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Do lipid peroxidation products contribute to atherosclerotic calcification?

Background: Cardiovascular diseases, including atherosclerosis, are the leading cause of death in the United States. Atherosclerotic lesions are formed by deposition of lipids in the intima of arteries. Upon exposure to oxidative stresses, low-density lipoprotein (LDL) is converted to highly atherogenic oxidized LDL (ox-LDL) particles, which contribute to disease development and progression. Advanced disease stages may result in calcification of lesions. This calcification process is important, as it has been shown to be associated with stable plaques that are less prone to rupture. Calcification is present in lipid rich domains of lesions and correlates well with overall plaque burden. However, neither the composition of the mineralized calcium deposits nor its relationship to lipid peroxidation is known.

Methods: In this study, the potential of lipid peroxide-derived lipophilic dicarboxylic acid (DCA, e.g. azelaic acid) to promote calcification upon exposure to vascular smooth muscle cells was tested. Using 13-Hydroperoxyoctadecadienoic acid (13-hydroperoxylinoleic acid, 13-HPODE) and thin-layer and gas chromatography–mass spectrometry we characterized the conditions where HPODE is decomposed to aldehyde product 9-oxo-nonanoic acid and its corresponding DCA azelaic acid (AZA).

Results: HPODE treatment resulted in the cellular conversion to ONA and AZA as determined by GC-MS. Both free AZA and intracellular delivery of AZA via lyso phosphatidylcholine (lysoPtdCho) micelles induced calcification of aortic smooth muscle cells, as determined by Von Kossa and alizarin red staining.

Conclusion: These results demonstrate that DCAs may contribute to atherosclerotic calcification thus accounting for the latter's relationship to plaque burden and association with lipids. This study also challenges the dogma that arterial calcification represents the deposition of calcium phosphate. Our future work aims to delineate the association of calcium with lipid rich plaques and lipid oxidation with calcification in animal and human atherosclerosis.

Biography

Sampath Parthasarathy MBA, PhD, was instrumental in the development of the concept of oxidized LDL and its contribution to atherosclerosis, a major form of cardiovascular disease. He is currently at University of Central Florida as the Florida Hospital Chair in Cardiovascular Sciences and the Associate Dean of Research. He has published over 250 articles and has served on numerous editorial boards and NIH committees. He has been continuously funded by NIH and other agencies for over 30 years and was awarded the distinguished service Award by the American Heart Association and by the American Association of Cardiologists of Indian Origin and from SASAT International. He is also the recipient of the prestigious van Deenen Memorial award for lipids and the Ranbaxy Award for excellence in cardiovascular research.

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