate. According to the current theory, oxLDL causes cells to become saturated with cholesterol, which leads to the development of cholesterol-loaded macrophages (foam cells) and defective endothelial cells.

A increasing body of research, however, suggests that rather than cholesterol loading, the effects of oxLDL on membrane cholesterol homeostasis may instead include cholesterol depletion and disruption of cholesterol-rich membrane regions (membrane rafts). Membrane rafts were first defined as microdomains rich in cholesterol and sphingolipids that serve as platforms for protein-protein interactions in a variety of signalling cascades. According to another definition, membrane rafts are "dynamic, nonoscale, sterol-sphingolipid-enriched, organized assembles of proteins and lipids" that are controlled by particular lipid-lipid, protein-lipid, and protein-protein interactions. This article aims to present the most current developments in our knowledge of how oxLDL affects membrane rafts.

The term "oxidized LDL" refers to LDL preparations that have undergone *ex vivo* oxidative modification under specific guidelines or that have been extracted from biological sources. Incubating LDL with metal ions, specifically $Cu2^{+}$, is the most usual method of LDL oxidation *ex vivo*. This process results in the production of numerous oxidized products in the LDL particle, including oxysterols, oxidized phospholipids, and changed apolipoprotein B. Based on the level of LDL oxidation, the oxidized LDL preparations discussed in the literature can be roughly categorised into two groups: Minimally Modified LDL (MM-LDL) and (completely or extensively) oxidized LDL (oxLDL). Depending on the length of exposure and ion concentration, $Cu2^{+}$ oxidation of LDL can produce both minimally changed and totally oxidized LDLs.

Enzymatic oxidation by 15-lypoxygenase or myeloperoxidase or incubating LDL with cells expressing 15-lypoxygenase is two other methods for producing oxLDL *ex vivo*. While it is debatable whether $Cu2^{+}$ oxidation happens *in vivo*, it has been demonstrated that oxLDL seen in atherosclerotic lesions and $Cu2^{+}$ oxidized LDL share many features. The majority of research that looked at how oxLDL affected membrane rafts used $Cu2^{+}$-oxidized LDL. It is generally recognised that caveolae are a physically distinct subpopulation of membrane rafts that contain a variety of signalling complexes and are crucial for controlling cell signalling.

It is also now widely accepted that noncaveolae membrane rafts exist since cholesterol-rich membrane domains can form in the absence of caveolin. Several excellent studies have summarised the ongoing debate over the precise form, morphology, size, density, and molecular makeup of noncaveolae rafts in cellular membranes; a thorough examination of these subjects is outside the purview of the present review. The clustering of the proteins that are tethered to Glycosylphosphatidylinositol (GPI-anchored proteins) or the presence of ganglioside GM1, another important membrane raft marker, are the two standard methods for identifying these noncaveolae membrane rafts.

Furthermore, it has been demonstrated that, despite partitioning into detergent-insoluble fractions that contain membrane domains rich in cholesterol, GPI-anchored proteins are diffusely dispersed at the cell surface and do not colocalize with caveolae unless they have been cross-linked. The surface area occupied by cholesterol-rich membrane domains appears to occupy a substantially larger area of the plasma membrane surface, as determined by monitoring several membrane raft markers, whereas caveolae are predicted to comprise only around 2%-7% of the cell surface. For instance, single lipid molecule tracking

Correspondence to: Irena Gooch, Department of Medicine, University of Illinois at Chicago, Chicago, USA, E-mail: irenagoochi@uic.edu

Received: 27-Jan-2023, Manuscript No. JMST-23-20351; **Editor assigned:** 30-Jan-2023, Pre QC No. JMST-23-20351 (PQ); **Reviewed:** 13-Feb-2023, QC No. JMST-23-20351; **Revised:** 20-Feb-2023, Manuscript No. JMST-23-20351 (R); **Published:** 02-Mar-2023, DOI: 10.35248/2155-9589.23.13.329

Citation: Gooch I (2023) Oxidized Low-Density Lipoproteins (oxLDLs) and their Risk Factors in Cardiovascular Diseases. J Membr Sci Technol. 13:329.

Copyright: © 2023 Gooch I. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

revealed that the regions of slow lipid motion, which are predicted to correspond to the organized domains of the membrane rafts rich in cholesterol, occupy around 15% of the plasma membrane. Similar or even larger values (10%-40%) were found using various probes in other research. Hence, it is commonly accepted that a sizable population of cholesterol-rich organized membrane domains includes noncaveolae rafts. OxLDL also causes the hydrolysis of Sphingomyelin (SM), a second important lipid component of membrane rafts, according to a number of studies.