

# The interrelationship between selenium and vitamin E in poultry

**Dr. Rob Shirley**  
**Adisseo USA**  
**Woodstock, IL 60098**

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## Abstract

From the standpoint of research and real-world livestock production (*i.e. swine, dairy, beef, aqua, poultry*), it is fair to say that today's nutrition programs are a blend of old and new technologies, product availability, and a compromise between old and new ideas regarding how nutrients and products both interact and function in today's high-performing animals. This is especially true when sources of selenium (**Se**; discovered in 1817 by Jöns Jacob Berzelius) and vitamin E (**Vit E**; discovered in 1922 by Evans and Bishop) are considered. Although Se and Vit E play a vital role in ensuring that tissues are protected from oxidative stress so that cellular functions and metabolism are maintained (*see below*), several forms of each exist in the marketplace, and they differ in both purity and efficacy.

Regardless of the animal's stage of production (*growing, producing or reproducing*), the metabolic demands are such that cumulatively, approximately 20 billion Reactive Oxygen Species (ROS) and Reactive Nitrogen Species (RNS) are produced per cell, per day (*personal communication with Dr. Peter Surai*); this does not include the oxidized or pro-oxidant ROS/RNS compounds that may be ingested. While the production of ROS/ RNS seems like a nebulous and staggering number, these are the byproducts of many chemical reactions that drive cellular functions: respiration (*ATP synthesis*), cellular division, intra-, extra-, and para-cellular signaling, synthesis and catabolism of fats, carbohydrates, proteins and nucleic acids, immune responses, chemotaxis, etc. Allowed to build up in and outside the cell, ROS/RNS will readily react with (*i.e. oxidize*) and degrade the structure and function of DNA, vitamins, various proteins that are synthesized and degraded, and poly unsaturated fatty acids that are an integral part of intra- and extracellular membranes. Cells that that experience significant damage can enter cell cycle arrest (*quiescence*) as a hibernation/ defense mechanism or senescence, whereby they eventually undergo either apoptosis (*programmed cell death*) or necrosis (*cell lysis*). Since tissues are made up of cells and there is substantial cross talk between tissues within the animal, the negative effects of ROS/RNS are often observed at the animal level. In other words, when significant oxidative stress occurs at a localized or systemic level, livestock growth, production and reproduction will be negatively affected.

To neutralize ROS/ RNS compounds and overcome oxidative stress, the animal (*i.e. cells within the animal*) relies on three categories of defense to reduce/ transform ROS and RNS into non-toxic compounds: 1. Prevention of redox formation (maintains the balance between redox generation and cell signaling), 2. Prevention of ROS/ RNS generation/ propagation and 3. Repair and elimination of damaged molecules. The animal's antioxidant system relies on the overlapping proton-donating properties of vitamins E and C, carotenoids, enzymes (*super oxide dismutase; SOD, catalase, selenoproteins, glutathione, etc.*). In the case of vitamin E, there are synthetic (*All-rac- $\alpha$ -tocopheryl and All-rac- $\alpha$ -tocopheryl acetate*) and natural forms (*R,R,R- $\alpha$ -tocoherol; R,R,R- $\beta$ -tocoherol; R,R,R- $\gamma$ -tocoherol; R- $\alpha$ -tocotrienol; R- $\beta$ -tocotrienol; R- $\gamma$ -tocotrienol*), each differing in Vit E activity. For Se, the FDA first recognized Se deficiency in poultry in 1973 and passed regulations in 1974 that allowed for the supplementation of 0.1 ppm Se to growing chickens and 0.2 ppm to turkeys via sodium selenate or sodium selenite (21 CFR 573.920). Not until 1987 did the FDA revise this regulation to include other species and increase the supplemental Se rate to 0.30 ppm in complete feed. That being the case, 21 CFR 573.920 has been updated since 1987 to include other sources of Se. Se sources in the marketplace include the first-generation (*inorganic forms; Se-proteinates, Se-Glycinates, sodium selenate*

and sodium selenite), second-generation (organic forms via SeYeast; *Saccharomyces cerevisiae* are grown in fermentation with sodium selenite to produce SeMet), and third-generation (organic, chemically synthesized forms; Selenomethionine (DL-SeMet) and Hydroxy-Selenomethionine (DL-OH-SeMet)). In terms of efficacy, the inorganics are effectively absorbed and used for the body's immediate Se needs; what's not used is excreted. The SeYeast products contain mostly protein-bound SeMet that, upon absorption, is stored within the animal's tissues as SeMet, replacing Methionine on a 1:1 basis. If not stored, SeMet is converted to hydrogen selenide and then selenocysteine (SeCys) as selenoproteins are synthesized, or the hydrogen selenide is methylated and excreted. Due to possible issues in fermentation, sources of SeYeast tend to be extremely variable in SeMet, with an average of 60% of the yeast's total Se being SeMet (~22 to 70% has been observed in the market). The DL-SeMet and DL-OH-SeMet are synthetic and the purest forms of SeMet ( $\geq 99\%$ ). These forms are readily absorbed, efficiently converted to L-SeMet, and are handled the same as SeMet from SeYeast. Biologically, DL-SeMet and DL-OH-SeMet are equally efficacious Se sources. However, while pure DL-SeMet is not stable and degrades quickly in feed and premixes (especially with choline), DL-OH-SeMet is an organic acid and hence significantly more stable.

It is fundamentally important that the audience understands that Se by itself is very reactive and that Se needs to be incorporated into selenoproteins so that it can catalytically reduce oxidized compounds. There are 26 selenoproteins in poultry that modulate immune responses, reduce oxygen radicals, hydroperoxides, peroxide, and oxidized thiols/disulfide bonds (ex. *GSSH to (2) GSH; glutathione*), and activate/ deactivate thyroid hormone. Although selenoproteins and Vit E are two very powerful parts of the antioxidant system in animals, the antioxidant system and normal metabolism are handicapped without selenoproteins. This presentation will focus on Se and Vit E nutrition of poultry and their benefits, alone and in combination.





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