Can Natural Antioxidants Protect Against Free Radicals?

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Evidence indicates that environmental pollutants (including pesticides, transition metals, toxic chemical wastes, cigarette smoke, radiation gasoline and its additives) help in the deterioration of biological macromolecules. This may result in gastrointestinal tract injury, brain and central nervous system damage, liver and kidney damage. Differences in the toxic manifestations of these pollutants may be related to differences in solubility, absorbability, transport, chemical reactivity and the complexes that are formed within the body.

Recent studies have demonstrated that these environmental pollutants produce oxygen free-radicals, resulting in deterioration of lipids, proteins and DNA, activation of procarcinogens, inhibition of cellular and antioxidant defense systems, depletion of sulflydryls, altered calcium homeostasis, and induction of abnormal proteins and altered gene expression.

Free radicals have been implicated in over 100 diseases, including arthritis, hemorrhagic shock, atherosclerosis, aging, ischemia and reperfusion injury of many tissues, central nervous system injury, gastritis, tumor promotion and carcinogenesis and AIDS. This wide range of diseases implies that increased free radical formation accompanies tissue and cellular injury in most, if not all, human diseases.

In most cases, free radicals are believed to significantly contribute to the disease pathophysiology. Oxygen-derived free radicals (and their metabolites) contribute to tissue injury leading to multistage carcinogenesis, which ultimately leads to cancer. Free radical production increases with stress and exposure to carcinogens. Antioxidants inhibit tumor promotion carcinogenesis at both initiation and transformation stages, and protect cells against oxidative damage. Natural antibiotic-producing microflora such as ***Lactobacillus acidophilus*** and ***Bifidobacterium bifdwn*** can provide protection in the gastrointestinal tract.

FREE RADICALS

Free radicals have an unpaired electron. Because of the natural tendency of electrons to form stable pans, the presence of unpaired electrons in free radicals makes them short-lived and extremely reactive.

Free radicals and other reactive oxygen species are continuously produced in the human body through a variety of physiologic and pathologic processes. Examples of free radicals and reactive oxygen species include: superoxide anion radical, hydroxyl radical, thiyl radical, trichloromethyl radical, hypochlorite radical plus hypochlorous acid, and also some potentially dangerous nonradicals such as hydrogen peroxide, singlet oxygen, hypochlorous acid, and the urban air pollutant ozone. Hydroxyl radical is a highly reactive species that can drastically attack all biological molecules including lipoproteins and DNA, usually setting off free-radical chain reactions.

FREE RADICALS AS MEDIATORS OF DISEASES

Many components within the cell are susceptible to attack by free radicals. Free radicals produce their deleterious effects by:

1. per-oxidizing cell membrane lipids, and thus altering the structural integrity of the membrane;
2. peroxidizing the lipids involved in maintaining the microenvironment of membrane protein;
3. reacting both with proteins and with membrane lipids separately or concurrently; and
4. producing DNA damage in the form of DNA single or double strand breaks and crosslinks.

The lipids that constitute the cell membrane are susceptible to free radical attack leading to the formation of lipid peroxides, ketones and aldehydes. Free radicals react with polyunsaturated fatty acids in the phospholipid membrane to regain the thermodynamically lowest energy state, defined by pairing of orbital electrons. The initiating radicals usually achieve this by abstracting a hydrogen atom from a molecule, giving rise to a new free radical. If the target molecule is an unsaturated fatty acid, available molecular oxygen can combine with the resultant fatty acid radical to yield a peroxy radical. The peroxy radical is also very reactive and can attain the paired electron configuration by combining with a hydrogen atom (from an adjacent molecule), forming a hydroperoxidized fatty acid molecule, and another free radical.

Continuation of this process leads to a series of molecular alterations — the early stages of per-oxidized molecular products in unstable or inactivated configurations. These products include peroxidized lipids and proteins, and numerous reactive degradation products (malondialdehyde, acetone, formaldehyde, acetaldehyde, propionaldehyde and methylethylketone) which facilitate intramolecular and intermolecular crosslinking among lipids and proteins. Consequent disorders in structure and