



Dietary antioxidants in cat and dog nutrition

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In recent years awareness has increased of the importance of oxidative damage in both the pathophysiology of disease and the aging process in all species. 'Free radicals' are usually responsible for oxidative damage: a free radical is any atom or molecule capable of independent existence that contains one or more unpaired electrons. Thus, the simplest free radical is an atom of the element hydrogen. But there many others, including superoxide, hydroxyl, thyl, and peroxy. The phrase 'reactive oxygen species' (ROS) is often used to describe all those capable of causing oxidative damage. ROS are produced continuously in mammalian systems as a consequence of normal

metabolic processes, but, if not inactivated, their chemical reactivity can cause damage to all cellular macromolecules.

The group of compounds known as antioxidants (sometimes referred to as 'free radical scavengers') are the major defence against oxidative stress and they protect membrane and cytosolic components against free radical damage. Many of the antioxidants present in mammalian systems prevent the formation of new radical species, including enzyme systems such as superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px). Other antioxidants trap radical species, preventing chain reactions; these include nutrients such as vitamin E,

vitamin C and taurine. In addition, others with an integral role in maintaining antioxidant defences include zinc, manganese, iron, selenium, and copper, all of which are involved in antioxidant enzyme systems. Many other dietary components, such as carotenoids or polyphenols, also trap radical species and can contribute to antioxidant status.

Although the complex array of mechanisms that prevent oxidative damage is impressive, the system is by no means completely efficient, and as well as the ROS generated in everyday life other factors can cause an increase in these dangerous molecules. For example, a diet rich in polyunsaturated fats (PUFA), especially the very highly unsaturated fats normally found in fish, increases the dietary requirement for antioxidants, particularly vitamin E. To accommodate this, the vitamin E requirement is increased at a rate of 1 IU per gram of PUFA.

Other non-dietary sources of oxidative stress include sunlight, pollution, anesthesia, and exercise (Table 1). A recent study has shown that an exercise bout of as little as 20 minutes can induce measurable oxidative damage in healthy, adult dogs (1). In this case, a measurable increase in plasma TBARS (an indicator of lipid peroxides) was detected two hours after exercise. These results indicate the potential for dietary lipid-phase antioxidant intervention to minimize oxidative damage in dogs undergoing regular exercise (Table 2).

The importance of oxidative stress in the pathogenesis of numerous disease processes is well documented and the list of conditions associated with it is extensive. It includes cancer, atherosclerosis, arteriosclerosis, inflammatory diseases (including rheumatoid arthritis), cataracts, age-related macular degeneration, central-nervous diseases such as Parkinson's disease, renal diseases, and asthma (2). While not all of these affect cats and dogs they serve to illustrate the point that a number of diseases normally associated with aging in pets have an oxidative stress component. Provision of dietary antioxidants which can operate both in the aqueous phase (e.g. vitamin C or taurine) and the lipid phase (e.g. vitamin E) offers the best chance of reducing the risk of oxidative damage and its associated problems.

Table 1
Some sources of free radicals

<i>Internally generated</i>	<i>External sources</i>
Mitochondria	Environmental pollution
Phagocytes	Radiation
Reactions involving transition metals	UV light
Exercise	Certain drugs, anesthetics
Inflammation	Dietary pro-oxidants
Ischemia/reperfusion	

Table 2
Plasma TBAR measurement as an indicator of oxidative stress in the dog following an acute bout of exercise

<i>Breed</i>	<i>Age (years)</i>	<i>Sex</i>	<i>Plasma TBARS pre-exercise (nmol/ml)</i>	<i>Plasma TBARS post-exercise (nmol/ml)</i>
Springer Spaniel	5	F	0.37	0.85
Poodle	7	M	0.58	0.61
Poodle	4	M	0.34	0.49
Beagle	4	F	0.74	1.23
Beagle	3	F	0.87	1.18
Beagle	4	F	1.05	1.12
Beagle	3	F	0.91	1.17
Labrador Retriever	6	F	0.76	0.80
Labrador	3	M	0.65	1.15
Labrador	3	F	0.72	0.77
Beagle	4	F	1.44	0.87
Beagle	3	F	0.64	0.96
Beagle	3	F	0.88	1.17
Beagle	3	F	0.49	0.60
Mean			0.74	0.92
Standard deviation			0.2848	0.2496

REFERENCES

1. Obra, R., Lunec, J., Harper, E. J. Exercise in dogs increases plasma TBARS – an indicator of oxidative stress. *Proceedings of the Nutritional Society* 1999; (in press).
2. Davies, K. J. A. Oxidative stress: the paradox of aerobic life. In: *Biochemistry Society Symposium* 61. London: Portland Press, 1995: 1-31.