The FRAP values of volatile oils of the knowledge of main componenst help to estimate the quality of volatile oil with 20-30% deviation. In the case of fragrance compositions with known essence style tendency, the values show the reducing power and they do not contain any other information.

It is recommended to determine the antioxidant values of all aromatic agents, volatile oils and essence-compositions beside physico-chemical characteristics.

Modification of fully activated NADPH oxidase activity by antioxidants

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The first reactive oxygen-derived substance is the superoxide anion produced by NADPH oxidase. This is a multicomponents enzyme containing several cytosolic (phox proteins) and membrane-bound (Cytochrome b558) parts. NADPH oxidase can be activated by receptor-mediated and non-receptor-mediated ways. During the activation the cytosolic components are phosphorylated and translocated to membrane, where they are combined with the membrane-bound part of Cytochome b558. Cytochrome b558 consists of two parts, gp91phox and p22phox proteins. During the last few years it became clear that this complex is not able to produce superoxide anion, it requires coupling of a Rac 1 or 2 GTPase G protein to complex. When coupling of Rac 1/2 is inhibited the NADPH oxidase enzyme could not produce superoxide anion. Now, it is well demonstrated that NADPH oxidase can be found not only in phagocytes such as neutrophils, monocytes, but many other cells as well, they are called to NOX enzymes. NOX was identified in uterus, renal cells, hepatocytes, endothelial cells, lymphocytes, smooth muscle cells etc. Those non-phagocyte NADPH oxidases (NOX1, NOX3, NOX4) differ from phagocyte one (NOX2) in the structure of gp91 phox subunit. This difference results in that non-phagocyte NADPH oxidase produces smaller amounts of superoxide anion and its activation does not require activation of PKC.

During the last years, our group studied the effects of several natural and synthetic antioxidants on superoxide anion production and activation of PKC in human neutrophils. These examinations involved the study the effects of antioxidants on superoxide anion production by fully activated NADPH oxidase (NOX2) as well. During experiments we have found that some antioxidants can decrease it, while others have not such effects. It was also demonstrated that the effects of antioxidants on fully activated NADPH oxidase was independent on PKC inhibiton; and as a consequence, independent from the modification of superoxide anion production in intact neutrophils induced by antioxidants. These differences were the most pronounced in case of tocopherols and their water soluble metabolites (CEHC). Both tocopherols and CEHC-compounds inhibited PKC and superoxide anion production in phorbol-ester stimulated neutrophils, and CEHC were more effective inhibitors as parent tocopherols. In contrast, superoxide anion production by fully activated NADPH oxidase was only decreased by lipid-soluble tocopherols.

On the basis of our observations, we suggest the following mechanism for the action of antioxidants on superoxide anion production by fully activated NADPH oxidase: the bound between the enzyme complex and the small Rac 1/2 protein - which is necessary for superoxide anion production by NOX – might be slack or broken by antioxidants, which is due to the counteraction of lipid-soluble antioxidants and cell membrane. This observation might be useful in those clinical states when activation of NADPH oxidase occurs - in either phagocyte or non-phagocyte cells-, since in these cases we can choose antioxidants which are able to decrease superoxide anion production by fully activated NADPH oxidase(s) (NOX) and prevents development of oxidative stress.

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