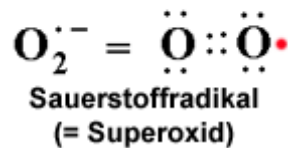
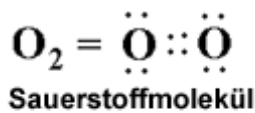


# What is oxidative stress?

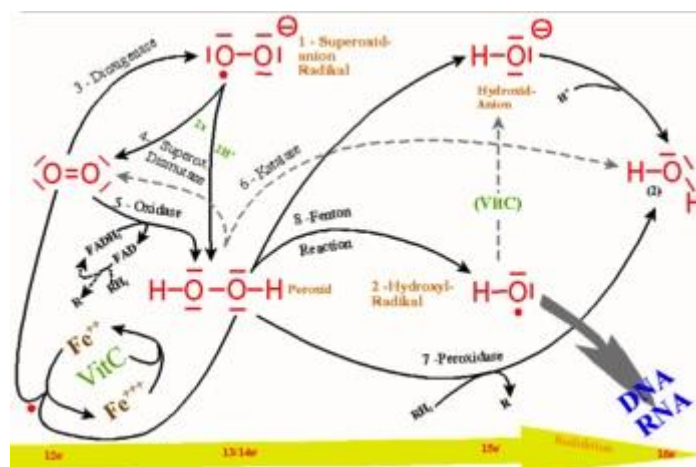
Oxidative stress is a frequently used term; unfortunately, very few people really know what it means. Oxidative stress plays a fundamental role in the development of heart attacks. Without oxidative stress heart attacks would hardly ever occur.

This section therefore deals with what is meant by oxidative stress. The next section explains its connection with heart attacks. The following text is an excerpt from my book “Herzinfarkt – Neue Wege” (45), which I have slightly altered.

Oxidative stress occurs when free radicals are able to develop unhindered. **What are “free radicals”?** Under normal circumstances, every cell in our body produces a certain small amount of oxygen-containing substances, which are highly reactive. In this case, the oxygen atom has just one unpaired electron on its outer electron coat. This oxygen atom is therefore very aggressive due to its drastic attempts to catch another electron to fill its coat. This is why they are called “free radicals”. They react with a variety of compounds that they destroy in oxidative reactions. A steady production of free radicals is part of the normal function of a healthy body.



Polyunsaturated fatty acids and DNS molecules are favourite targets to oxidative attack by these short-lived, aggressive oxygen free radicals. When a “**superoxide radical**”, a typical example of a free radical, meets a polyunsaturated fatty acid, a new radical develops from this reaction. A destructive chain reaction is set in motion during which the cell wall and the cell nucleus may be transformed into oxidative waste.



Oxidative stress in action

The organism is not helpless with regard to these radicals. It has various protective mechanisms to prevent such reactions and to make sure that “superoxide radicals” do no damage. In addition, the human body has also succeeded in turning the destructive capabilities of these radicals to its own use. “Macrophages”, so-called “killer cells”, have “radical canons”. They produce “superoxide radicals” that can destroy bacteria, viruses, parasites and diseased cells.

## **Increase in “radicals” – weakened immune system: oxidative stress**

Oxidative stress occurs when free radicals develop unhindered in an organism. This may occur when too many radicals are produced, or it may result from weakness in the immune system. **The lifestyle in industrialized countries goes hand-in-hand with a great increase in the production of radicals, and at the same time, this lifestyle undermines the body’s solid protection against oxidation.**

Auto and industrial emissions stimulate the production of radicals in the body, as do also ozone and respirable dust. Cigarette smoke, pesticides in foodstuffs, large amounts of medication, and various chemicals used in the home increase the oxidative burden. UV and x-rays lead to the production of “superoxide radicals”. **The technology of daily life is accompanied by a huge increase in the production of free radicals.**

**Luckily, the body is able to cushion the free radical burden if the immune system is intact. The first line of defence** is the blood stream, through which all poisonous substances must pass. Here, various metabolic products such as **uric acid or “HDL cholesterol” (“good” cholesterol)** have a protective function. Only when these substances are oxidised and their function as radical scavengers is exhausted, can the free radicals attack the tissues and cells.

**The second line of defence** is in the cells. Here various enzymes neutralize free radicals. A particular trace **element, either copper, zinc, manganese or selenium**, is found in the core of these enzymes. It has been found that a lack of trace elements is more serious than a large production of “superoxide radicals”. If this defence system is adequate, the lifetime of a “superoxide radical” is limited to a fraction of a second. On the other hand, if protection against oxidization is lacking, free radicals are active for some hours (46).

**If superoxide radicals remain active for a long period**, they approach the fat-containing cell walls. **The antioxidative vitamins, vitamin E, C and “beta-carotin” (“provitamin A”)** then come into play as radical scavengers. Vitamin E destroys superoxide radicals and protects the vulnerable polyunsaturated fatty acids in the cell wall against an oxidative chain reaction. **When the supply of vitamins is exhausted, nothing stands in the way of the free radicals.** The cell membranes and the various cell structures are destroyed and transformed into **oxidative waste**.

## **Diet plays a major role**

**Diet plays a prominent role in providing the body with adequate protection against oxidation.** An adequate supply of trace elements, antioxidative vitamins and “secondary plant substances” is only guaranteed by a varied diet and gentle preparation of fresh food. **Fresh food plays the primary role in preventing oxidation.**

So-called “secondary plant substances”, which are responsible for how plants look and taste, are a complex of roughly 4000 substances, whose combined antioxidative action stabilizes health (47). “Flavonoids” play a special role. There is a wealth of flavonoids to be found in herbal teas and natural fruit juices. Flavonoids can recycle oxidized vitamin C and E, thus greatly increasing their effectiveness. Only a tenth of the optimal amount of selenium, a quarter of the amount of manganese and copper, and a third of the amount of zinc required is covered in an average person’s normal mixed diet (47). This insufficient supply is the result of industrially produced and manufactured foodstuffs and diet habits in industrialized countries.

**Industrially produced food greatly lacks freshness.** Folic acid is susceptible to air. Vitamin B is susceptible to light. Designed for durability, industrially produced food products often lack important micronutrients. The body’s oxidation protection system therefore suffers.

## **The official cholesterol diet results in oxidative stress**

**Every unbalanced diet reduces our antioxidative resistance.** This applies to one-sided grain diets as well as the diets aimed at reducing levels of cholesterol that all doctors normally recommend to their patients. These dietary guidelines, which drastically reduce the consumption of butter, eggs, and meat and increase the intake of polyunsaturated fatty acids, always result in oxidative stress. Every polyunsaturated fatty acid requires a certain amount of vitamin E to protect it against oxidative attack; this diet therefore automatically leads to a depletion of vitamin E in the body (47). A lack of vitamin E heats up the oxidative fire and “LDL cholesterol” (“bad” cholesterol) is a favourite target. **The diet officially recommended by conventional medicine makes cholesterol itself susceptible to oxidation (48). Such a diet increases the risk of arteriosclerosis.** If this diet is followed consistently, the exact opposite is achieved to what was aimed at

Life in industrial societies leads to an increasing burden of free radicals. But “society” cannot be “blamed for everything”. Our individual lifestyle is just as important. Too much alcohol, too many cigarettes, too much medication, too little movement or over-exertion at sport, all lower the individual resistance against free radicals. Bad diet undermines any effective protection against oxidation. **We are not completely at the mercy of “free radicals”.**