Seletoc® (Schering); (Rx): Each mL contains selenium 1 mg (as sodium selenite) and Vitamin E 68 IU in 10 mL vials. Approved for use in dogs. Dose: Dogs: Initially, 1 mL per 20 pounds of body weight (minimum 0.25 mL; maximum 5 mL) SC, or IM in divided doses in 2 or more sites. Repeat dose at 3 day intervals until satisfactory results then switch to maintenance dose. If no response in 14 days reevaluate. Maintenance dose: 1 mL per 40 lbs body weight (minimum 0.25 mL) repeat at 3–7 day intervals (or longer) to maintain.

SELENIUM

Several factors are known to alter selenium toxicity; however, in general, a single acute oral dose of selenium in the range of 1–10 mg/kg may be lethal in most animals. Parenteral selenium products are also quite toxic, especially to young animals, and have caused deaths in piglets, calves, lambs, and dogs at dosages as low as 1 mg/kg.

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Research Article

Role of Selenium in Pets Health and Nutrition: A Review

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Abstract

Balanced nutrition is a key element to ensure a happy and healthy life. Trace minerals are very essential nutrients in animal diet. Selenium, like the other trace minerals is necessary to sustain life of canines. Selenium is one of the critical nutritional factors for immune system along with zinc, vitamin E, vitamin B_6 and linoleic acid. Adequate selenium is necessary for the normal functioning of the immune system and thyroid gland. Selenium is getting significant consideration for its role in various functions such as anticancer, joint health, skin and coat, immune resistance and antioxidant properties etc. Selenium forms the active center for selenoenzymes that carryout redox reactions such as glutathione

peroxidase (GPx), thioredoxin reductase, thyroid hormone deiodinase and so on. Animal studies have shown a beneficial effect of selenium in the prevention of cancer. Selenium deficiency has become increasingly recognized as a practical problem in animal industry. Insufficient selenium intake can cause serious health problems, including Kashin-Beck disease in human beings, which is characterized by the degeneration of the articular cartilage between joints, thyroid disease and a variety of cancers. Selenium supplementation is required to overcome the deficiency symptoms and the bioavailability of the same depends upon the nature of supplements used. It is generally found that organoselenium compounds have substantially greater bioavailability than that of inorganic selenium.

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INTRODUCTION

Selenium, like the other trace minerals is necessary to sustain life and is essential for basic physiological functions in both animals and humans. While the daily requirement for this mineral is obviously less, its importance and impact on the health and well being of livestock and humans are well documented in research. It was

discovered in 1817 by Swedish chemist (Mugesh and Singh, 2000). It was considered as a poison until identified as a micronutrient for bacteria, mammals and birds (Schwarz and Foltz, 1957). It has been found to be present in at least 15 different mammalian selenoproteins and up to seven microbial selenoenzymes so far (Sunde, 1997).

The animal body is under constant attack from **free radical**s, formed as a natural consequence of the body's normal metabolic activity and as part of the immune system's strategy for destroying invading microorganisms. It has been calculated that about $2x10^{10}$ molecules of Reactive Oxygen Species (ROS) are generated per cell per day (Chance *et al.*, 1979). These reactive species are involved in the initiation, propagation and maintenance of both acute and chronic inflammatory processes (Halliwell *et al.*, 1982; Mugesh and Singh, 2000). Ultimately this leads to aging, Alzheimer disease, inflammation and certain types of cancer. There is a need for defensive action against these reactive species. An antioxidant may be defined as any substance that when present at low concentrations, compared with those of the oxidizable substrate, significantly delays or inhibits oxidation of that substrate (Gutteridge, 1994). Antioxidants play an important role since they protect the cells from oxidative cell damage. Scientific evidence suggests that antioxidants reduce the risk for chronic diseases including cancer and heart disease. The main characteristic of an antioxidant is its ability to trap **free radical**s. Antioxidants which scavenge the active oxygen species are found in variety of food stuff. Selenium has been known to be intimately involved in the activity of enzymes such as glutathione peroxidase (Gpx) and thioredoxin reductase and protect

the biomolecules against reactive oxygen species and **free radical** damage (Nordberg and Arner, 2001).

Dietary sources are the good sources of selenium for the body and supplements are needed for the consumers living in the areas that are deficient of selenium. Selenium deficiencies have been reported to suppress the **immune response** in various species (Sheffy and Schultz, 1979). Selenium deficiency reduces T-cell dependent antibody responses, which further gets magnified with vitamin E deficiency. Several metabolic disorders have been reported due to the dietary deficiencies of selenium and vitamin E in several species like chickens (Schwarz *et al*., 1957), turkeys (Scott *et al*., 1967), rats (Schwarz and Foltz, 1957), calves, lambs with several health disorders. Current selenium supplements rely on inorganic forms such as sodium selenite (Na_2SeO_3) or sodium selenate (Na_2SeO_4). Organoselenium compounds have been found to be an alternative to inorganic selenium compounds with greater **bioavailability** (Jacob *et al*., 2004). More importantly, organic selenium is usually found to be less toxic than inorganic forms (Narajji *et al*., 2007; Arenholt-Bendsleve *et al*., 1988). The margin between its protective role and adverse effect is very low and depends on the form of selenium being used.

Role of selenium in dogs: Research studies to date have indicated that selenium does have beneficial physiological effects on mammals. For example, it is known that selenium, when ingested, reduces the rate of oxidative damage caused by chemicals, by entering the membranes of the body's cells and protecting the contents of the cells from reacting with oxygen in a manner that damages the cells. Selenium deficiency may be associated with a myopathy in dogs (Manketlow, 1963). The diet of these dogs was

principally mutton from an area of New Zealand where selenium-responsive diseases of sheep were noted. A fatal, myocardial necrosis was seen in young pups and a skeletal myodegeneration in an adult dog. Two bitches that had lost litters during previous perinatal periods were dosed with selenium during pregnancy and subsequently whelped normal litters.

Synergistic effect of selenium with vitamin E was observed when administered together to Beagles. It was demonstrated that Beagles which were initially 5 to 8 weeks old, developed clinical signs of vitamin E-selenium deficiency after 40 to 60 days of consuming an unsupplemented semisynthetic diet (Van Vleet, 1975). Generalized muscular weakness progressed from unsteadiness to prostration and coma. In a case study it was observed that multiple severe deaths due to acute myocardial degeneration occurred in a commercial kennel. Further losses were prevented when vitamin E and selenium supplementation was instituted (Green and Lemckert, 1977).

Selenium has been found to reduce incidences of cancer in both dogs and human. Selenium supplementation has shown to decrease DNA damage and increase epithelial cell apoptosis within the aging canine prostate (Waters *et al*., 2003). Research suggests that selenium also helps with improving long-term joint health and can reduce risks of Kashin-Beck disease (Sudre and Mathieu, 2001; Levander and Beck, 1997; Beck *et al*., 2003) which involves the articular cartilage between joints degenerating, thyroid disease and cancers. Selenium is also thought to help prevent hip dysplasia (Hamilton, 1999).

Selenium can increase the health of the skin, potentially reducing dandruff and dry skin. It plays an important role in hair growth. Selenium can also improve the hair coat quality, making it more soft

and shiny. As a result of a healthier coat, there is also the possibility of less shedding and hair loss. In a study conducted by Yu *et al.* (2006) it was demonstrated that both low and high selenium in diet reduced hair growth in adult dogs.

Pet animals obtain dietary selenium from cereals and grains, or from the tissues of other animals, depending on dietary habit. The forms of dietary selenium from both plant and animal sources include a range of inorganic and organic selenium compounds (Whanger, 2002). The primary form of selenium in plants is selenomethionine, together with smaller amounts of selenocysteine and selenite. The forms of selenium found in animals include selenoproteins (formed from biologically active selenocysteine, e.g., glutathione peroxidase, selenoprotein P), Se-containing proteins (formed from non-specific incorporation of selenomethionine or selenocysteine) as well as nonprotein and inorganic selenium (selenite, selenate) and methylated selenium (forms that are excreted) (Lobinski *et al.*, 2000).

Biochemical role of selenium in mammals is clearly established. It forms the active center for selenoenzymes. Glutathione peroxidase is one such enzyme which contains selenol (-SeH) group in the active center. Glutathione peroxidase catalyzes the oxidation of reduced glutathione and allows for the reduction of hydrogen peroxide to water, preventing **lipid peroxidation** and cellular damage (Rotruck *et al.*, 1973). The GPx catalytic site includes a Selenocysteine residue in which the selenium undergoes a redox cycle involving the selenol (ESeH) as the active form that reduces **hydrogen peroxide**s and organic peroxides. Selenocysteine is known as 21st **amino acid** (Rayman, 2005). It is located in the N-terminal end of helix α_1 (Epp *et al.*, 1983). The selenol is oxidized

to selenenic acid (ESeOH), which reacts with reduced glutathione (GSH) to form a selenenyl sulphide adduct (ESeSG). A second glutathione then regenerates the active form of the enzyme by attacking the ESeSG to form the oxidized glutathione (GSSG). Thus, in the overall process, two equivalents of glutathione are oxidized to the disulphide and water, while the hydroperoxide is reduced to the corresponding alcohol as shown in Fig. 1 (Roy *et al.*, 2005).

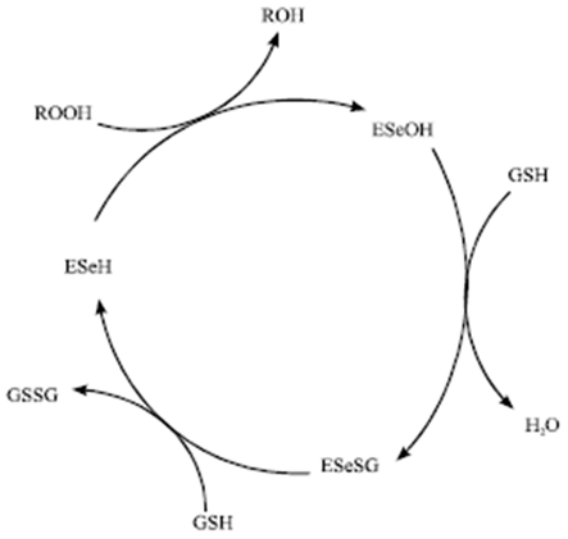

Fig. 1: Schematic diagram of proposed catalytic mechanism for reduction of hydroperoxides by Gpx

Table 1: Selenium levels in different food items

Food	Levels (µg)
Brazil nuts, dried, unblanched, 1 ounce	544
Tuna, light, canned in oil, drained, 3 ounces	63
Beef, cooked, 32 ounces	35
Spaghetti w/ meat sauce, frozen entrée, 1 serving	34
Cod, cooked, 3 ounces	32
Turkey, light meat, roasted, 32 ounces	32
Beef chuck roast, lean only, roasted, 3 ounces	23
Chicken Breast, meat only, roasted, 32 ounces	20
Noodles, enriched, boiled, 1/2 cup	17
Macaroni, elbow, enriched, boiled, 1/2 cup	15
Egg, whole, 1 medium	14
Cottage cheese, low fat 2%, 1/2 cup	12
Oatmeal, instant, fortified, cooked, 1 cup	12
Rice, white, enriched, long grain, cooked, 1/2 cup	12
Rice, brown, long-grained, cooked, 1/2 cup	10
Bread, whole wheat, commercially prepared, 1 slice	10
Walnuts, black, dried, 1 ounce	5
Bread, white, commercially prepared, 1 slice	4
Cheddar cheese, 1 ounce	4

*DV: Daily value. DVs are reference numbers developed by the Food and Drug Administration (FDA) to help consumers determine if a food contains a lot or a little of a specific nutrient. The DV for selenium is 70 µg. Most food labels do not list a food's selenium content. The percent DV (%DV) listed on the table indicates the percentage of the DV provided in one serving. A food providing 5% of the DV or less is a low source while a food that provides 10-19% of the DV is a good source. A food that provides 20% or more of the DV is high in that nutrient. It is important to remember that foods that provide lower percentages of the DV also contribute to a healthful diet. For foods not listed in this table, please refer to the U.S. Department of Agriculture's Nutrient Database Web site: http://www.nal.usda.gov/fnic/cgi-bin/nut_search.pl

Finally glutathione disulfide gets reduced by NADPH which is catalyzed by glutathione reductase. This results in the formation of glutathione.

Nutritional forms of selenium: The Brazil nut (*Bertholletia excelsa*) which is a South American tree in the family Lecythidaceae have relatively high selenium concentrations (Table 1). Foods of

low protein content, including most fruits and vegetables, provide little selenium (Simcock *et al*., 2005).

Selenium and pet food diets: Fish, meat, poultry, whole grains and dairy products are typical sources of this nutrient. AAFCO and the FDA have approved a selenium supplement to animal diets, most commonly in the form of sodium selenite for pet foods. It has been observed that inorganic selenium is less available when the animal is under stress. In addition, there is a significant loss of selenium after periods of stress, making selenium levels in the blood less stable. Depending on the nature of ingredients used in pet food formulations, the selenium levels vary in all diets. Research (Fan and Kizer, 1990; Olson, 1986) proved that inorganic selenium sources can be toxic in high doses; affecting an animal's blood, liver and muscles. Inorganic selenium cannot be fully metabolized or stored in the body. Consequently, selenium deficiencies still arise in animals that are supplemented with inorganic selenium (Lopez *et al*., 1969).

SELENIUM METABOLISM IN MONOGASTRIC SPECIES

The wild dog would consume selenium obtained from animal tissue, however as the wild dog would not usually eat a whole carcass at one time, the amount and form of selenium ingested depends on the part of the animal consumed and its metabolic pathway as indicated in Fig. 2. For example the liver is a primary site of selenoprotein synthesis and contains selenoprotein P, glutathione peroxidase and other functional selenoenzymes. In contrast, the gastrointestinal tract may contain plant material that the prey animal has eaten and therefore may contain inorganic selenite or selenate, as well as organic selenomethionine.

Since, cat generally consumes a diet of whole prey such as rats or mice, the forms of selenium ingested would be in the form of functional selenoproteins such as glutathione peroxidase and selenoprotein P, the seleno**amino acid**s selenomethionine and selenocysteine, as well as non-functional stored selenium that has been incorporated into body proteins such as skeletal muscle, hair and nails. The AAFCO has defined he minimum selenium requirement in cats and dogs (Table 2).
Hence, animal tissues play a primary role as source of dietary selenium for both cats and dogs, i.e., the organic forms of selenomethionine and selenocysteine. The pets which are fed with commercial diets today are somewhat different from what their ancestors would have eaten. Most of these diets contain a high proportion of plant material and for the cat as a true carnivore this may not be particularly suitable. Selenium concentrations in pet foods is highly variable.

Most dry and canned dog foods today use an inorganic type of selenium, sodium selenite or sodium selenate. In addition to selenium present in the pet food ingredients, additional selenium sources are added in commercial diet formulations. Selenite (Forceville and Meaux, 2007) was found to have a pro-oxidant effect and therefore the use of selenate was preferred.

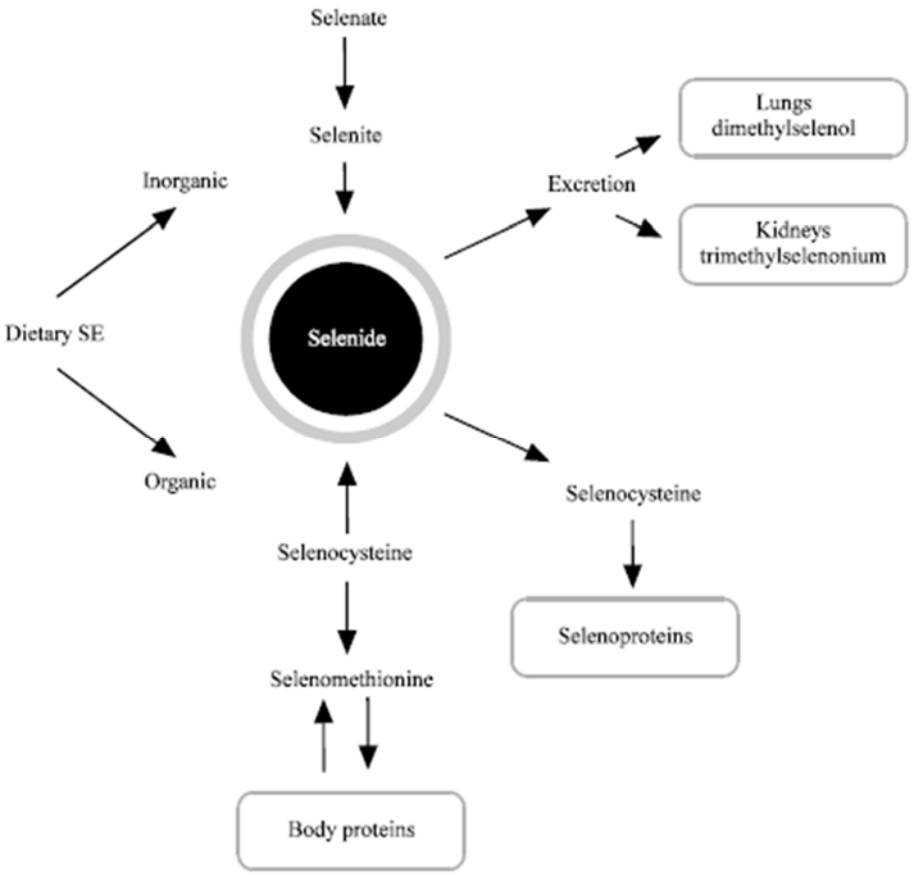

Fig. 2: Overview of selenium metabolism in monogastric species

Table 2: Selenium nutrient profiles as published in 2008[a] by AAFCO for dog and cat food

Nutrient	Units DM basis	Growth and reproduction minimum	Adult maintenance minimun
Dog	mg kg^{-1}	0.11	0.11
Cat	mg kg^{-1}	0.10	0.10

However because inorganic selenium cannot be stored in the body, organic forms of selenium are being increasingly used as they are safer and more efficiently used in the body.

CONCLUSIONS

The role of trace mineral selenium in animals particularly pets is discussed and reviewed. Selenium deficiency is found to be one of the key factor behind many diseases like Kashin beck disease, cardiomyopathy, etc., Selenium levels and its forms in the diets play an important role in managing the trace mineral level in the body of the pet animal. However, toxicity associated with the high dose of selenium makes it vulnerable to pets and other animals due to the narrow difference between its required dose and the toxic dose. Current selenium supplements are mainly dependent on inorganic sources like sodium selenite which are found to be less bioavailable and also toxic. However, relative uses of selenium and its forms would be dependent on its nature of application and end use requirement. Keeping safety of the pet animals and environment as main focus areas, organoselenium compounds would be a good and alternate prospective choices for research scientists working in pet animal nutrition.

References

Arenholt-Bendsleve, D., M. Abdulla, A. Jepsrn and E. Pedeson, 1988. Effect of organic and inorganic selenium on human keratinocytes. Trace. Elem. Med., 5: 29-34.

Beck, M.A., O. Levander and J. Handy, 2003. Selenium deficiency and viral infection. J. Nutr., 133: 1463-1467.

PubMed | Direct Link |

Chance, B., H. Sies and A. Boveris, 1979. Hydroperoxide metabolism in mammalian organs. Physiol. Rev., 59: 527-605.

PubMed | Direct Link |

Epp, D., R. Landenstein and A. Wendel, 1983. The refined structure of the selenoenzyme glutathione peroxidase at 0.2 nm resolution. Eur. J. Biochem., 133: 51-69.

Fan, A.M. and K.W. Kizer, 1990. Selenium. nutritional, toxicologic and clinical aspects. West J. Med., 153: 160-167.

PubMed |

Forceville, X. and C.H. Meaux, 2007. Effects of high doses of selenium, as sodium selenite, in septic shock patients a placebo-controlled, randomized, double-blind, multi-center phase II study-selenium and sepsis. J. Trace Elem. Med. Biol., 21: 62-65.

PubMed |

Green, P.D. and J.W.H. Lemckert, 1977. Vitamin E and selenium responsive myocardial degeneration in dogs. Can. Vet. J., 18: 290-291.

PubMed |

Gutteridge, J.M.C., 1994. Free radicals and aging. Rev. Clin. Gerontol., 4: 279-288.

Halliwell, B., J.R. Hoult and D.R. Blake, 1982. Oxidants, inflammation and anti-inflammatory drugs. FASEB J., 2: 2867-2873.

PubMed |

Hamilton, D., 1999. Homeopathic Care for Cats and Dogs: Small

Doses for Small Animals. North Atlantic Books, California, USA., ISBN-13: 978-1556432958.

Jacob, J.H., A.M. Khalil and A.O. Maslat, 2004. *In vitro* cytogenetic testing of an organoselenium compound and its sulfur analogue in cultured rat bone marrow cells. J. Carcinog., 3: 5-13.

CrossRef | PubMed | Direct Link |

Levander, O.A. and M.A. Beck, 1997. Interacting nutritional and infectious etiologies of Keshan disease: Insights from coxsackie virus B-induced myocarditis in mice deficient in selenium or vitamin E. Biol. Trace Elem. Res., 56: 5-21.

PubMed |

Lobinski, R., J.S. Edmonds, K.T. Suzuki and P.C. Uden, 2000. Species-selective determination of selenium compounds in biological materials. Pure Applied Chem., 72: 447-461.

Direct Link |

Lopez, P.L., R.L. Preston and W.H. Pfander, 1969. Whole-body retention, tissue distribution and excretion of Selenium-75 after oral and intravenous administration in lambs fed varying selenium intakes. J. Nutr., 97: 123-132.

Direct Link |

Manketlow, B.W., 1963. Myopathy of dogs resembling white muscle disease of sheep. N. Z. Vet. J., 11: 52-55.

Mugesh, G. and H.B. Singh, 2000. Synthetic organoselenium

compounds as antioxidants: Glutathione peroxidase activity. Chem. Soc. Rev., 29: 347-357.

Narajji, C., M.D. Karvekar and A.K. Das, 2007. Biological importance of organoselenium compounds. Indian J. Pharm. Sci., 69: 344-351.

Direct Link |

Nordberg, J. and E.S.J. Arner, 2001. Reactive oxygen species, antioxidants and the mammalian thioredoxin system. Free Radic. Biol. Med., 31: 1287-1312.

CrossRef | PubMed | Direct Link |

Olson, O.E., 1986. Selenium toxicity in animals with emphasis on man. Int. J. Toxicol., 5: 45-70.

CrossRef |

Rayman, M.P., 2005. Selenium in cancer prevention: A review of the evidence and mechanism of action. Proc. Nutr. Soc., 64: 527-542.

PubMed |

Rotruck, J.T., A.L. Pope, H.E. Ganther, A.B. Swanson, D.G. Hafeman and W.G. Hoekstra, 1973. Selenium: Biochemical role as a component of glutathione peroxidase. Science, 179: 588-590.

CrossRef | PubMed | Direct Link |

Roy, G., B.K. Sarma, P.P. Phadnis and G. Mugesh, 2005. Selenium-containing enzymes in mammals: Chemical perspectives. J. Chem.

Sci., 117: 287-303.

CrossRef |

Schwarz, K. and C.M. Foltz, 1957. Selenium as an integral part of factor 3 against dietary liver degeneration. J. Am. Chem. Soc., 79: 3292-3293.

Direct Link |

Schwarz, K., J.G. Bieri, G.M. Briggs and M.L. Scott, 1957. Preventions of exudative diathesis in chicks by Factor 3 and selenium. Proc. Soc. Exp. Biol. Med., 95: 621-625.

PubMed |

Scott, M.L., G. Olson, L. Krook and W.R. Brown, 1967. Selenium-responsive myopathies of myocardium and smooth muscle in the young poultry. J. Nutr., 91: 573-583.

Direct Link |

Sheffy, B.E. and R.D. Schultz, 1979. Influence of vitamin E and selenium on immune response mechanisms. Fed. Proc., 38: 2139-2143.

PubMed |

Simcock, S.E., S.M. Rutherfurd, T.J. Wester and W.H. Hendriks, 2005. Total selenium concentrations in canine and feline foods commercially available in New Zealand. N. Z. Vet. J., 53: 1-5.

PubMed |

Sudre, P. and F. Mathieu, 2001. Kashin-beck disease: From etiology

to prevention or from prevention to etiology. Int. Orthop., 25: 175-179.

Sunde, R.A., 1997. Selenium. In: Handbook of Nutritionally Essential Mineral Elements, O'Dell, B.L. and R.A. Sunde (Eds.). Marcel Dekker, New York, pp: 493-556.

Van Vleet, J.F., 1975. Experimentally induced vitamin E-selenium deficiency in the growing dog. J. Am. Vet. Med. Assoc., 166: 769-774.

PubMed |

Waters, D.J., S. Shen, D.M. Cooley, D.G. Bostwick and J. Qian *et al*., 2003. Effects of dietary selenium supplementation on DNA damage and apoptosis in canine prostate. J. Nat. Cancer Inst., 95: 237-241.

PubMed |

Whanger, P.D., 2002. Selenocompounds in plants and animals and their biological significance. J. Am. College Nutr., 21: 223-232.

Direct Link |

Yu, S., K.J. Wedekind, C.A. Kirk and R.F. Nachreiner, 2006. Primary hair growth in dogs depends on dietary selenium concentrations. J. Anim. Physiol. Anim. Nutr., 90: 146-151.