DOWNLOAD PDF RADICALS IN THE BODY

Chapter 1 : Fighting Free Radicals & Free Radical Damage - Dr. Axe

As the body ages, it loses its ability to fight the effects of free radicals. The result is more free radicals, more oxidative stress, and more damage to cells, which leads to degenerative.

Enzymes Every cell in the body creates its own "bomb squad"-antioxidant enzymes complex, machine-like proteins whose specialty is defusing oxy radicals and ROS. The most thoroughly studied defense enzyme, superoxide dismutase SOD , takes hold of molecules of superoxide - a particularly destructive free radical-and changes them to a much less reactive form. SOD and another important antioxidant enzyme set, the glutathione system, work within the cell. By contrast, circulating biochemicals such as uric acid and ceruloplasmin react with free radicals in the intercellular spaces and bloodstream. Nutrients The substances that plants create to fight free radicals can help the human body do the same thing. Among the many substances used are Vitamins C and E, beta-carotene, and bioflavonoids. Some free radical researchers believe that to quench free radicals effectively, the general level of all of these free-radical-fighting nutrients needs to be much higher than nutritional experts have generally thought. Self repair The body also has systems to repair or replace damaged building blocks of cells. These systems are rapid and thorough. For example, the system for repairing damage to DNA and other nucleic acids is particularly elaborate and efficient, with various specialized enzymes that locate damaged areas, snip out ruined bits, replace them with the correct sequence of molecules, and seal up the strand once again. Every aspect of the cell receives similar attention. Most protein constituents in the cell, for example, are completely replaced every few days. Scavenger enzymes break used and damaged proteins into their component parts for reuse by the cell. The body needs only to strike the right balance between the number of free radicals generated and the defense and repair mechanisms available. The goal is to keep oxidative stress from exceeding the capacity of the normal repair and replacement mechanisms. Oxy radicals might, for instance, slip through the enzyme and nutrient defenses and attack the DNA; but ideally these attacks would be few enough that the DNA repair mechanisms could fix the damage and maintain the genetic code intact. How, then, can we keep the desired balance? That question is a major area of research, but the results have been mixed. Vitamins and beta-carotene have shown far fewer benefits than expected. One long-term, large-scale study found beta-carotene to have no effect whatsoever in reducing malignant neoplasms, cardiovascular disease, or death from all causes Hennekens et al Yet we have seen that each cell can produce a wide variety of free radicals. Two recent studies suggest another problem. Beta-carotene and vitamin E were found not to prevent lung cancer in male smokers; in fact, beta-carotene was linked with higher incidence of lung cancer Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group, Beta-carotene and vitamin A supplements, too, were found to increase the risk of lung cancer in smokers and in workers exposed to asbestos Omerun et al A reason for the harmful effects may be that the vitamins, after quenching free radicals, become oxidized themselves unless they have been given in correct doses or regenerated by additional antioxidants, which must be in proper doses themselves. In addition, large amounts of any one micronutrient may inhibit absorption of other micronutrients needed for proper nutritional balance. Such problems might be offset if vitamins were taken in their natural condition, surrounded by dozens of apparently inactive ingredients that modulate their effects. This conclusion is suggested by a study that found vitamin E to have no effect in reducing death from coronary heart disease in postmenopausal women when taken in the form of supplements, but to have significant benefits when absorbed from food Kushi et al Even if these problems with vitamin supplements were solved, the supplements would still have a significant shortcoming. When a molecule of vitamin C or E sacrifices an electron to appease a free radical, the vitamin molecule becomes damaged and useless. Only if it is regenerated by a helpful companion can it re-enter the fray. Enzymes, however, can run through thousands of destructive free radicals and ROS without help and without pause. Unfortunately, while internally produced enzymes are far more powerful than vitamins, they cannot be taken by mouth. They are gigantic protein molecules that cannot pass through the walls of the digestive system and into the bloodstream. Digestive juices break them down into their component amino acids. SOD has been injected directly into inflamed joints, but the procedure is not practical

for home use. Moreover, it has limited effects, because SOD has only a brief half-life in the bloodstream. Recently, the Japanese researcher Tatsuya Oda found a way around this: Riding on the polymers, the SOD lasts in the bloodstream for at least 5 hours. As promising as this finding is, it raises questions about the long-term effects of adding an enzyme to the body in large quantities. These effects are not known as yet, and the question is far from trivial. A preferred solution would be antioxidant substances with 1 low molecular weight, so they can slip from the digestive tract to the bloodstream undamaged, 2 the anti-oxidant ability, weight for weight, of an enzyme such as SOD, and 3 the ability to defuse a wide range of free radicals. In the next section, we will examine research on herbal compounds that appear to satisfy these criteria.

Chapter 2 : Free-radical theory of aging - Wikipedia

In the body, free radicals cause similar deterioration, as they destroy cell membranes and make cells vulnerable to decay and pathogens. These free radicals damage DNA and mitochondria, the basic building blocks of all tissues, and leave in their path many health problems.

Received Mar 26; Accepted May 5. This article has been cited by other articles in PMC. Abstract Free radicals and oxidants play a dual role as both toxic and beneficial compounds, since they can be either harmful or helpful to the body. They are produced either from normal cell metabolisms in situ or from external sources pollution, cigarette smoke, radiation, medication. When an overload of free radicals cannot gradually be destroyed, their accumulation in the body generates a phenomenon called oxidative stress. This process plays a major part in the development of chronic and degenerative illness such as cancer, autoimmune disorders, aging, cataract, rheumatoid arthritis, cardiovascular and neurodegenerative diseases. This mini-review deals with the taxonomy, the mechanisms of formation and catabolism of the free radicals, it examines their beneficial and deleterious effects on cellular activities, it highlights the potential role of the antioxidants in preventing and repairing damages caused by oxidative stress, and it discusses the antioxidant supplementation in health maintenance. When cells use oxygen to generate energy, free radicals are created as a consequence of ATP adenosine triphosphate production by the mitochondria. These by-products are generally reactive oxygen species ROS as well as reactive nitrogen species RNS that result from the cellular redox process. These species play a dual role as both toxic and beneficial compounds. The delicate balance between their two antagonistic effects is clearly an important aspect of life. At high concentrations, they generate oxidative stress, a deleterious process that can damage all cell structures 1 - Oxidative stress plays a major part in the development of chronic and degenerative ailments such as cancer, arthritis, aging, autoimmune disorders, cardiovascular and neurodegenerative diseases. The theory of oxygen-free radicals has been known about fifty years ago 4. However, only within the last two decades, has there been an explosive discovery of their roles in the development of diseases, and also of the health protective effects of antioxidants. This mini-review deals with the taxonomy, the mechanisms of formation and catabolism of the free radicals, it examines their beneficial and deleterious effects on cellular activities, it highlights the potential role of the antioxidants in preventing and repairing damages caused by oxidative stress, and it discusses the advantages and inconveniences of the antioxidant supplementation in health maintenance. Radicals are less stable than non-radical species, although their reactivity is generally stronger. A molecule with one or more unpaired electron in its outer shell is called a free radical 1 - 5. Free radicals are formed from molecules via the breakage of a chemical bond such that each fragment keeps one electron, by cleavage of a radical to give another radical and, also via redox reactions 1 , 2. Biological free radicals are thus highly unstable molecules that have electrons available to react with various organic substrates such as lipids, proteins, DNA. Enzymatic reactions generating free radicals include those involved in the respiratory chain, the phagocytosis, the prostaglandin synthesis and the cytochrome P system 1 - 9. H2O2 a non radical is produced by the action of several oxidase enzymes, including aminoacid oxidase and xanthine oxidase. The last one catalyses the oxidation of hypoxanthine to xanthine, and of xanthine to uric acid. This reaction is known as the Fenton reaction 3 - 8. Hypochlorous acid HOCl is produced by the neutrophil-derived enzyme, myeloperoxidase, which oxidizes chloride ions in the presence of H2O2. Free radicals can be produced from non-enzymatic reactions of oxygen with organic compounds as well as those initiated by ionizing radiations. The nonenzymatic process can also occur during oxidative phosphorylation i. Endogenous free radicals are generated from immune cell activation, inflammation, mental stress, excessive exercise, ischemia, infection, cancer, aging. After penetration into the body by different routes, these exogenous compounds are decomposed or metabolized into free radicals. The importance of ROS production by the immune system is clearly exemplified by patients with granulomatous disease. Other beneficial effects of ROS and RNS involve their physiological roles in the function of a number of cellular signaling systems 7 - 9. Their production by nonphagocytic NADPH oxidase isoforms plays a key role in the regulation of intracellular signaling cascades

in various types of nonphagocytic cells including fibroblasts, endothelial cells, vascular smooth muscle cells, cardiac myocytes, and thyroid tissue. For example, nitric oxide NO is an intercellular messenger for modulating blood flow, thrombosis, and neural activity 7. NO is also important for nonspecific host defense, and for killing intracellular pathogens and tumors. Another beneficial activity of free radicals is the induction of a mitogenic response 7 , 8. Oxidative stress can arise when cells cannot adequately destroy the excess of free radicals formed. For example, hydroxyl radical and peroxynitrite in excess can damage cell membranes and lipoproteins by a process called lipid peroxidation. This reaction leads to the formation of malondialdehyde MDA and conjugated diene compounds, which are cytotoxic and mutagenic. Lipid peroxidation occurs by a radical chain reaction, i. If not regulated properly, oxidative stress can induce a variety of chronic and degenerative diseases as well as the aging process and some acute pathologies trauma, stroke. Cancer and oxidative stress The development of cancer in humans is a complex process including cellular and molecular changes mediated by diverse endogenous and exogenous stimuli. It is well established that oxidative DNA damage is responsible for cancer development. Cancer initiation and promotion are associated with chromosomal defects and oncogene activation induced by free radicals. A common form of damage is the formation of hydroxyled bases of DNA, which are considered an important event in chemical carcinogenesis 3 , 9. This adduct formation interferes with normal cell growth by causing genetic mutations and altering normal gene transcription. Oxidative DNA damage also produces a multiplicity of modifications in the DNA structure including base and sugar lesions, strand breaks, DNA-protein cross-links and base-free sites. For example, tobacco smoking and chronic inflammation resulting from noninfectious diseases like asbestos are sources of oxidative DNA damage that can contribute to the development of lung cancer and other tumors 3 , 6. The highly significant correlation between consumption of fats and death rates from leukemia and breast, ovary, rectum cancers among elderly people may be a reflection of greater lipid peroxidation 5 , Cardiovascular disease and oxidative stress Cardiovascular disease CVD is of multifactorial etiology associated with a variety of risk factors for its development including hypercholesterolaemia, hypertension, smoking, diabetes, poor diet, stress and physical inactivity amongst others 2 , 15 , Recently, research data has raised a passionate debate as to whether oxidative stress is a primary or secondary cause of many cardiovascular diseases Further in vivo and ex vivo studies have provided precious evidence supporting the role of oxidative stress in a number of CVDs such as atherosclerosis, ischemia, hypertension, cardiomyopathy, cardiac hypertrophy and congestive heart failure 2 , 5 , 15 , Pulmonary disease and oxidative stress There is now substantial evidence that inflammatory lung diseases such as asthma and chronic obstructive pulmonary disease COPD are characterized by systemic and local chronic inflammation and oxidative stress 21 - Oxidants may play a role in enhancing inflammation through the activation of different kinases and redox transcription factors such as NF-kappa B and AP-1 23 , Rheumatoid arthritis and oxidative stress Rheumatoid arthritis is an autoimmune disease characterized by chronic inflammation of the joints and tissue around the joints with infiltration of macrophages and activated T cells 4 , 25 , Oxidative damage and inflammation in various rheumatic diseases were proved by increased levels of isoprostanes and prostaglandins in serum and synovial fluid compared to controls Nephropathy and oxidative stress Oxidative stress plays a role in a variety of renal diseases such as glomerulonephritis and tubulointerstitial nephritis, chronic renal failure, proteinuria, uremia 5 , The nephrotoxicity of certain drugs such as cyclosporine, tacrolimus FK , gentamycin, bleomycin, vinblastine, is mainly due to oxidative stress via lipid peroxidation 27 - Heavy metals Cd, Hg, Pb, As and transition metals Fe, Cu, Co, Cr -induced different forms of nephropathy and carcinogenicity are strong free radical inducers in the body 11 , Ocular disease and oxidative stress Oxidative stress is implicated in age-related macular degeneration and cataracts by altering various cell types in the eye either photochemically or nonphotochemically Under the action of free radicals, the crystalline proteins in the lens can cross-link and aggregate, leading to the formation of cataracts In the retina, long-term exposure to radiation can inhibit mitosis in the retinal pigment epithelium and choroids, damage the photoreceptor outer segments, and has been associated with lipid peroxidation Fetus and oxidative stress Oxidative stress is involved in many mechanisms in the development of fetal growth restriction and pre-eclampsia in prenatal medicine 34 - In pregnancies complicated by pre-eclampsia, increased expression of

NADPH oxidase 1 and 5 isoforms which are the major enzymatic sources of superoxide in the placenta is seen

Chapter 3 : What are Free Radicals? (with pictures)

The body is under constant attack from oxidative stress. Oxygen in the body splits into single atoms with unpaired electrons. Electrons like to be in pairs, so these atoms, called free radicals.

We should cleanse our colon first to absorb all vitamins and minerals just for 30 seconds. We are overfed but undernourished. I recommend people eat super foods, natural and organic. However, there are so many natural ways to battle them. With the help of a healthy lifestyle and exercise, and by taking a supplement for healthy skin, like in my case I use Astaxanthin. Microwaving and irradiation helps create free radicals. Slow down and use your oven to heat things up on the stove, especially breast milk and formula. I have seen people in their 80s living healthily by using natural food. Some have not had injected medicine even once and most cases are from rural places where there is little pollution. Unnatural and bad foods work as poison. But at least it attacked one problem. This antioxidant cures everything: Someone told my wife that it will "cure" infections leg so I take it but cannot believe in it. Just wanted to know. What a bunch of crap. Free radicals are necessary for us to live. We are evolved to create them and manage them. DrCellular Post 21 A toxic environment along with natural aging will produce free radicals in the healthiest of people. There is now a product that is an optimized, balanced and stabilized "Redox Signaling Molecules" on the market as of Oct. From South America packed with minerals and vitamins. In addition to feeling great, you will probably lose many pounds. In addition, machines like the Novalite Tesla Photon Generator provide the extra electrons needed to combat aging and disease. I have been making my own now for six months and take it for bowel tolerance, with amazing success. I guarantee you will see results in a week. Food nutrition is the answer to so many ailments and related diseases. Moses kalema- Kampala, Uganda anon Post 8 I still believe the food supply of antioxidants is the best and probably most affordable especially to au in sub-saharan Africa where supplements are economically impossible to have. Moreover, most foods eaten by ruling folks here are locally grown. So it is better they are educated to grow and consume antioxidants rich foods. Ghana anon Post 7 I am with Twigs. Our fruits and vegetables are picked from depleted and over chemically treated soils. Therefore we can not get these vitamins naturally anymore. These days supplementation is the only way. Many, many commercially produced foods are grown in mineral-depleted soils and are, therefore, deficient in many of the required nutrients.

Chapter 4 : Foods That Increase Free Radicals | Healthy Eating | SF Gate

The damage done by free radicals in the body is known as oxidation: Best Ways to Fight Free Radical Damage. 1. Start Eating More Foods Rich in Antioxidants.

Axe content is medically reviewed or fact checked to ensure factually accurate information. With strict editorial sourcing guidelines, we only link to academic research institutions, reputable media sites and, when research is available, medically peer-reviewed studies. Note that the numbers in parentheses 1, 2, etc. The information in our articles is NOT intended to replace a one-on-one relationship with a qualified health care professional and is not intended as medical advice. Our team includes licensed nutritionists and dietitians, certified health education specialists, as well as certified strength and conditioning specialists, personal trainers and corrective exercise specialists. Our team aims to be not only thorough with its research, but also objective and unbiased. December 12, Dr. Axe on Facebook Dr. Axe on Twitter 26 Dr. Axe on Instagram Dr. Axe on Google Plus Dr. Axe on Youtube Dr. These things have become trendy topics as far as health and longevity are concerned. Many people have heard that foods with antioxidants protect us from free radical damage, which is responsible for many of the effects of aging on both the body and mind. But what exactly are free radicals, why are they bad and where do they come from? In order to know how to best protect yourself from health problems linked to free radical damage â€" and there are many â€" it helps to understand what types of lifestyle habits or dietary choices cause them to accumulate in the first place. Despite contributing to the aging process, free radicals are also essential players in the immune system. Our bodies produce free radicals as byproducts of cellular reactions, metabolism of foods, breathing and other vital functions. Why are free radicals thought to be dangerous then? Electrons exist in pairs, and free radicals are missing an electron. This is their weapon of sorts: Free radicals ultimately harm and age the body over time because they damage DNA, cellular membranes, lipids fats stored within blood vessels and enzymes. The damage done by free radicals in the body is known as oxidation: Oxidation is the same process that browns an apple or rusts metal. Rampaging free radicals react with compounds in the body and oxidize them. The amount of oxidation in the body is a measure of oxidative stress. Oxidative stress is believed to lead to the development of the most prevalent chronic diseases and disorders killing adults today, especially heart disease, cancer and diabetes. Oxidation lays the foundation for the proliferation of free radicals and damage to cells, muscles, tissue, organs, etc. Our bodies use antioxidants to lessen the impact of free radicals, and our diets give us the tools to do so. Copper and manganese have roles in antioxidant production as well. Many other phytochemicals from plants also seem to play antioxidant roles. Once consumed they help reduce inflammation and the effects from oxidation. The antioxidant lipoic acid repairs essential enzymes in the body. Even cholesterol can have antioxidant benefits. This helps stop the buildup of fatty plaque on artery walls atherosclerosis and keeps blood flowing to the heart. While we can never entirely stop the aging process, as diet high in antioxidant foods helps us age much more gracefully â€" living longer, healthier, more vibrant lives. Major Sources of Free Radicals So what causes free radicals to proliferate? Free radicals are generated due to oxidation and when toxins are broken down in the body. The liver produces free radicals as it breaks down compounds and removes them. The major sources of free radicals include: Many processed and refined foods contain oxidized fats that add free radicals to the body. Excessive amounts of sugar and sweeteners are other sources of free radical growth that contribute to aging, weight gain and inflammation. Stress hormones like too much cortisol can generate free radicals. The score given to a particular food is known as its ORAC score. Here are just a few foods that have very high ORAC scores: These antioxidant foods help reduce sunburn and wrinkles while protecting your vision. According to the International Dermal Institute, oxygen free radicals are implicated in the overall aging process and are responsible for photoaging, cancer and inflammation in the skin. Spinach and other leafy greens like kale are high in lutein , and tomatoes and red peppers contain lycopene, all of which have anti-aging effects. Berries, grapes and red wine â€" These deeply hued fruits are some of the highest in antioxidants, such as resveratrol. Green and white tea â€" White and green teas are very minimally processed and contain less caffeine than coffee or even other varieties of tea.

They also contain a very high concentration of antioxidants called polyphenols that have been shown to have cancer-fighting properties. Herbs and spicesâ€" These include things like cinnamon, oregano, ginger, turmeric and rosemary. Additionally, essential oils made from the same plants can also be a great source of antioxidant, anti-inflammatory compounds. While eating more antioxidant foods is a big step in the right direction, you also benefit from limiting intake of pesticide- and herbicide-laden foods those that are not organically grown and by avoiding too much sugar, refined oil or refined grains. And be sure to limit intake of antibiotic- and hormone-laden foods, such as farm-raised meat or fish. Avoid Toxin or Pollutant Exposure Besides improving your diet, here are other ways to start reducing free radical damage: Keep in mind that while being sedentary is definitely not helping you to age any slower, either is overworking yourself. Exhaustion, mental fatigue and burnout also cause the immune system and body more damage. According to some experts, there are literally thousands of different antioxidants in the human diet, and they exist in many different forms. Because of the complexities of how antioxidants work in the body to combat free radicals, some scientists believe that only in food form do phytonutrients or antioxidants interact beneficially with our bodies. Surveys shows that about 30 percent of Americans are taking some form of antioxidant supplement. Hensrud points out that most foods with high ORAC scores like cocoa, green tea or acai berries, for example offer great benefits beyond just supplying antioxidants, such as containing fiber, protein, vitamins and minerals. Variety and interaction of many different antioxidants as they exist in food seem to be most beneficial for longevity and optimal health. Final Thought on Free Radicals Our bodies produce free radicals as byproducts of ordinary cellular reactions like breathing or other vital functions, in addition to exposure to pollutants, a poor diet, radiation, high amounts of stress and other toxins. Antioxidants help slow down the effects of free radicals and protect us from disease or signs of early aging. Antioxidant sources include plant foods like fruits or veggies, green or white teas, cocoa, red wine, spices, and herbs. The best way to reduce free radical damage is through a healthy diet and lifestyle, rather than taking supplements. Antioxidant supplements can sometimes cause unwanted effects and are not as beneficial as eating whole plant foods. Top 10 High Antioxidant Foods From the sound of it, you might think leaky gut only affects the digestive system, but in reality it can affect more. Click here to learn more about the webinar.

Chapter 5 : Reactive oxygen species - Wikipedia

Normally, the body can handle free radicals, but if antioxidants are unavailable, or if the free-radical production becomes excessive, damage can occur. Of particular importance is that free radical damage accumulates with age.

Ozone Free radicals in biology Free radical reactions are expected to produce progressive adverse changes that accumulate with age throughout the body [Table 1]. However, superimposed on this common pattern are patterns influenced by genetics and environmental differences that modulate free radical damage. These are manifested as diseases at certain ages determined by genetic and environmental factors. Cancer initiation and promotion is associated with chromosomal defects and oncogene activation. It is possible that endogenous free radical reactions, like those initiated by ionizing radiation, may result in tumor formation. The highly significant correlation between consumption of fats and oils and death rates from leukemia and malignant neoplasia of the breast, ovaries, and rectum among persons over 55 years may be a reflection of greater lipid peroxidation. These compounds induce endothelial cell injury and produce changes in the arterial walls. These injured tissues produce increased radical generating enzymes e. The initiation, promotion, and progression of cancer, as well as the side-effects of radiation and chemotherapy, have been linked to the imbalance between ROS and the antioxidant defense system. Cardiovascular diseases Heart diseases continue to be the biggest killer, responsible for about half of all the deaths. The oxidative events may affect cardiovascular diseases therefore; it has potential to provide enormous benefits to the health and lifespan. Poly unsaturated fatty acids occur as a major part of the low density lipoproteins LDL in blood and oxidation of these lipid components in LDL play a vital role in atherosclerosis. Oxidized LDL is antherogenic and is thought to be important in the formation of anthersclerosis plaques. Furthermore, oxidized LDL is cytotoxic and can directly damage endothelial cells. Antioxidants like B-carotene or vitamin E play a vital role in the prevention of various cardiovascular diseases. Carcinogenesis Reactive oxygen and nitrogen species, such as super oxide anion, hydrogen peroxide, hydroxyl radical, and nitric oxide and their biological metabolites also play an important role in carcinogenesis. Numerous investigators have proposed participation of free radicals in carcinogenesis, mutation, and transformation; it is clear that their presence in biosystem could lead to mutation, transformation, and ultimately cancer. Induction of mutagenesis, the best known of the biological effect of radiation, occurs mainly through damage of DNA by the HO. Radical and other species are produced by the radiolysis, and also by direct radiation effect on DNA, the reaction effects on DNA. The reaction of HO. Radicals is mainly addition to double bond of pyrimidine bases and abstraction of hydrogen from the sugar moiety resulting in chain reaction of DNA. These effects cause cell mutagenesis and carcinogenesis lipid peroxides are also responsible for the activation of carcinogens. B-carotene may be protective against cancer through its antioxidant function, because oxidative products can cause genetic damage. Thus, the photo protective properties of B-carotene may protect against ultraviolet light induced carcinogenesis. Immunoenhancement of B-carotene may contribute to cancer protection. B-carotene may also have anticarcinogenic effect by altering the liver metabolism effects of carcinogens. Vitamin E, an important antioxidant, plays a role in immunocompetence by increasing humoral antibody protection, resistance to bacterial infections, cell-mediated immunity, the T-lymphocytes tumor necrosis factor production, inhibition of mutagen formation, repair of membranes in DNA, and blocking micro cell line formation. The administration of a mixture of the above three antioxidant reveled the highest reduction in risk of developing cardiac cancer. Free radical and aging The human body is in constant battle to keep from aging. Research suggests that free radical damage to cells leads to the pathological changes associated with aging. Some of the nutritional antioxidants will retard the aging process and prevent disease. Based on these studies, it appears that increased oxidative stress commonly occurs during the aging process, and antioxidant status may significantly influence the effects of oxidative damage associated with advancing age. Research suggests that free radicals have a significant influence on aging, that free radical damage can be controlled with adequate antioxidant defense, and that optimal intake of antioxidant nutrient may contribute to enhanced quality of life. Recent research indicates that antioxidant may even positively influence life span. Oxidative damage to

protein and DNA Oxidative damage to protein Proteins can be oxidatively modified in three ways: Protein containing amino acids such as methionine, cystein, arginine, and histidine seem to be the most vulnerable to oxidation. Oxidative damage to protein products may affect the activity of enzymes, receptors, and membrane transport. Oxidatively damaged protein products may contain very reactive groups that may contribute to damage to membrane and many cellular functions. Peroxyl radical is usually considered to be free radical species for the oxidation of proteins. ROS can damage proteins and produce carbonyls and other amino acids modification including formation of methionine sulfoxide and protein carbonyls and other amino acids modification including formation of methionine sulfoxide and protein peroxide. Protein oxidation affects the alteration of signal transduction mechanism, enzyme activity, heat stability, and proteolysis susceptibility, which leads to aging. Lipid peroxidation Oxidative stress and oxidative modification of biomolecules are involved in a number of physiological and pathophysiological processes such as aging, artheroscleosis, inflammation and carcinogenesis, and drug toxicity. Lipid peroxidation is a free radical process involving a source of secondary free radical, which further can act as second messenger or can directly react with other biomolecule, enhancing biochemical lesions. Lipid peroxidation occurs on polysaturated fatty acid located on the cell membranes and it further proceeds with radical chain reaction. Hydroxyl radical is thought to initiate ROS and remove hydrogen atom, thus producing lipid radical and further converted into diene conjugate. Further, by addition of oxygen it forms peroxyl radical; this highly reactive radical attacks another fatty acid forming lipid hydroperoxide LOOH and a new radical. Thus lipid peroxidation is propagated. Due to lipid peroxidation, a number of compounds are formed, for example, alkanes, malanoaldehyde, and isoprotanes. These compounds are used as markers in lipid peroxidation assay and have been verified in many diseases such as neurogenerative diseases, ischemic reperfusion injury, and diabetes. It has been reported that especially in aging and cancer, DNA is considered as a major target. It has been reported that mitochondrial DNA are more susceptible to oxidative damage that have role in many diseases including cancer. It has been suggested that 8-hydroxydeoxyguanosine can be used as biological marker for oxidative stress. These antioxidants delay or inhibit cellular damage mainly through their free radical scavenging property. Some of such antioxidants, including glutathione, ubiquinol, and uric acid, are produced during normal metabolism in the body. History The term antioxidant originally was used to refer specifically to a chemical that prevented the consumption of oxygen. In the late 19th and early 20th century, extensive study was devoted to the uses of antioxidants in important industrial processes, such as the prevention of metal corrosion, the vulcanization of rubber, and the polymerization of fuels in the fouling of internal combustion engines. However, it was the identification of vitamins A, C, and E as antioxidants that revolutionized the field and led to the realization of the importance of antioxidants in the biochemistry of living organisms. Both enzymatic and nonenzymatic antioxidants exist in the intracellular and extracellular environment to detoxify ROS. Antioxidants may exert their effect on biological systems by different mechanisms including electron donation, metal ion chelation, co-antioxidants, or by gene expression regulation. The first line of defense is the preventive antioxidants, which suppress the formation of free radicals. Although the precise mechanism and site of radical formation in vivo are not well elucidated yet, the metal-induced decompositions of hydroperoxides and hydrogen peroxide must be one of the important sources. To suppress such reactions, some antioxidants reduce hydroperoxides and hydrogen peroxide beforehand to alcohols and water, respectively, without generation of free radicals and some proteins sequester metal ions. Glutathione peroxidase, glutathione-s-transferase, phospholipid hydroperoxide glutathione peroxidase PHGPX , and peroxidase are known to decompose lipid hydroperoxides to corresponding alcohols. PHGPX is unique in that it can reduce hydroperoxides of phospholipids integrated into biomembranes. Glutathione peroxidase and catalase reduce hydrogen peroxide to water. Various endogenous radical-scavenging antioxidants are known: Vitamin C, uric acid, bilirubin, albumin, and thiols are hydrophilic, radical-scavenging antioxidants, while vitamin E and ubiquinol are lipophilic radical-scavenging antioxidants. Vitamin E is accepted as the most potent radical-scavenging lipophilic antioxidant. The third line of defense is the repair and de novo antioxidants. The proteolytic enzymes, proteinases, proteases, and peptidases, present in the cytosol and in the mitochondria of mammalian cells, recognize, degrade, and remove oxidatively modified proteins and prevent the accumulation of oxidized

proteins. The DNA repair systems also play an important role in the total defense system against oxidative damage. Various kinds of enzymes such as glycosylases and nucleases, which repair the damaged DNA, are known. There is another important function called adaptation where the signal for the production and reactions of free radicals induces formation and transport of the appropriate antioxidant to the right site. This detoxification pathway is the result of multiple enzymes, with superoxide dismutases catalyzing the first step and then catalases and various peroxidases removing hydrogen peroxide. Mn-SOD is present in mitochondria and peroxisomes. Fe-SOD has been found mainly in chloroplasts but has also been detected in peroxisomes, and CuZn-SOD has been localized in cytosol, chloroplasts, peroxisomes, and apoplast. The first is a dimer consists of two units , while the others are tetramers four subunits. To this end, catalase is frequently used by cells to rapidly catalyze the decomposition of hydrogen peroxide into less reactive gaseous oxygen and water molecules. This system is found in animals, plants, and microorganisms. There are at least four different glutathione peroxidase isozymes in animals. The glutathione S-transferases show high activity with lipid peroxides. These enzymes are at particularly high levels in the liver and also serve in detoxification metabolism. As it cannot be synthesized in humans and must be obtained from the diet, it is a vitamin. In cells, it is maintained in its reduced form by reaction with glutathione, which can be catalyzed by protein disulfide isomerase and glutaredoxins. Glutathione has antioxidant properties since the thiol group in its cysteine moiety is a reducing agent and can be reversibly oxidized and reduced. In cells, glutathione is maintained in the reduced form by the enzyme glutathione reductase and in turn reduces other metabolites and enzyme systems as well as reacting directly with oxidants. Melatonin, once oxidized, cannot be reduced to its former state because it forms several stable end-products upon reacting with free radicals. Therefore, it has been referred to as a terminal or suicidal antioxidant. In fact, uric acid may have substituted for ascorbate in human evolution. There are a number of synthetic phenolic antioxidants, butylated hydroxytoluene BHT and butylated hydroxyanisole BHA being prominent examples. These compounds have been widely uses as antioxidants in food industry, cosmetics, and therapeutic industry. However, some physical properties of BHT and BHA such as their high volatility and instability at elevated temperature, strict legislation on the use of synthetic food additives, carcinogenic nature of some synthetic antioxidants, and consumer preferences have shifted the attention of manufacturers from synthetic to natural antioxidants. It has been reported that there is an inverse relationship between the dietary intake of antioxidant-rich food and medicinal plants and incidence of human diseases. The use of natural antioxidants in food, cosmetic, and therapeutic industry would be promising alternative for synthetic antioxidants in respect of low cost, highly compatible with dietary intake and no harmful effects inside the human body. Many antioxidant compounds, naturally occurring in plant sources have been identified as free radical or active oxygen scavengers. Research has demonstrated that nutrition plays a crucial role in the prevention of chronic diseases, as most of them can be related to diet. Functional food enters the concept of considering food not only necessary for living but also as a source of mental and physical well-being, contributing to the prevention and reduction of risk factors for several diseases or enhancing certain physiological functions. Broccoli, carrots, and tomatoes are considered functional foods because of their high contents of physiologically active components sulforaphen, B-carotene, and lycopene, respectively. Green vegetables and spices like mustard and turmeric, used extensively in Indian cuisine, also can fall under this category.

Chapter 6 : How Free Radicals are Formed In the Body |calendrierdelascience.com

Free radicals, also known simply as radicals, are organic molecules responsible for aging, tissue damage, and possibly some diseases. These molecules are very unstable, therefore they look to bond with other molecules, destroying their health and further continuing the damaging process.

The resulting damage caused by singlet oxygen reduces the photosynthetic efficiency of chloroplasts. In plants exposed to excess light, the increased production of singlet oxygen can result in cell death. In addition to direct toxicity, singlet oxygen acts a signaling molecule. Levels of jasmonate play a key role in the decision between cell acclimation or cell death in response to elevated levels of this reactive oxygen species. These include not only roles in apoptosis programmed cell death but also positive effects such as the induction of host defence [12] [13] genes and mobilization of ion transport systems. In particular, platelets involved in wound repair and blood homeostasis release ROS to recruit additional platelets to sites of injury. These also provide a link to the adaptive immune system via the recruitment of leukocytes. They may also be involved in hearing impairment via cochlear damage induced by elevated sound levels , in ototoxicity of drugs such as cisplatin , and in congenital deafness in both animals and humans. Specific examples include stroke and heart attack. This prevents the spread of the pathogen to other parts of the plant, essentially forming a net around the pathogen to restrict movement and reproduction. In the mammalian host, ROS is induced as an antimicrobial defense. To highlight the importance of this defense, individuals with chronic granulomatous disease who have deficiencies in generating ROS, are highly susceptible to infection by a broad range of microbes including Salmonella enterica, Staphylococcus aureus, Serratia marcescens, and Aspergillus spp. The exact manner in which ROS defends the host from invading microbe is not fully understood. One of the more likely modes of defense is damage to microbial DNA. More recently, a role for ROS in antiviral defense mechanisms has been demonstrated via Rig-like helicase-1 and mitochondrial antiviral signaling protein. This induction of ROS led to the induction of type III interferon and the induction of an antiviral state, limiting viral replication. In addition to energy, reactive oxygen species ROS with the potential to cause cellular damage are produced. ROS are produced as a normal product of cellular metabolism. In particular, one major contributor to oxidative damage is hydrogen peroxide H2O2 , which is converted from superoxide that leaks from the mitochondria. Catalase and superoxide dismutase ameliorate the damaging effects of hydrogen peroxide and superoxide, respectively, by converting these compounds into oxygen and hydrogen peroxide which is later converted to water , resulting in the production of benign molecules. While ROS are produced as a product of normal cellular functioning, excessive amounts can cause deleterious effects. In particular, the accumulation of oxidative damage may lead to cognitive dysfunction, as demonstrated in a study in which old rats were given mitochondrial metabolites and then given cognitive tests. Results showed that the rats performed better after receiving the metabolites, suggesting that the metabolites reduced oxidative damage and improved mitochondrial function. Additional experimental results suggest that oxidative damage is responsible for age-related decline in brain functioning. Older gerbils were found to have higher levels of oxidized protein in comparison to younger gerbils. Treatment of old and young mice with a spin trapping compound caused a decrease in the level of oxidized proteins in older gerbils but did not have an effect on younger gerbils. In addition, older gerbils performed cognitive tasks better during treatment but ceased functional capacity when treatment was discontinued, causing oxidized protein levels to increase. This led researchers to conclude that oxidation of cellular proteins is potentially important for brain function. While studies in invertebrate models indicate that animals genetically engineered to lack specific antioxidant enzymes such as SOD , in general, show a shortened lifespan as one would expect from the theory , the converse manipulation, increasing the levels of antioxidant enzymes, has yielded inconsistent effects on lifespan though some studies in Drosophila do show that lifespan can be increased by the overexpression of MnSOD or glutathione biosynthesizing enzymes. Also contrary to this theory, deletion of mitochondrial SOD2 can extend lifespan in Caenorhabditis elegans. Deleting antioxidant enzymes, in general, yields shorter lifespan, though overexpression studies have not with some recent exceptions consistently extended lifespan. Numerous studies have shown that 8-OHdG

increases in different mammalian organs with age [26] see DNA damage theory of aging. Male infertility[edit] Exposure of spermatozoa to oxidative stress is a major causative agent of male infertility. But under oxidative stress conditions, excessive ROS can damage cellular proteins, lipids and DNA, leading to fatal lesions in cell that contribute to carcinogenesis. Cancer cells exhibit greater ROS stress than normal cells do, partly due to oncogenic stimulation, increased metabolic activity and mitochondrial malfunction. ROS is a double-edged sword. On one hand, at low levels, ROS facilitates cancer cell survival since cell-cycle progression driven by growth factors and receptor tyrosine kinases RTK require ROS for activation [31] and chronic inflammation, a major mediator of cancer, is regulated by ROS. On the other hand, a high level of ROS can suppress tumor growth through the sustained activation of cell-cycle inhibitor [32] [33] and induction of cell death as well as senescence by damaging macromolecules. In fact, most of the chemotherapeutic and radiotherapeutic agents kill cancer cells by augmenting ROS stress. Modest levels of ROS are required for cancer cells to survive, whereas excessive levels kill them. As a result, production of NADPH is greatly enhanced, which functions as a cofactor to provide reducing power in many enzymatic reactions for macromolecular biosynthesis and at the same time rescuing the cells from excessive ROS produced during rapid proliferation. The resulting genomic instability directly contributes to carcinogenesis. Both exogenous and endogenous ROS have been shown to enhance proliferation of cancer cells. The role of ROS in promoting tumor proliferation is further supported by the observation that agents with potential to inhibit ROS generation can also inhibit cancer cell proliferation. Excessive ROS can induce apoptosis through both the extrinsic and intrinsic pathways. DNA damage, oxidative stress, and loss of mitochondrial membrane potential lead to the release of the pro-apoptotic proteins mentioned above stimulating apoptosis. Autophagy can be induced by ROS levels through many different pathways in the cell in an attempt to dispose of harmful organelles and prevent damage, such as carcinogens, without inducing apoptosis. When this type of cell death occurs, an increase or loss of control of autophagy regulating genes is commonly co-observed. Autophagy and apoptosis are two different cell death mechanisms brought on by high levels of ROS in the cells, however; autophagy and apoptosis rarely act through strictly independent pathways. There is a clear connection between ROS and autophagy and a correlation seen between excessive amounts of ROS leading to apoptosis. When mitochondria are damaged and begin to release ROS, autophagy is initiated to dispose of the damaging organelle. If a drug targets mitochondria and creates ROS, autophagy may dispose of so many mitochondria and other damaged organelles that the cell is no longer viable. The extensive amount of ROS and mitochondrial damage may also signal for apoptosis. This crosstalk and connection between autophagy and apoptosis could be a mechanism targeted by cancer therapies or used in combination therapies for highly resistant cancers. Cancer cells with elevated ROS levels depend heavily on the antioxidant defense system. The result is an overall increase in endogenous ROS, which when above a cellular tolerability threshold, may induce cell death. Radiotherapy also relies on ROS toxicity to eradicate tumor cells. However, modulation of ROS signaling alone seems not to be an ideal approach due to adaptation of cancer cells to ROS stress, redundant pathways for supporting cancer growth and toxicity from ROS-generating anticancer drugs. Combinations of ROS-generating drugs with pharmaceuticals that can break the redox adaptation could be a better strategy for enhancing cancer cell cytotoxicity.

Chapter 7 : Understanding Free Radicals and Antioxidants

But as we get older, free radicals can cause significant distress on the body's homeostatic state. Basically, it causes things to go "out of whack", and it works like a snowball effect in the sense that the damage becomes worse and worse.

Antioxidants and Free radicals Antioxidants are intimately involved in the prevention of cellular damage -- the common pathway for cancer, aging, and a variety of diseases. The scientific community has begun to unveil some of the mysteries surrounding this topic, and the media has begun whetting our thirst for knowledge. The purpose of this article is to serve as a beginners guide to what antioxidants are and to briefly review their role in exercise and general health. What follows is only the tip of the iceberg in this dynamic and interesting subject. Once formed these highly reactive radicals can start a chain reaction, like dominoes. Their chief danger comes from the damage they can do when they react with important cellular components such as DNA, or the cell membrane. Cells may function poorly or die if this occurs. To prevent free radical damage the body has a defense system of antioxidants. Antioxidants are molecules which can safely interact with free radicals and terminate the chain reaction before vital molecules are damaged. Although there are several enzyme systems within the body that scavenge free radicals, the principle micronutrient vitamin antioxidants are vitamin E, beta-carotene, and vitamin C. The body cannot manufacture these micronutrients so they must be supplied in the diet. A fat soluble vitamin present in nuts, seeds, vegetable and fish oils, whole grains esp. Ascorbic acid is a water soluble vitamin present in citrus fruits and juices, green peppers, cabbage, spinach, broccoli, kale, cantaloupe, kiwi, and strawberries. The RDA is 60 mg per day. Intake above mg may be associated with adverse side effects in some individuals. Beta-carotene is a precursor to vitamin A retinol and is present in liver, egg yolk, milk, butter, spinach, carrots, squash, broccoli, yams, tomato, cantaloupe, peaches, and grains. Because beta-carotene is converted to vitamin A by the body there is no set requirement. Vitamin A has no antioxidant properties and can be quite toxic when taken in excess. Preventing cancer and heart disease -- do antioxidants help? Epidemiologic observations show lower cancer rates in people whose diets are rich in fruits and vegetables. This has lead to the theory that these diets contain substances, possibly antioxidants, which protect against the development of cancer. There is currently intense scientific investigation into this topic. Thus far, none of the large, well designed studies have shown that dietary supplementation with extra antioxidants reduces the risk of developing cancer. In fact one study demonstrated an increased risk of lung cancer in male smokers who took antioxidants vs. Whether this effect was from the antioxidants is unknown but it does raise the issue that antioxidants may be harmful under certain conditions. Antioxidants are also thought to have a role in slowing the aging process and preventing heart disease and strokes, but the data is still inconclusive. Therefore from a public health perspective it is premature to make recommendations regarding antioxidant supplements and disease prevention. New data from ongoing studies will be available in the next few years and will shed more light on this constantly evolving area. Perhaps the best advice, which comes from several authorities in cancer prevention, is to eat 5 servings of fruit or vegetables per day. Exercise and oxidative damage Endurance exercise can increase oxygen utilization from 10 to 20 times over the resting state. This greatly increases the generation of free radicals, prompting concern about enhanced damage to muscles and other tissues. The question that arises is, how effectively can athletes defend against the increased free radicals resulting from exercise? Do athletes need to take extra antioxidants? Because it is not possible to directly measure free radicals in the body, scientists have approached this question by measuring the by-products that result from free radical reactions. If the generation of free radicals exceeds the antioxidant defenses then one would expect to see more of these by-products. These measurements have been performed in athletes under a variety of conditions. Several interesting concepts have emerged from these types of experimental studies. Regular physical exercise enhances the antioxidant defense system and protects against exercise induced free radical damage. This is an important finding because it shows how smart the body is about adapting to the demands of exercise. These changes occur slowly over time and appear to parallel other adaptations to exercise. On the other hand, intense exercise in untrained individuals overwhelms defenses resulting in increased free radical damage. Thus, the "weekend warrior" who

is predominantly sedentary during the week but engages in vigorous bouts of exercise during the weekend may be doing more harm than good. To this end there are many factors which may determine whether exercise induced free radical damage occurs, including degree of conditioning of the athlete, intensity of exercise, and diet. Can antioxidant supplements prevent exercise induced damage or enhance recovery from exercise? Although it is well known that vitamin deficiencies can create difficulties in training and recovery, the role of antioxidant supplementation in a well nourished athlete is controversial. The experimental studies are often conflicting and conclusions are difficult to reach. Nevertheless, most of the data suggest that increased intake of vitamin E is protective against exercise induced oxidative damage. It is hypothesized that vitamin E is also involved in the recovery process following exercise. Currently, the amount of vitamin E needed to produce these effects is unknown. The diet may supply enough vitamin E in most athletes, but some may require supplementation. There is no firm data to support the use of increased amounts of the other antioxidants. Performance In general, antioxidant supplements have not been shown to be useful as performance enhancers. The one exception to this is vitamin E which has been shown to be useful in athletes exercising at high altitudes. A placebo controlled study done on mountaineers demonstrated less free radical damage and decline in anaerobic threshold in those athletes supplemented with vitamin E. Although difficult to generalize, this finding suggests that supplementation with vitamin E might be beneficial in those triathletes who are adapting to higher elevations. How much is enough? Although there is little doubt that antioxidants are a necessary component for good health, no one knows if supplements should be taken and, if so, how much. Antioxidants supplements were once thought to be harmless but increasingly we are becoming aware of interactions and potential toxicity. It is interesting to note that, in the normal concentrations found in the body, vitamin C and beta-carotene are antioxidants; but at higher concentrations they are pro-oxidants and, thus, harmful. Also, very little is known about the long term consequences of megadoses of antioxidants. Taking chemicals without a complete understanding of all of their effects may disrupt this balance. Recommendations Follow a balanced training program that emphasizes regular exercise and eat 5 servings of fruit or vegetables per day. This will ensure that you are developing your inherent antioxidant systems and that your diet is providing the necessary components. Weekend warriors should strongly consider a more balanced approach to exercise. Failing that, consider supplementation. For extremely demanding races such as an ultradistance event , or when adapting to high altitude, consider taking a vitamin E supplement to IU, approximately 10 times the RDA per day for several weeks up to and following the race. Look for upcoming FDA recommendations, but be wary of advertising and media hype. Increased blood antioxidant systems of runners in response to training load. Journal of Nutrition 3 suppl: Medicine and Science in Sports and Exercise. Prospects for the use of antioxidant therapies.

Chapter 8 : Avoid Free Radicals

Free radicals are unstable molecules that can damage the cells in your body. They form when atoms or molecules gain or lose electrons. They often occur as the result of normal metabolic processes.

Free radicals are the natural by-products of chemical processes, such as metabolic process. The Threat of Free Radicals According to Rice University, as soon as complimentary radicals are formed, a chain reaction can occur. The first complimentary radical pulls an electron from a particle, which destabilizes the molecule and turns it into a complimentary radical. That particle then takes an electron from another particle, destabilizing it and tuning it into a complimentary radical. This cause and effect can ultimately interfere with and harm the whole cell. The complimentary extreme chain reaction may result in broken cell membranes, which can alter what enters and exits the cell, according to the Harvard School of Public Health. The chain reaction may change the structure of a lipid, making it more likely to become caught in an artery. The harmed molecules might alter and grow growths. Or, the cascading damage may change DNA code. Oxidative stress occurs when there are a lot of totally free radicals and too much cellular damage. Oxidative stress is connected with damage of proteins, lipids and nucleic acids, according to an article in the Pharmacognosy Review. Free radicals are also associated with aging. Symptoms of Oxidative Stress According to a short article in Methods of Molecular Biology, there are no officially recognized symptoms of oxidative stress. Testing for Free Radicals It is not possible to straight measure the quantity of complimentary radicals in the body, according to Rice University. According to a short article in the American Journal of Clinical Nutrition, there are indirect methods of measuring oxidative stress, normally involving analysis of the byproducts of lipid peroxidation. Antioxidants and Complimentary Radicals Antioxidants keep complimentary radicals in check. Anti-oxidants are particles in cells that prevent complimentary radicals from taking electrons and causing damage. Antioxidants are natural substances whose task is to tidy up free radicals. Much like fiber cleans up waste products in the intestinal tracts, antioxidants tidy up the complimentary extreme waste in the cells. Popular antioxidants include beta-carotene and other carotenoids, lutein, resveratrol, vitamin C, vitamin E, lycopene and other phytonutrients. Our body produces some antioxidants on its own, however an inadequate amount. Oxidative stress takes place when there is an imbalance of totally free radicals and antioxidants a lot of complimentary radicals and too few anti-oxidants , according to the Pharmacognosy Review. Anti-oxidants can be gotten through diet. During the subsequent years, researchers have performed lots of studies on the results of anti-oxidants with combined results. Wright offered a few examples. On the other hand, Wright pointed out that a beta-carotene trial among Finnish men who were heavy cigarette smokers discovered an increase in lung cancer among those taking beta-carotene supplements. Scientists do not entirely comprehend the mixed arise from the trials or the precise mechanism that makes antioxidants effective or ineffective against free radicals, but according to Wright, the study results suggest that it is more effective and potentially safer to obtain anti-oxidants through whole foods rather than supplements. Free Radicals and Exercise According to a short article in Biochemical Society Transactions, intense aerobic exercise can induce oxidative stress. Burning fuel in high-intensity cardio exercise causes chemical reactions that make complimentary radicals form at a much faster rate. According to a post in the American Journal of Clinical Nutrition, frequent workout training appears to reduce the oxidative stress at first brought on by workout. This is because regular physical exercise enhances antioxidant defenses. Stimulated by the concern that intense workout could cause oxidative stress, several studies were conducted to look at the results of antioxidant supplementation for athletes. The American Journal of Clinical Nutrition article stated that supplementing high strength exercise with antioxidant supplements produced no useful results, nevertheless. Routine workout alone sufficed to develop antioxidant defenses against the preliminary exercise-induced oxidative stress. For that reason, out of shape and irregular exercisers who do a spontaneous bout of intense exercise might invoke oxidative stress, while those who are regularly active need to not stress.

Chapter 9 : Free radical | Define Free radical at calendrierdelascience.com

To prevent free radical damage the body has a defense system of antioxidants. Antioxidants are molecules which can safely interact with free radicals and terminate the chain reaction before vital molecules are damaged.

Free radicals atoms with unpaired electrons damage the molecules of our cells by stripping away electrons in a chain reaction of oxidative electron stealing damage that continues until antioxidants stop that chain reaction. We get old because we get damaged at a molecular level and cellular level, molecule by molecule, cell by cell. A scientific way to look at this process is to think of it as rusting which is oxidizing. Hence, the current health circle buzzword â€" antioxidant, meaning to stop oxidizing. So, whatever oxidizes our cells is bad. The normal byproducts of energy production in our body with burning sugar doing twice as much damage as burning fat. Heavy metals, especially dental metals and cadmium in cigarette smoke. Chemical compounds we breathe, eat or drink. This includes chlorine, pesticides, solvents and much more. We can think of oxidation in terms of oxidative hits, or free radical attacks, kind of like bullets being fired at our cells that cause a chain reaction of destruction until stopped by an antioxidant. If a bullet free radical damages only a cell wall, that is one kind of damage, if it damages cell protoplasm that is another kind, mitochondrial damage is another, and if free radical damages reaches to the DNA of our cells that is the worst kind of damage because DNA damage permanently ages us. The reason that damage to DNA is so much worse is that DNA contains the genetic code for replacement cells that take the place of old cells every two to five months. The DNA code is extremely complex. Slight changes to it render new cells incapable of functioning optimally. Slight changes to DNA even create cancer. About two hundred thousand free radical attacks against each and every cell of our body occur every second. Many of these attacks are quickly stopped by the antioxidant armies in our bodies. However, obviously, if we could slow down the continuous onslaught of free radical damage, we could live much longer lives and healthier lives. We need to act now to stop free radical damage by choosing what we eat and otherwise let into our bodies. Even those of us who fully understand the danger that free radicals represent, often fail to take action to defend ourselves from free radicals. Be assured that free radicals do not stop damaging us just because we have good intentions to do something eventually to stop them. We have to do take action. And, we need to do it now! The most important of all the ways that we stop free radicals is to avoid them as much as possible. Every free radical we do not allow into our body means that we need fewer antioxidants to protect our cells and molecules from free radical damage. The most important ways to avoid free radical damage are to: Supplement your diet with antioxidants Seven Essentials , Earthing and Glutathione Enhancers are your top antioxidant choices. Eat natural food which has far less free radical potential and contains numerous antioxidants and antioxidant components Drink pure, unchlorinated water chlorine is an oxidant Breathe fresh, clean air cigarette smoking is a fundamental violation of an effort to avoid free radicals Avoid all use of dental metals except titanium. Take ETS Zeolite daily to remove pesticides and metals daily from your body. Keep our internal systems free of microorganisms and parasites. Use the Eight Day Cleanse once a year to remove these. Since it is inevitable that free radicals will be created in our bodies and will enter because of the polluted world in which we live, we also need to defend ourselves from free radicals by increasing the numbers of antioxidants in our body. This is the role that optimum nutrition plays. It is also a role of good sleeping patterns, since sleep is the most effective time for our bodies to create antioxidants.