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Inorganic Versus Organic Selenium Supplementation: A Review

¹Mahima, ²Amit Kumar Verma, ³Amit Kumar, ⁴Anu Rahal, ¹Vinod Kumar and ¹Debashis Roy

¹Department of Animal Nutrition,

²Department of Veterinary Epidemiology and Preventive Medicine,

³Department of Veterinary Microbiology and Immunology,

⁴Department of Veterinary Pharmacology and Toxicology,

Uttar Pradesh Pandit Deen Dayal Upadhyay Pashu Chikitsa Vigyan Vishvidhyalaya Ewam Go-Anusandhan Sansthan (DUVASU), Mathura 281001, India

Abstract: Selenium is an essential trace element in the diets which is required for maintenance of health, growth and biochemical-physiological functions. The area covered in this review has been rapidly unfolding in recent years and has already acquired a vast spread. This study presents a concise introductory overview of the effect of organic and inorganic selenium on growth performance, carcass traits, daily egg production, egg quality, Se uptake in various tissues and plasma and plasma glutathione peroxidase activity in animals.

Key words: Selenium enriched yeast, bioavailability, egg quality, glutathione peroxidase, growth performance, metabolism, organic sources

INTRODUCTION

For health, growth and biochemical and physiological functions, essential trace elements are necessary in diets of the animals (Scott *et al.*, 1982). Among those essential trace elements is Selenium (Se), known to have important roles in a number of biochemical functions in human and animals, such as biological antioxidant, immune function, reproduction and thyroid hormone metabolism (Surai, 2006). Selenium (Se) is a one of the essential trace mineral (NRC, 1994). After its discovery in 1817 by the Swedish chemist, Jons Jakob Berzelius in the flue dust of iron pyrite burners, in Stockholm, Sweden (Levander, 1986; Sunde, 1997), it was thought to be toxic; direct cause of alkali disease and blind staggers (Franke, 1934a, b; Franke and Potter, 1935; Moxon, 1937) and as a carcinogen (Nelson *et al.*, 1943) for many years. Since, selenium was identified in association with tellurium which was named after the Latin term, *tellus*, for earth; so Se was named Se after the Greek term, *selene*, for moon. However, Schwarz and Foltz (1957) first time reported that Se prevent liver necrosis (along with vitamin E and cystine) in rats, since then nutritionists around the world started studies to discover the metabolic function of this element and document the effects of its deficiency in the food of human and animals. In both the human and animals, its deficiency causes various diseases like necrosis of liver, exudative diathesis, nutritional muscular

dystrophy, poor feathering, retention of placenta, mastitis, cystic ovaries, cancer, cardiovascular diseases, immunodeficiencies, poor fertility etc. (Shamberger, 1983).

It has been identified to be an integral part of more than 30 distinct selenoproteins, including the enzyme particularly glutathione peroxidases (Rotruck *et al.*, 1973), a group of antioxidant enzymes that help in the protection of the body cells from the damage caused by free-radicals (Sunde, 1997; Arthur, 2000). Free radicals, being highly reactive, induce a series of events leading to pathogenesis of various diseases affecting cardiovascular system and aging process etc. (Puri, 2002). Although the dietary need for Se has been established but still it is considered to be the most toxic dietary essential trace mineral. Therefore, the FDA regulates supplementation of Se into diets (FDA, 2000). Therefore, it is very common practice to supplement diets with Se. Selenium occurs in both inorganic and organic form (Daniels, 1996). Among the inorganic forms (i.e., selenates, selenides and selenite), the selenide form is more frequently found in the food supply. The organic form includes selenomethionine and selenocysteine and is found in plants (Schrauzer, 2000) and animals respectively (Kincaid *et al.*, 1999; Boldizarova *et al.*, 2004). Free mineral ions released during the process of digestion may form complexes with other dietary substances and become difficult to absorb or, totally unabsorbable. Thus, the availability of the element may vary significantly. Because of these

uncertainties, the levels provided in the diet are often kept on higher side than the required amount for optimum performance. This may sometimes result in excess of supply and wastage. Therefore, after the approval by FDA (2000), researchers are interested in the studies using organic forms, such as Selenomethionine (SeM) or Se-enriched Yeast (SeY), as supplemental sources of Se (Rayman, 2004).

Although, previous studies have reported about the use and benefits associated with inorganic and organic Se supplementation. The area covered in this review has been rapidly unfolding in recent years and has already acquired a vast spread. This study presented a concise introductory overview of the effect of organic and inorganic selenium on growth performance, carcass traits, daily egg production, egg quality, Se uptake in various tissues and plasma and plasma glutathione peroxidase activity in animals.

CHEMISTRY OF SELENIUM

With the atomic number 34 and atomic weight 78.96, Se is a member of Group VIA along with oxygen, sulphur, tellurium and polonium (Sunde, 1997) in the periodic table of elements and classified as a metalloid element i.e., having the properties of both metal and non-metal. It has four common oxidative states: selenide (-2), Se (0), selenite or selenious acid (+4) and selenate or selenic acid (+6). These different states play significant role in the biochemistry of Se.

SOURCES OF SELENIUM

The plant based (Burk, 1976) and meat based (Levander, 1986; Cai *et al.*, 1995) primary and natural sources of selenium are selenoamino acids such as selenomethionine, selenocysteine and selenocystine. The selenoamino acids are bound in protein, mainly as selenomethionine and selenocysteine and constitute 50-80% of the total selenium present in plants and grains (Butler and Peterson, 1967). These foods differ tremendously in concentration of Se depending upon the concentration of selenium in the soil of that region in which they are found. Longnecker *et al.* (1991) reported the low amount of selenium and dietary selenium deficiency has been reported in selenium deficient regions of China and Russia. Similarly, animals that take fodder that were grown in selenium rich soil have higher levels of selenium.

DISTRIBUTION IN BODY

Selenium can be found in all the cells and tissues of the body but its level in blood and tissues are very much

influenced by dietary selenium form and intake. Behne and Wolters (1983) and Behne and Hofer-Bosse (1984) conducted a study on rats with the diets supplemented with 0.3 ppm of Se and found its highest concentration in the kidneys, followed by testes, liver, adrenals, erythrocytes, plasma, spleen, pancreas, lungs, heart, thymus, gastrointestinal tract, skeleton, brain and muscles. Based on the data provided by Behne and Wolters (1983) and Behne and Hofer-Bosse (1984) in 1997, Sunde (1997) calculated total amounts of Se and reported that the largest total amount of Se was in muscle followed by the liver, plasma, erythrocytes and kidneys. Similar distribution of Se was reported by Schroeder *et al.* (1970) from the samples taken from autopsies of North Americans.

METABOLISM OF SELENIUM

Selenium plays a vital role in a most of the physiological processes in the body. The metabolism of Se depends on its chemical state and on the amount supplemented in diet. Selenium is assimilated more effectively from plant food than animal products but some dietary contents viz., vitamin C and vitamin E affect its absorption. The majority of Se is absorbed in the duodenum (Wright and Bell, 1966; Whanger *et al.*, 1976) followed by jejunum and ileum but practically none from the stomach (Whanger *et al.*, 1976) and is transported across the intestinal brush border actively or passively. The type of absorption depends on the source of selenium in diet. It was found that inorganic sources such as SS or selenates are absorbed by simple diffusion process, while organic sources like as SY or Selenomethionine (SeM) are actively absorbed via amino acid transport mechanisms (Combs Jr. and Combs, 1986).

Due to reducing nature of rumen, absorbing ability for inorganic Se is poor in ruminants. Wright and Bell (1966) reported that rumen microbes reduce most of dietary inorganic selenium to unabsorbable inorganic selenide forms. So the selenite selenium availability to ruminants is 25-30%.

First of all selenate is converted to selenite (Axley and Stadtman, 1989). Then, selenite is non-enzymically reduced via., formation of selenodiglutathione (GS-Se-SG) to selenide (Ganther, 1966; Hsieh and Ganther, 1977; Foster and Sumar, 1997). Now, selenide may have several different options. Selenides plays an important role in the mixed function oxidase system of microsomal and other cellular membranes (Chatterjea and Shinde, 2002). Methylation of Selenide form methylselenol (CH_3SeH) which then form dimethylselenide or trimethylselenonium ion ($(\text{CH}_3)_3\text{SeH}$) (Hsieh and Ganther, 1977). Selenide can also bind to the Se-binding proteins or it can be a substrate for

selenophosphate synthetase for the tRNA-mediated synthesis of selenoproteins (Sunde, 1997). This last step converts inorganic Se into the organic forms of Se that are found in tissues of mammals.

Sunde (1997) found that the metabolism of organic Se is different than inorganic Se. Initially, all the dietary selenomethionine is incorporated into protein. Hoffman *et al.* (1970) and McConnell and Hoffman (1972). Selenomethionine can be metabolized to Se-adenosyl methionine (SeAM) and further to Se-adenosyl homocysteine (SeAH) (Markham *et al.*, 1980). Then the SeAH is converted to selenocysteine by the enzymatic activity of cystathionine β -synthase and cystathionine γ -lyase. Subsequently, selenocysteine can be incorporated into proteins or degraded, releasing the selenite or it can be degraded by selenocysteine lyase enzyme, releasing elemental Se which can be reduced to selenide (Esaki *et al.*, 1982). Another fate for selenomethionine is to be transaminated to methylselenol (Steele and Benevenga, 1979) and then methylselenol can be transformed to selenide via S-methyltransferase (Sunde, 1997). At this point, selenide would be metabolized as discussed above.

ABSORPTION AND BIOAVAILABILITY

Absorption and bioavailability of trace minerals including Se is generally one of the most important issue in their utilization. Most of the times, term absorption is coined with availability because selenium must be absorbed before its utilization. During the process of digestion in the gastrointestinal tract the Se from inorganic sources are released and may re-combine with other components of feed and fodder or we can say digesta in the intestine making insoluble complexes and excreted, thus reducing its absorption across the small intestine whereas, the organic minerals absorbs actively utilizing peptide and/or amino acid uptake mechanisms in the intestine (Ashmead, 1993; Power and Horgan, 2000; Schweizer *et al.*, 2004). Within the complex, selenium is chemically inert due to the coordinate covalent and ionic bonding by the amino ligands thus, more stable and less prone to interactions. The mineral is also protected from physicochemical factors with dietary components such as phytate (Fairweather-Tait, 1996), remained electrically neutral. Bioavailability could be affected by any of the factor such as species, sex, age, physiological stage of the animals like growth, pregnancy, lactation, nutritional status, health, gastrointestinal secretions and microflora (Johnson, 1989; Fairweather-Tait, 1996).

In terms of bioavailability of dietary inorganic versus organic Se supplementation, many comparisons have

been made in the last two decades using a variety of domestic animal species chicken (Collins *et al.*, 1993), cattle (Ortman and Pehrson, 1999; Surai, 2006; Peters and Mahan, 2008), goat (Pavlata *et al.*, 2011). Researchers have proved that organic Se is having 120-200% more bioavailability in comparison to sodium selenite (Pagan *et al.*, 1999; Hall *et al.*, 2011; Mansoub, 2011) in cattle (Pehrson *et al.*, 1989; Nicholson *et al.*, 1991; Gunter *et al.*, 2003; Juniper *et al.*, 2008; Liao *et al.*, 2011), pig (Mahan and Kim, 1996) and guinea pig (Mahima, 2006; Chaudhary *et al.*, 2010; Mahima *et al.*, 2011). However, in contrast there were no significant differences in absorption and subsequent metabolism following supplementation with the two different forms of selenium i.e., inorganic and organic (Parsons *et al.*, 1985; Pavlata *et al.*, 2011) even higher in sheep fed inorganic selenium (Koenig *et al.*, 1997).

SECRETION IN MILK OR COLOSTRUMS

Selenium is very important for the proper development of the newly born animal and its deficiency adversely affect the growth, health and fertility (Schwarz and Foltz, 1957). During pregnancy, Se passes through the placental barrier and the newborn receives a sufficient Se supply even if the animal is moderately Se deficient (Gunter *et al.*, 2003). In the first weeks of life, milk is the only dietary source of selenium for newborn animals (Ortman and Pehrson, 1997, 1999). Selenium status of animals at birth and weaning can be affected by the dam's body Se reserves, dietary Se concentration and source of Se (Mahan, 2000; Mahan and Peters, 2004). There are studies in pigs (Acda and Chae, 2002; Mahan and Peters, 2004; Yoon and McMillan, 2006; Svoboda *et al.*, 2008), cattle (Knowles *et al.*, 1999; Givens *et al.*, 2004; Slavik *et al.*, 2008; Ceballos *et al.*, 2009) that clear that concentration of selenium in milk or colostrum increases in both the inorganic and organic supplementation but were significantly greater when dams were fed organic Se (Mahan, 1994; Mahan and Kim, 1996; Mateo *et al.*, 2007). The increase has ranged from 34% (Juniper *et al.*, 2006) to 90% (Ortman and Pehrson, 1997).

GROWTH, PERFORMANCE AND MEAT QUALITY

As far as the growth and production performance in terms of average daily gain, average daily feed intake or gain: feed ratio is concerned, the majority of studies in cattle (Nicholson *et al.*, 1991; Gunter *et al.*, 2003; Richards and Loveday, 2004; Davis *et al.*, 2008), chicken (Spears *et al.*, 2003; Payne and Southern, 2005; Ryu *et al.*, 2005) did not detect any difference between inorganic and

organic selenium supplementation. However, a significant improvement in feed intake (Cantor *et al.*, 1982) and FCR (Naylor *et al.*, 2000; Sevcikova *et al.*, 2006; Wang and Xu, 2008) in chicken, Guinea pig (Mahima, 2006; Chaudhary *et al.*, 2010), average daily gain in cattle (Clyburn *et al.*, 2001) fed with organic Se were observed.

Studies on chicken (Payne and Southern, 2005), turkey (Niedzwiedzka *et al.*, 2008), pigs (Mahan, 1996; Mahan and Parret, 1996) have shown that organic selenium is deposited more effectively in muscles than inorganic selenium. This increased tissue concentrations of selenium not only decrease oxidative stress, including protection of unsaturated fatty acids from peroxidation damage (Tapiero *et al.*, 2003; Komiluk *et al.*, 2007) but can also reduce drip loss from meat and the incidence of pale soft, exudative meat (Downs *et al.*, 2000; Naylor *et al.*, 2000) and improves the shelf life during refrigeration (Yoon *et al.*, 2007; Smet *et al.*, 2008; Skrivan *et al.*, 2008). This indicates that supplementation of selenium, particularly organic selenium, improves meat quality and shelf life of poultry meat (Sevcikova *et al.*, 2006).

GLUTATHIONE PEROXIDASES AND OTHER SELENOPROTEINS

For our body, the oxidation and reduction processes are very necessary and this gaining and losing of an electron keeps our body processes in proper function. During the respiration, various peroxides, including hydrogen peroxide are produced in the body which can be harmful to the body as they can lead to generation of free radicals which can damage or destroy cells (Arthur, 2000). A group of enzymes, the glutathione peroxidases (GSH-Px), help to protect the body from these harmful peroxides (Arthur, 2000). Till now, four structurally and genetically different forms of selenium-containing GSH-Px have been functionally described (Ursini *et al.*, 1999) and exist in different tissues or parts of the cell. These enzymes catalyze a reaction that removes hydrogen peroxide from erythrocytes via reduced glutathione. The reduced glutathione is made via the enzyme, glutathione reductase, from oxidized glutathione. Se is the integral part of glutathione peroxidases (Rotruck *et al.*, 1973; Tapiero *et al.*, 2003). Glutathione and glutathione peroxidase protect the unsaturated bonds of membrane phospholipids from the attack of free radicals (Komiluk *et al.*, 2007; Rayman, 2004). Besides the glutathione peroxidase family of enzymes, there are 20 different selenoproteins (Table 1) have been identified, though estimates of the existence of 30-50 such selenoproteins have been made based on electrophoretic separation (Kohrle *et al.*, 2000).

Table 1: Selenium containing proteins and their role

Name	Functions
Glutathione peroxidase	An antioxidant enzyme, decomposes H ₂ O ₂ and other hydroperoxidase Maintains intracellular redox milieu Replenishes a number of crucial antioxidants e.g., vitamin E and C from their oxidized state (Sen, 1995) Forms a structural protein and shields the developing sperm cells (Ursini <i>et al.</i> , 1999)
Thioredoxin reductase	Provides protection to skin from free radicals (Schallreuter and Wood, 1986) Protein thiol redox regulation Vitamin C recycling and DNA synthesis (Mustacich and Powis, 2000) Regulation of apoptosis Thioredoxin-(SH) ₂ complexes with ASK1 preventing downstream signalling for apoptosis.
Iodothyronine deiodinases	Synthesis of active thyroid hormone (Vasudevan and Sreekumari, 2001)
Selenoprotein 1R	Provide protection against free radical mediated oxidative stress
Selenoprotein P	Protect endothelial cells against oxidants and its ability to bind metals supports this point (Burk and Hill, 1993) In the transport of selenium (Schweizer <i>et al.</i> , 2004) Protection against hepatitis B virus X protein induced lipid peroxidation (Yi <i>et al.</i> , 2003)
Selenoprotein S	Regulation of cellular redox balance (Schwartz and Foltz, 1957)

Being a component of GSH-Px, Selenium also acts as a second line of defence against cellular peroxide damage due to the inability of vitamin E to destroy all metabolic peroxides. Most of the times, selenium and vitamin E are mutually replaceable and each acts as a sparing mechanism for the other (Combs Jr., 1981). Wang and Xu (2008) reported that Se supplementation increased plasma GPx activity and further told that this increase is less pronounced with the supplementation of inorganic selenium in comparison to organic selenium. However, in contrast, several studies. (Acda and Chae, 2002; Mahan and Peters, 2004; Payne and Southern, 2005; Yoon and McMillan, 2006; Svoboda *et al.*, 2008) reported that GPx activity was not affected by Se source or concentration or GSH-Px activity increase much faster with selenite in comparison to organic selenium (Pavlata *et al.*, 2011).

CONCLUSION

The practical use of organic selenium will depend on the performance response, health status of animals and environmental impact. These responses will determine the cost effectiveness of organic selenium. Positive responses to organic Se have been reported in relation to bioavailability, secretion in colostrums and milk thus improving neonatal health, improved carcass quality, shelf life and can also prevent drain losses. However, reviews presented revealed no consistent effect of organic sources for Se on growth, performance variables and glutathione peroxidase activity.

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