AGEING, CANCER AND FREE RADICALS

by Prof. Serge Jurasunas, Lisboa

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He is the author of several works and numerous scientific publications, and has just published an important work in Portuguese "**REVOLUTION IN HEALTH**", which introduces and comments on the last technics and theories in molecular biology, nutrition and modern society diseases.

In Lisbon, he runs a Centre where several and modern tests are used, allowing the evaluation and control of the processes concerning oxidation, immune functions, nutritional deficiencies and prevention

The ageing phenomenon has always attracted the attention of the biologists. Nowadays we are starting to understand better certain mechanisms of the human body which are liable to be connected to this phenomenon. Simultaneously, the question of essential nutrients, the anti-oxidants, is closely connected to our defence system. Certain pioneers and researchers, such as Paul Bragg or Bernard Jensen have always associated longevity to an adequate food diet, and from the professional point of view, I have been following and testing similar theories.
We'll try to analyse and understand, from a personal point of view and at the same time scientific, this so Important factor in modem society;

"AGEING AND CANCER"
We will verify, in an increasing manner, that these two problems are inter-connected, associated to parallel causes that in certain individuals will lead the organism to age and in others to get cancer.
Two vitally important factors are pointed out by the latest discoveries:

1. The oxidation and free radicals theory
2. The importance of certain genomes, which are more resistant than others, that may or may not protect us against old age and diseases.

Faced with the theory of free radicals and the dissimilarity of human beings against a disease, researchers have kept trying through experiences, to understand the ageing phenomenon. However, a curious experience with flies, conducted by Michael Rose from the University of Irvine, should be noted

- Flies with higher longevity were selected, generation after generation, until flies with twice the longevity (life expectancy) of the normal flies were obtained.

- Other important factor to be noted is that these flies, are more robust, have a higher resistance to stress (a great producer of free radicals) and even when they get older, they are much more resistant than others, which are younger, from ordinary groups.

Impressed with this experience, another French researcher had the idea to place the more resistant group of flies in a richer-oxygen atmosphere.
The group of normal flies aged two times faster than the group of resistant flies.

If we admit that cancer is an “old age” disease and that old age is related to internal biological factors, we have to conclude that biological age of the human beings may be altered and may not correspond to the aged attributed by dates. I have carried out some studies on this subject, from which I am presenting some conclusions in the chapters dedicated to cancer, in my last book. But not everything is that simple and several biologists have abandoned a long time ago, the only and simplest ageing theory, to privilege several mechanisms many times controlled by genes. These mechanisms are connected to the defence and repair systems, that abound in the cellular arrangement.
Biologists have been concentrating their researches for a long time at the DNA cellular level, as the probable cause for this mutation.
And lately, modem research suggest that mitochondrion cellular DNA (MTDNA) is probably linked to the probable cause of cellular anarchy, cancer and ageing
In fact, several researchers, such as Dr. Seeger, had openly suggested this theory. And the free radical study plus the respective pathologies take once more the mitochondrion path.

If certain biologists recognised the error factors, added up in the nucleic DNA, the same did not happen regarding the same errors in MTDNA, which were neglected.
In 1979, I was in contact, in the USA, by the American researcher, Bruce Halstead M.D, who at the time had already surmised the role of free radicals and their contribution in degenerative processes leading to ageing through mitochondrion,

Nowadays it has been proved that mitochondrion are extremely fragile, susceptive to genetic and chemical defects, which may cause cellular anomalies. The consequences result in organic weaknesses, the fatigue syndrome, passing through a number of diseases, including cancer.

We understand that the cellular nucleic DNA is much more resistant and despite the constant attacks from free radicals (more than 10.000 a day) the amino acids sequences are kept correct. On the other hand the MTDNA oxidises faster, and what is more serious, it does not possess enzymatic repair mechanics. Furthermore, it is confirmed that mitochondrion themselves are the main cellular source of free radicals, and therefore more vulnerable. What is even more serious is that MTDNA nucleus, in opposition to cellular DNA do not possess histones - proteins that fix themselves to the nuclear DNA and protect against free radicals attacks. We know that free radicals can destroy the genetic material of cellular DNA by electrocution.
Well visible deformities are observed at the electronic microscope (cross linking), fractures, loss of parts from the DNA helix stem which would be reproduced and amplified along the cellular growth.
The strongest consequence to such reactions, at the MTDNA level, may cause disturbances in the cellular breathing which can cause death of tissues. The mitochondria mechanism, which produce the necessary energy (A.T.P.) for the proteins, enzymes and hormones synthesis, also produces free radicals. One of them, the radical superoxyde OH2 which turns itself into hydrogen peroxide (H2O2)

thus forming an extremely aggressive free radical hydroxyl (OH2) which causes diseases such as rheumatoid arthritis and cancer.
These free radicals in the mitochondrion, damage the cellular constituents, that is, proteins and lipids They also contribute to destabilise cellular communications, causing biochemical disorders and also may cause pathologies. For example, recent researches showed the existing relation between the Parkinson disease and free radicals.
When studying the brain of patients that had died, the footprint of free radicals was found in them, at the same time it was also noted a very low level of antioxidants.
Researchers also noted a reduction in the activity of an enzyme, known as complex I, contained in the mitochondrion of affected neurones. As soon as complex I fails, the production of minimum energy decreases, free radicals level rises and at the same time antioxidants decrease.

Therefore, it is quite clear that an excessive production of free radicals with a simultaneous drop in the production of endogenic oxidative enzymes and the lack of macromolecular antioxidants leads, unavoidably to deterioration and degeneration factors.
Under normal conditions the organism has a defence barrier against free radicals and the lipids oxidation, especially of the enzyme superoxyde dismutase (S.O.D.) which I will call "super enzyme". The S.O.D. is in fact one of the most important components of the human oxidative defence system. It was proved that human tissue and tissue from other older living species, in average produce more superoxyde dismutase and are more resistant to oxidation.

Several research teams dedicated themselves to the study of the relationship between ageing and the action of free radicals on mitochondrion and also its relationship to the production of dismutase superoxyde.
By observing certain animals, such as cows, pigs or flies we conclude that ageing is linked by an abnormal level of free radicals in mitochondrion. It is also seen that proteins and MTDNA suffer oxidative attacks due to previously explained reasons.
In the human beings, it seems that there are certain groups of individuals that possess less dismutase superoxyde than others, and who are therefore, more susceptive to get older faster or to develop diseases of the degenerative type. For example, patients suffering from lateral amiotropic sclerosis have an abnormal gene that reduces S.O.D. activity by 40%.
This detail could also explain the reason why certain women, due to cancerigenous substances develop breast cancer faster than others. In fact, the higher the absence or lack of S.O.D. the more sensitive the cells become to cancerigenous substances.
Cancerigenous factors tend to disturb the P53 protein, which provides protection against cancer. Thus, 60% of women who do not have active P53, S.O.D. will be the protection protein.
Furthermore, cellular antioxidant systems go through alterations along the carcinogenesis, especially S.O.D., whose levels become especially low in different cells. Chinese scientists analysed S.O.D.

activity from the blood plasma of 151 cancer patients, who were divided into 5 groups:

1. Lung cancer
2. Digestive system cancer
3. Breast cancer
4. Gynaecologic cancer
5. Other types of cancer

The S.O.D. level contained in each groups' blood plasma was higher than in a control group of healthy individuals. This essentially means that S.O.D. actively participates in the fight against cancer. But 31 of the patients presented a especially low level of S.O.D. It was found out that contrary to others, these patients had been submitted to chemo and radiotherapy sessions. It is admitted that heavy treatments have damaging effects in the S.O.D. production level.

In the lung cancer group there was a significant inhibition of S.O.D. which caused Chinese scientists to conclude that S.O.D. plays an important role in the destruction of lung tumour cells. According to these researchers, in lung cancer, to evaluate the S.O.D. level in the blood is an important method of diagnosis.

It would quite interesting to introduce S.O.D. Cu-Zn, at the level of cancer tumour.

Several teams have been working for some time on this therapeutic hypothesis that presents great problems due to the difficulty in penetrating these cells, which reduces the therapeutic capacities. It would not be a treatment to destroy the tumour cells but to differentiate them

S.O.D. has a relatively short circulatory life time. One assumes that an oral or injection S O D Cu-Zn administration was used.

One of the work teams wanted to check it using a lower molecule imitating the S.O.D. (Cu Dips) activity in mice, to whom the Ehrlich ascites carcinoma had been transplanted.

In this case, there was a survival percentage of 85% to 140%.

Basically, CuDips acted positively, either by inhibiting the cellular division or in the increase, in vitro, of the cellular differentiation in mice with neuroblastomatic cells.

An antioxidants cocktail has been successfully used, such as the S.O.D. and the glutathione, in injection, especially in the mammary gland carcinomas. It may also be used in perfusion. which is more effective, but requiring a greater medical surveillance in clinics or hospital environments

Recently, a kind of metallic rock, the S.G.E.S., has been used, which acts in a similar way to S.O.D. with remarkable results. A quite fast inhibition of the (mammary) tumour occurs. The S.G.E.S. reduces the lipid oxidations and presents a similar therapeutic spectrum to S.O.D. The Japanese Niwa greatly contributed to the research and study of S.G.E.S. use in the treatment of cancer.

S.O.D. is equally interesting to deactivate saturated fat before they penetrate in the organism through the intestines This prevents peroxidations at the membrane level, a task normally carried out by vitamin

E, whenever the blood content is normal. Vitamin E captures lipid peroxides at the membrane level and biologists perceived that S.O.D. has a structure which is comparable to vitamin E.

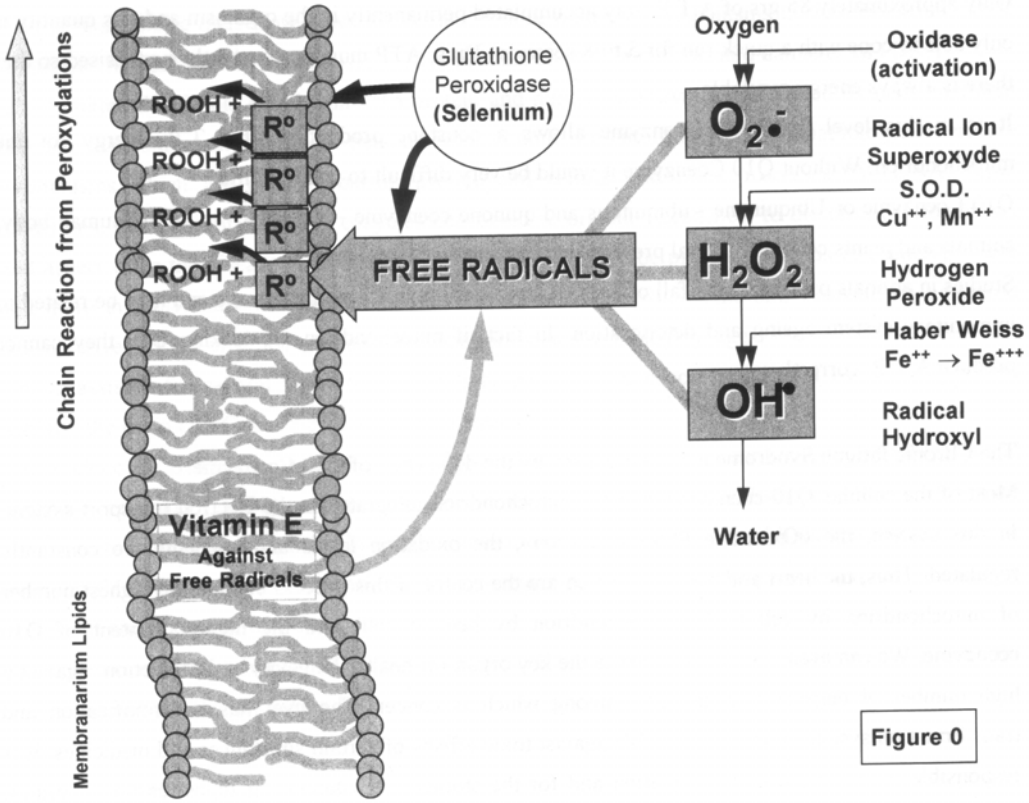


Figure 0

For example, Prof Esterbaner from Austria proved that L.D.L. with 6 to 8 vitamin E molecules is able to resist, in vitro, to oxidation for 60 to 80 minutes.

Vitamin E has an important role due to the possibility of interfering in the lipid membrane oxidation, increasing at the same time the action of vitamin C. In patients with lung cancer, even before the diagnosis definition, it is already noted low vitamin E blood concentrations.

Recent studies support and prove that vitamin E emphasises the anti-neoplasic effect of selenium, especially against mammary gland tumours.

Recently, Bruce Ames, molecular biologist in the University of Berkeley, California, showed evidence about one other powerful antioxidant and LIPOFILE - Q10 Coenzyme. Q10 Coenzyme, also called Ubiquinone, is the main enzyme in the mitochondrial activity for the production of Adenosine Triphosphate (A.T.P.).

A.T.P. consists of an adenine and ribose molecule called adenosine, combined with three phosphate atoms and oxygen. A great quantity of energy is stored in the A.T.P. molecules. This energy is next released in the organism in the form of calorie. Even being used as the energetic current to all cells, the A.T.P. quantity is limited.

Only approximately 85g of A.T.P. stay accumulated permanently in the organism and this quantity is only able to cope with a quick run for 5 to 8 seconds. Thus, ATP must be constantly synthesised so that there is always energy available.
It is in this level that Q10 Coenzyme allows a constant production of A.T.P. energy for the mitochondrion. Without Q10 Coenzyme it would be very difficult to maintain life.
Q10 Coenzyme or Ubiquinone - ubiquitous and quinone coenzyme - are found in all the human body, animals and plants cells. Its natural presence in plants is the platquinone.
Studies in animals prove that the fall of the Q10 coenzyme level accompanies age and may be related to immune system ageing and deterioration. In fact, if mitochondrion are lacking Q10 they cannot produce A.T.P. correctly.

The Chronic fatigue Syndrome is in part caused by the deficiency of Q10 Coenzyme.
Most of the cellular Q10 coenzyme is in the mitochondrion integrated in the electron transport system. In this system, the «Oxidative Phosphorylation», the oxidation levels and nutrients are constantly regulated. Thus, the heart and the liver, which are the centre of this process contain the highest number of mitochondrion by cell (4000 mitochondrion by hepatic cell) and the highest content of Q10 coenzyme. We can even say that the liver is the key organ for health, longevity and protection against a high number of degenerative diseases, among which is cancer. The liver has a detoxification and transformation system (Cytochrome P 150) against toxic effects of certain substances and medicines. It is responsible for the nutrients assimilation and for the storage of vitamins. It digests carbon hydrates, releases glycogen and next maintains sugar level in the blood. It manufactures enzymes, cholesterol, proteins and blood coagulation factors. And we are only citing some examples. Excessive consumption of medicine on weakened livers, or in old people is a factor that is able to hasten a drop in the hepatic functions and cause pathologies and premature ageing.
Q10 Coenzyme operates in the immune system deficiencies and pathologies connected to ageing.

The presence of Q10 Coenzyme in each of our cells depends in 85% of the internal biosynthesis and 15% of the nutritional part. The ultra complex endogen production is disturbed by multiple factors among which are, cellular ageing, disease, stress and pollution.
Bruce Ames compares Q10 coenzyme to a powerful antioxidant, and he believes in a co-relation between it and the prevention of mortal ageing diseases.

More recently, random tests prove that Q10 Coenzyme, in 390mg daily doses, inhibits cancer tumours and in particular mammary tumour. It is clear that in all situations, nourishment has an important role, I would even say, vital, parallel to a correct digestion.
The excess of animal proteins, against which I have fought for several years, privileging a lacto vegetarian regime, proves to be correct. Several recent studies have connected different food regimes with free radicals.

Certain hypocaloric diets slow down ageing and reduce oxidative mechanisms in mitochondrion, while the normal modem society food causes a great production of free radicals and damages MTDNA.

It is admitted that in old people, half the protein and numerous enzymes are altered by non-functional reactions and oxidation. In an old person, even without presenting any precise pathology, there are mechanisms which are out of control, protein and cellular oxidation processes (MTDNA), lipid oxidations, reduction of oxidative enzymes concerned to organic decadence, to the physical conditions and aspect or to the disease. These abnormal mechanisms may be present, and this is occurring more and more in individuals whose ages vary between thirty and forty years old, showing an advanced degenerative process in which civil age does not correspond to biological age.

I have assisted for several years to a slow liver degenerative process, followed by the kidney and actually the brain (AVC that increases in the 35 years old age group).

The work of R. Sohal and from his collaborators proves that concentrations of superoxyde radical ($O2^0$) and hydrogen peroxide (H_2O_2) in mitochondrion removed from the brain, liver and kidneys of mice submitted to hypercaloric regimes. We may find in this fact some analogies with the experiences carried out with Parkinson patients that showed the consequences of an inadequate nourishment, too rich in animal fat and poor in antioxidants.

The decadence of the brain capacities which affects all the population , especially students, proves the need to correct our dietary habits.

Fig. 1

Good red blood corpuscles - excellent negative charge - good balance of endogenous and exogenous antioxidants

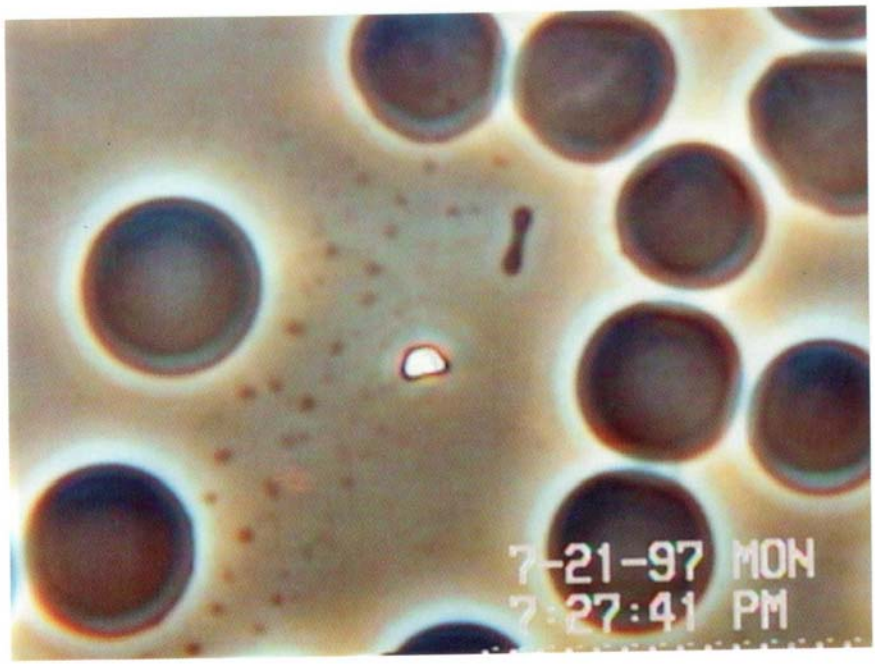


Fig. 2

Inactive and morphologically deformed red corpuscles. A very common situation in old people.
Reduction of endogenous antioxidants - lack of vitamins A, C, E, B6, B12, folic acid, iron, etc.

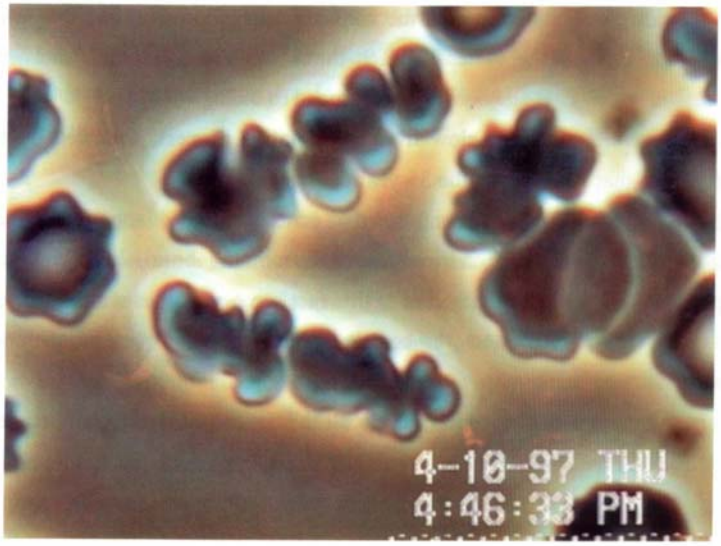


Fig. 3

Agglutination red corpuscles (rolled). We can also see spider webs (spiculas) showing an hepatic stress. Excess of lipids, viscous blood and lack of cellular energy. A very low level of oxygen.

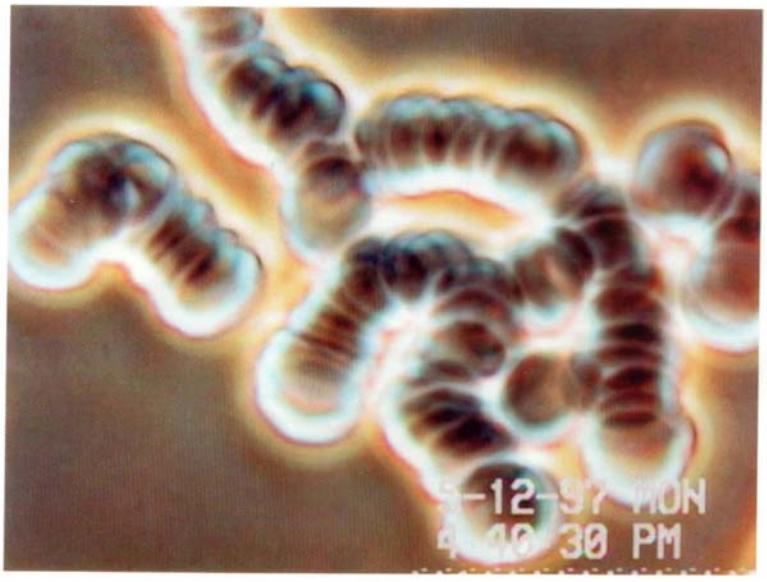
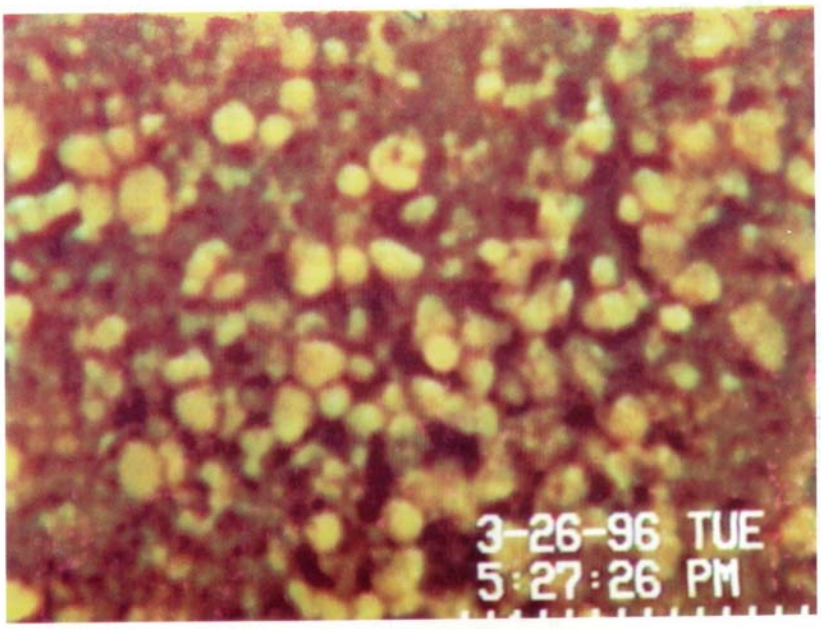
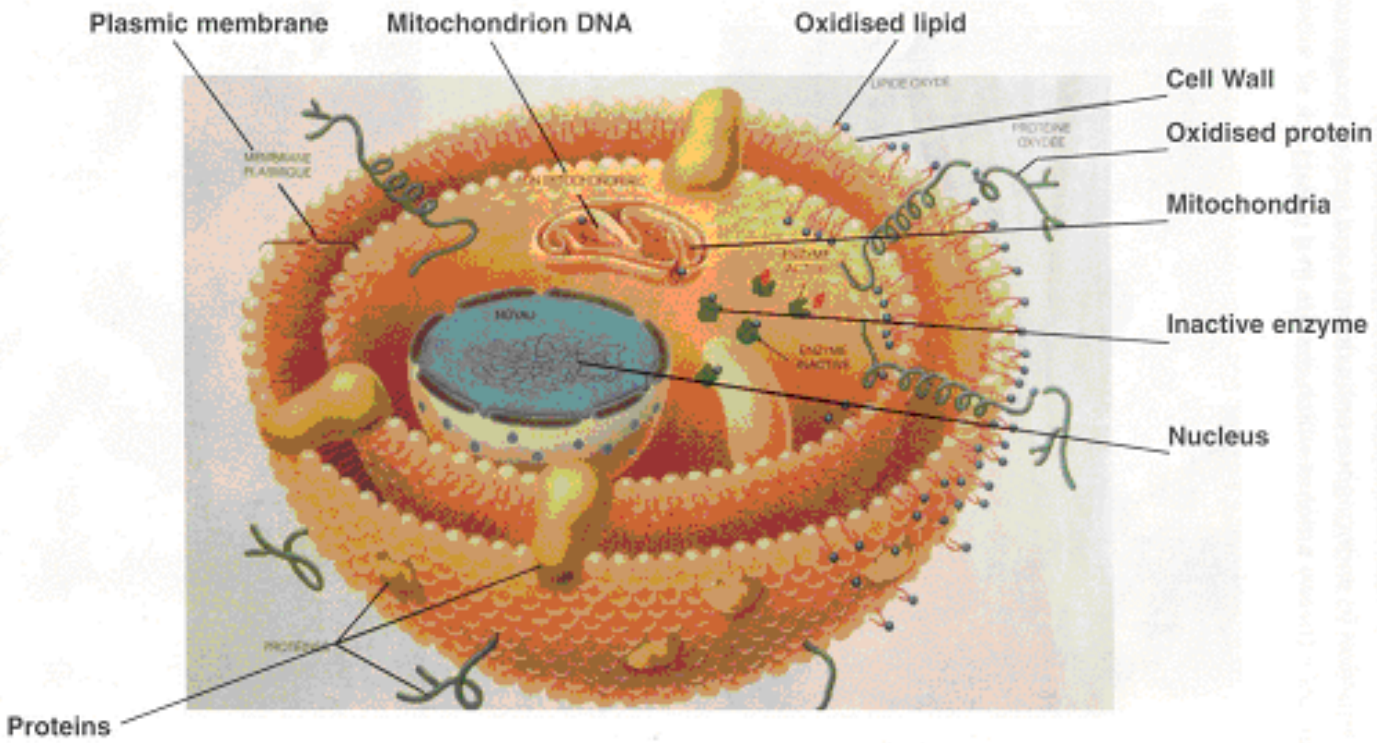


Fig. 4

Lipid peroxidation with Candidas agglutination. Excess of animal fats and Omega 3 and 6 deficiency. Reduction in endogenous antioxidants and lack of exogenous antioxidants (heavy smoker which results in a great lack of vitamin C).



DAMAGES CAUSED BY FREE RADICALS



With time damaged cell will create sister cells that lose their capacity to produce ATP energy. They will start to get old faster towards a premature ageing, pathologies and cancer.

ANTI-RADICAL TESTS

Faced with the new theories, it is necessary to verify the "oxidation" onditions of the individuals and not only their pathologies. In the United States, Bruce Ames is working on a system that allows the elaboration of the patient's lipid profile.

He is building a device to measure sialidase oxidation caused by inflammatory conditions.

There are also laboratory tests to evaluate radical conditions:

- **Lipid oxidation**
- **Protein oxidation**
- **Enzyme deactivation**
- **Endogenous antioxidants reduction**

We also have been using for more than fifteen years very precise oxidative and molecular tests, which are able to provide the individual profile, concerning the conditions connected to lipid peroxidations, reduction of exogenous antioxidants, immune functions and morphology of blood cells.

One of these tests is called L.B.M.A. (Live Blood Microscopy Analysis) which provides a study of live blood, due to variable projection microscope with high resolution and amplification (B.V.P.M.), manufactured in the United States of America.

DEFENSE SYSTEMS
AGAINST OXIDATION REACTIONS

Endogenous antioxidants*		**Enzymes ----- Dismutase superoxyde* transforms superoxyde radical (O2) into hydrogen peroxyde (H2O2).**
(neutralize free radicals or limit its activity)	**Glutathione* Peroxydades* and Catalases***	**Transform hydrogen peroxide into water and in molecular oxygen.**
Vitamin E* and betacaroten*		**React with free radicals preventing them from attacking cellular constituents. Soluble in fat, protect membranes.**
Vitamin C* and uric acid		**react with free radicals in cytoplasm**

***Contained in Zell-Oxygen®**

REPAIR SYSTEMS

Proteins Repair

- **Proteinase – divide oxidised proteins**
- **Proetoses – divide fragments produced by proteins**
- **Peptidases – divide products resulting from proteoses activities : amino acids are then recycled to produce new proteins**

LIPIDS REPAIR

Phospholyrases – remove damaged parts from oxidised lipids so that the other enzymes may repair degraded parts

Acetyltransferases – replace acid fat eliminated by lipids. Participate in the repair of oxidised fat tissues, without eliminating important fragments from membranes. Eliminate damaged DNA segments.

Glutathione Peroxidade and Transferase

DNA Repair	**Exonuclease and Endonuclease**
Glycoliases	**Fill up spaces left by exonucleases and by endonucleases**
Ligase	**Insulates repairs**

Live cells from ferments possess besides the antioxidant enzymes, they also have enzymes to repair damaged proteins and lipids.

PROTECTING ANTIOXIDANTS OR IN CASE OF INFLAMMATION

- Vitamin C
- Vitamin E
- SOD
- Glutathion
- Ginkgo Biloba (fresh plant)
- Selenium
- Q10 Coenzyme
- Live cells from yeast (1)

An antioxidant formula is suggested, which includes all these elements. Always take SOD with a "gradual release" supplement of vitamin C, especially in case of inflammation.

(1) Zell-Oxygen®

SERIOUS INFLAMMATORY PROCESS (Painful)

- Glutathion
- L-Cysteine
- SOD
- L.Taurine
- Vitamin C

We may find an oral formula, but the i.m. injection one shows faster results.

PROTECTION AGAINST AGEING

Reduction of brain capacities, drop of memory and concentration, anxiety and sadness, brain fatigue

- Live cells from yeast (1)
- Oligopeptides
- Ginkgo Biloba (a fresh plant)
- Vitamin E (1000mg daily)
- Mucopolysacharides
- Q10 Coenzyme

(1) Zell-Oxygen®

ANTI-CANCER

- S.G.E.S.
- Biologic Flavonoids
- Molecular biologic compound (1)
- Live cells from yeast (2)
- Geoxy 132 (3)

(1) Apizellin®
(2) Zell-Oxygen®
(3) Biogermax®

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