## Lipid peroxidation

Lipid peroxidation is the oxidative degradation of lipids. It is the process in which free radicals "steal" electrons from the lipids in cell membranes, resulting in cell damage. This process proceeds by a free radical chain reaction mechanism. It most often affects polyunsaturated fatty acids, because they contain multiple double bonds in between which lie methylene bridges (-CH2-) that possess especially reactive hydrogen atoms. As with any radical reaction, the reaction consists of three major steps: initiation, propagation, and termination. The chemical products of this oxidation are known as lipid peroxides or lipid oxidation products (LOPs).

Our understanding of lipid peroxidation in biology and medicine is rapidly evolving, as it is increasingly implicated in various diseases but also recognized as a key part of normal cell function, signaling, and death (ferroptosis). Not surprisingly, the root and consequences of lipid peroxidation have garnered increasing attention from multiple disciplines in recent years. Here we "connect the dots" between the fundamental chemistry underpinning the cascade reactions of lipid peroxidation (enzymatic or free radical), the reactive nature of the products formed (lipid-derived electrophiles), and the biological targets and mechanisms associated with these products that culminate in cellular responses. We additionally bring light to the use of highly sensitive, fluorescence-based methodologies enable visualizing and quantifying each reaction in the cascade in a cellular and ultimately tissue context, toward deciphering the connections between the chemistry and physiology of lipid peroxidation. The review offers a platform in which the chemistry and biomedical research communities can access a comprehensive summary of fundamental concepts regarding lipid peroxidation, experimental tools for the study of such processes, as well as the recent discoveries by leading investigators with an emphasis on significant open questions <sup>1)</sup>.

Estradiol administration inhibited the formation of lipid peroxidation products and restored middle cerebral arterial viscoelasticity in aged female rats<sup>2</sup>.

1)

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