Editorial

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Biomarkers

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Free radical biologists have used the word "biomarker" from molecular epidemiology to characterise a molecular alteration in a biological molecule caused by reactive oxygen, nitrogen, or halide species. It is equally applicable to compounds formed from the consumption of lipids, DNA, proteins, and antioxidants, when the chemical basis of the process is proton abstraction, electron transfer, or direct addition. The rates at which these molecules react with the hydroxyl radical reveal that the reaction is governed by diffusion, and hence by closeness to the source of the radical. Peroxy radicals, on the other hand, are more stable, with a half-life of seconds, allowing dispersion to a remote location. The examination of distinct biomarkers isolated from tissues/organelles/fluids can reveal information about the nature of the denaturing radical as well as the localization of oxidative stress. As a result, biomarkers have been employed to assess the efficacy of a variety of antioxidants *in vitro*, *ex vivo*, and *in vivo*, with inconsistent results. Biomarkers can provide information on disease outcome on three levels:

- As measurable endpoints of damage to proteins/amino acids, oxidised lipids, and oxidised DNA bases
- As functional markers of blood flow, platelet aggregation, or cognitive function
- As endpoints related to specific disease

A set of biomarkers, each validated in turn, would be preferable. To that goal, the link between a biomarker and a disease must be established. While no known biomarker exists to bridge the gap between exposure and outcome, our growing understanding of the functional repercussions of biomolecule oxidation is bringing this prospect closer. Current ‘first level' biomarkers may one day be shown to be directly related to functional alterations and, eventually, disease. Meanwhile, they can provide valuable insight into the nature of radical damage and antioxidant action *in vivo*, particularly with regard to pro-oxidant effects, compartmentalization, and bioavailability.

The effects of chosen radicals generated by radiolysis on the enzyme lysozyme were the subject of the first research on oxidation of a physiologically essential protein. The Thiocyanate Radical (a selective modulator of tryptophan) and the hydroxyl radical were discovered to inactivate the enzyme, showing that tryptophan residues are required for biological activity. Similarly, a single methionine residue at position 358 in a 1-antitrypsin rendered the protein inactive. Methionine sulphoxide was discovered after further hydrolysis and amino acid analysis. This was one of the first studies to relate amino acid oxidation to protein denaturation and loss of function. Oxidative changes to histidine and lysine in Low Density Lipoproteins (LDL) produce changed receptor recognition; LDL modified in this way is preferentially taken up by scavenger receptors in an unregulated process, demonstrating the importance of protein oxidation in terms of altered function. New functional groups, such as hydroxyls and carbonyls, are typically introduced during protein oxidation, resulting in altered function and turnover. According to the amount of oxidative damage, improved characterization of the consequences of protein oxidation has discovered a range of secondary effects such as fragmentation, cross-linking, and unfolding, which may speed up or slow down proteolytic and proteosome-mediated turnover. The creation of oxidative biomarkers has been hampered by the complexity of protein structure resulting from the primary sequence and the role of carbohydrate moieties in structure stabilisation, as well as a lack of specialised and sensitive techniques. Those amino acids that can delocalize charge, such as those with aromatic or thiol side chains, are more vulnerable to oxidative assault. However, the formation of protein carbonyl moieties causes oxidation of a vast number of aliphatic residues. The study of certain protein-bound oxidised amino acids has also piqued interest. Aromatic and sulphydryl-containing residues have been identified as being particularly vulnerable to oxidative alteration, generating LDOPA (L-3,4-Dihydroxyphenylalanine) from tyrosine, ortho-tyrosine from phenylalanine, sulphoxides and disulphides from methionine and cysteine, and kynurenines from tryptophan, respectively. Later work has documented the finding of valine and leucine hydroxides reduced from hydroperoxide intermediates.