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Effects of Oxidative Stress on Chicken Testis, Causes & Solutions

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Abstract: *Oxidative stress is an important factor for development of infertility, Even more important is the poultry industry, because of very high rate of cell division and mitochondrial oxygen consumption in Poultry Testicular Tissue as well as comparably higher levels of unsaturated fatty acids in this tissue than in other tissues. Testicular tissue is highly predisposed to activity of free radicals, oxidative stress and apoptosis due to several reasons including high cell division rate, cell competition for oxygen rate, low oxygen pressure due to weakened vessels as well as high levels of unsaturated fatty acids. Furthermore, since the body's antioxidant system, including antioxidant enzymes produced in the body is not able to neutralize all free radicals, the use of antioxidant supplements In diets is recommended to fight adverse effects of oxidative stress, enhance spermatogenesis and increase enhance fertility in Breeding poultry.*

Keyword: *Oxidative stress, Testicular Tissue, unsaturated fatty acids, free radical activity, Antioxidants, apoptosis, infertility, Breeding poultry*

I. INTRODUCTION

The lipid composition of chicken Testicular tissue and semen is an important Determines its quality and fertility capacity in the future. Chicken spermatozoa are characterized by comparatively high levels of 20:4n-6 and22:4n-6 fatty acids within their phospholipids. As a result of this high proportion of polyunsaturated fatly acids (PUFA) chicken semen is susceptible tolipid peroxidation , which could lead to Testicular tissue deterioration and then with that sperm during storageSeveral studies have demonstrated that peroxidative damage to poultry Testicular tissue can occur during in vitro incubation. On the other,The antioxidant system of avian semen is poorly understood. Chicken semen contains the natural antioxidant vitamin E, together with antioxidant enzymes glutathione peroxidase (GSH-Px) and superoxide dismutase (SOD) As for other antioxidants, including vitamins A and C, carotenoids, glutathione and uric acid, information is not still available. In avian species, there is also evidence that seminal plasma can protect spermalozoa against peroxidation. In chicken semen, the presence of seminal plasma reducedlipid peroxidation in the sperm by more than 3 .A similar protective effect was observed with turkey semen. However, the antioxidant capactty of seminal plasma was not sufficient to totally prevent peroxidative damage tothe spermatozoa during storage of turkey semen. The role of oxidative stress in establishing apoptosis in the pathophysiology of the contralateral testis of birds and affecting testicular tissue and spermatogenesis has not been

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clearly defined. However, the hypothesis that oxidative stress is a cost of reproduction has been challenged due to inconsistent results reported by manipulations of reproductive effort. Therefore, the purpose of this study is to review the causes and strategies for Effects of Oxidative Stress on Chicken Testis.

II. The Effect Of Oxidative Stress On Chicken Testis

Oxidative stress is an important factor for development of male infertility because of very high rate of cell division and mitochondrial oxygen consumption in testicular tissue as well as comparably higher levels of unsaturated fatty acids in this tissue than in other tissues. Moreover, the level of oxygen pressure is low due to the weakness of testicular artery; therefore, there is a severe cell competition for oxygen. Therefore, the testicular tissue and male reproductive system are particularly susceptible to oxidative stress[5].

Oxidative stress is considered one of the main mechanisms promoting ageing in birds. It is a disturbance in the pro-oxidant/antioxidant balance in favor of the oxidants, leading to a disruption of redox signaling and control and/or molecular damage . Reproductive process in birds and other animals could increase oxidative stress by consuming antioxidants otherwise allocated to self-maintenance or as an inevitable consequence of a higher cell metabolism rising reactive oxygen species (ROS) levels [6].

Testis of tissue remains vulnerable to oxidative stress due to the abundance of highly unsaturated fatty acids and the presence of potential reactive oxygen species (ROS)-generating systems. ROS generation can be from the mitochondria and a variety of enzymes including the xanthine- and NADPH- oxidases,5,6 and the cytochrome P450s.7 These enzymes specialize in the professional generation of ROS or produce these toxic metabolites as an inadvertent consequence of their biochemical activity. In order to address this risk, the testes have developed a sophisticated array of antioxidant systems comprising both enzymatic and non-enzymatic constituent[7].It is shown in Figure 1 Major pathways of reactive oxygen species generation and metabolism.

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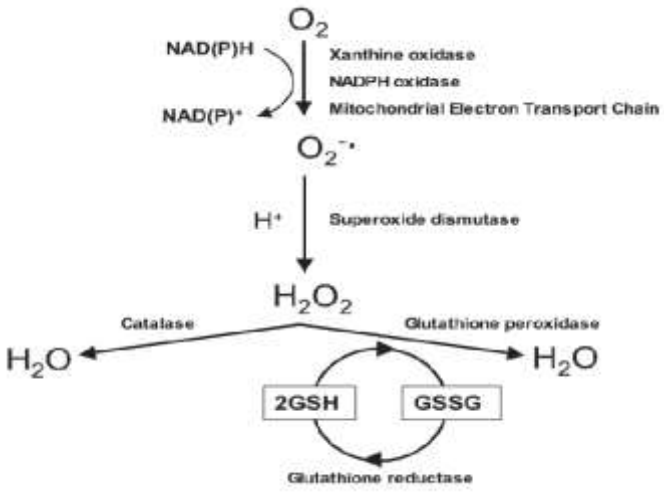

Figure 1: Major pathways of reactive oxygen species generation and metabolism.

III. RELATIONSHIP BETWEEN OXIDATIVE STRESS AND APOPTOSIS

The role of Oxidative stress in the establishment apoptosis in the pathophysiology of contralateral testis of birds and affects testicular tissue and spermatogenesis has not been clearly defined. But there is some evidence to show Oxidative stress is part of the main mechanisms That ca, stimulate reactive oxygen species (ROS) and cause oxidative stress by affecting the balance between the prooxidant and the antioxidant in the body[8] . In the defense mechanisms against oxidative stress, the free radical scavenger glutathione (GSH) and antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD) and glutathione peroxidase (GPx) are one of the most important defense mechanisms of ROS, which are good indicators of assessment. However, lipid peroxidation occurs when these enzymes are not able to offset excess ROS . Malondialdehyde (MDA) is an end product of lipid peroxidation, mainly comes from peroxidation of polyunsaturated fatty acids in biological system, is widely used as a biological marker to evaluate oxidative damage[9]. Apoptosis and autophagy are two major pathways of programmed cell death under developmental and/or stressful conditions, and they are closely related (Bongaerts, 2008). Apoptosis can be triggered via the extrinsic pathway, which is activated through Tumor Necrosis Factor (TNF) and Factor associated suicide (Fas), or the intrinsic pathway, which is mediated by B cell lymphoma/leukemia 2 (Bcl-2) gene family releasing. Moreover, both pathways induce apoptosis through activation of effector caspases[10] .Autophagy is an evolutionary mechanism in all eukaryotic cells that plays a key role in maintaining cell homeostasis and adapting to various stress conditions, and mediates by various proteins like Beclin-1 and Microtubuleassociated protein light chains 3 (LC3)[11] . The cross-talk between apoptosis and autophagy can exist at the level of signal transduction

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10 Yang, J., Y. Zhang, S. Hamid, J. Cai, Q. Liu, H. Li, R. Zhao, H. Wang, S. Xu, and Z. Zhang. 2017. Interplay between autophagy and apoptosis in selenium deficient cardiomyocytes in chicken. J. Inorg. Biochem. 170:17–25.
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pathways that sense death/survival signals and translate them into molecular events that activate or repress specific death processes .. Recent studies have demonstrated that Bcl-2 gene family which is well-known as apoptosis-related genes can alsoregulate autophagy. An apoptosis-related gene, Bcl-2, also showed inhibition of autophagy. For example, Beclin-1 can bind Bcl-2/Bcl-XL to form constitutive Bcl-2/Bcl-XL-Beclin-1 to reduce Beclin-1 monomeric to inhibit autophagy (Levine et al., 2008). Moreover,protein 53 (p53) can regulate autophagy through inhibitingthe mammalian target of rapamycin (mTOR)pathway. mTOR is considered as a key homeostatic regulator by upregulated protein, lipid synthesis and inhibiting excessive autophagy. What's more, mTOR not only regulates autophagy, but is also regulated by apoptosis factors, which further illustrates the cross-talk between autophagy and apoptosis .In addition, in the case of oxidative stress, excessive ROS can activate both apoptosis and autophagy. Kroemer et al. (2010) showed that autophagy can be activated by ROS in cardiomyocytes[12]. Studies have proved that lead (Pb) can affect the balance of mitochondrial dynamics by inducing oxidative stress, resulting in autophagy, which ultimately causes the chicken spleen immune dysfunction[13]. As2O3 could trigger extrinsic and intrinsic apoptosis pathways in immune organs of chickens, meanwhile, ROS generated by oxidative stress might be an important driver of excessive apoptosis. Testes are important reproductive organs of chickens, which are related to the production and reproduction of the poultry industry. If the testicles cause poisoning, the quality of sperm in the testes will be affected, eventually leading to sperm dysplasia or deformity, which will affect the success rate of mating. Or even if the mating is successful, the offspring will be stunted, which will affect the meat quality and harm the development of the breeding industry[14].

IV. APOPTOTIC MARKERS FOR PREDICTION OF SPERM FERTILIZABILITY

Spermatogenesis, the process by which male spermatogonia develop into mature spermatozoa, is a complex biological process involving mitosis, meiosis, and cell differentiation. Testicular activity is controlled both genetically and hormonally, but environmental conditions may also have an influence. Spermatogenesis is thus a valuable indicator of ecological and anthropogenic factors affecting animal reproduction. Because of its dynamic nature, spermatogenic activity requires proliferative and degenerative processes to be appropriately balanced[15].

Apoptosis is an active, genetically controlled, signalinduced process leading to selective cell death[16] . Both spermatogonial cell proliferation and spontaneous degeneration of spermatocytes frequently concur during normal testis function .

12 Kroemer, G., G. Marino, and B. Levine. 2010. Autophagy and theintegrated stress response. Mol. Cell. 40:280–293.
13 Han, Y., C. Li, M. Su, Z. Wang, N. Jiang, and D. Sun. 2017. Antagonisticeffects of selenium on lead-induced autophagy by influencing mitochondrial dynamics in the spleen of chickens. Oncotarget. 8:33725–33735
14 Zhao, P., Y. Guo, W. Zhang, H. Chai, H. Xing, and M. Xing. 2017b.Neurotoxicity induced by arsenic in Gallus Gallus: Regulation ofoxidative stress and heat shock protein response. Chemosphere.166:238–245.
15 Rajesh K. Dadhich, Francisca M. Real, Federico Zurita, Francisco J. Barrionuevo, Miguel Burgos, Rafael Jiménez, Role of Apoptosis and Cell Proliferation in the Testicular Dynamics of Seasonal Breeding Mammals: A Study in the Iberian Mole, Talpa occidentalis, Biology of Reproduction, Volume 83, Issue 1, 1 July 2010, Pages 83–91,
16 Schwartzman RA, Cidlowski JA. Apoptosis: the biochemistry andmolecular biology of programmed cell death. Endocr Rev 1993; 14:133–151.

Here, apoptosis plays an important role to control the number of testicular cells, according to a hormonally controlled process that precisely regulates the homeostasis between Sertoli and germ cells. Spermatogonia are the most frequent apoptotic cells in the testis, but this process may also affect other cell types, such as spermatocytes and spermatids . Germ-cell apoptosis seems to be key in preserving the genomic integrity of male gametes, eliminating irreparably damaged cells[17] .

Seasonal breeders show a particular case of spermatogenesis regulation, as males are subject to circannual cycles of testis activation and involution. This phenomenon represents the natural demonstration that gametogenesis activity may be reversibly blocked in mammalian testes, a fact that increases the interest of studying the mechanisms controlling it, as a basis for the development of new male contraceptive strategies. In seasonal breeding males, testis inactivity coincides with low concentrations of plasma gonadotropins and reduced testicular testosterone production. Totally or partially reduced spermatogonial proliferation during the postbreeding period results in low germ-cell numbers and testis volume. The dramatic reduction by 80%–90% of the testis mass during the nonbreeding season strongly suggests that apoptosis may play a major part in the process. Apoptosis is also induced in response to meiotic arrest in nonseasonally reproducing species. Some studies have demonstrated that apoptosis contributes to testicular regression in hamsters , white-footedmice, and the European brown hare[18] . However, contradictory results have been reported on the role of apoptosis in the testicular regression of another seasonal breeder, the roe deer (Caprolus capreolus). Whereas some authors have indicated the existence of an inverse relationship between proliferation and apoptosis in this species, suggesting that apoptosis promotes testis involution[19], more recentstudies have shown that apoptosis is not the cause of seasonal involution of the roe deer testes[20] . It is worth mentioning Studies are very limited Regarding Occurrence of apatosis in testicular tissue in farmed birds and there is a pressing need for research in this area.

V. EFFECTIVE ANTIOXIDANTS IN DECREASING TESTICULAR OXIDATIVE STRESS

In addition to the major ROS processing enzymes, the testes rely heavily on small molecular weight antioxidant factors for protection against oxidative damage. These factors include ions and a wide variety of free radical scavengers, the nature of which are reviewed below:

Vitamin E (α-tocopherol) is a potent, lipophilic antioxidant, which is vital to protect and maintain mammalian sperm. Besides that, this element contributes greatly to the activity of Sertoli cell lines and spermatocytes . Similarly, vitamin C (ascorbic acid) plays an important part in the process of spermatogenesis. As a result, vitamins C or E deficiency leads to induction of testicular oxidative stress and hence disturbs spermatogenesis and

17 Gartner A, Boag PR, Blackwell TK. Germline survival and apoptosis.WormBook 2018; 4:1–20.
18 Strbenc M, Fazarinc G, Bavdek SV, Pogacnik A. Apoptosis and proliferation during seasonal testis regression in the brown hare (Lepus europaeus L.). Anat Histol Embryol 2003; 32:48–53.
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20 Blottner S, Scho¨n J, Roelants H. Apoptosis is not the cause of seasonaltesticular involution in roe deer. Cell Tissue Res 2007; 327:615–624.

production of testosterone[21] . In contrast, a study demonstrated that feeding with ascorbate caused stimulation of spermatogenesis and secretion of testosterone in healthy animals. In addition, use of vitamins C and E is highly effective to fight testicular oxidative stress due to exposure to oxidants such as arsenic, cadmium, endosulfan and alcohol and can considerably decrease the complications due to these substances[22]. A study demonstrated that vitamin E was effective on testicular function through suppressing lipid peroxidation in testicular and mitochondrial microsomes and fighting adverse effects of oxidative stress due to exposure to certain agents such as ozone gas, iron overload, intense exercise, aflatoxin, cyclophosplamide and formaldehyde[23] .

Zinc is recognized as an essential trace metal required for health ‘Zinc is an acknowledged antioxidant factor that as well as being a core constituent of free radical scavenging enzymes such as SOD and a recognized protector of sulfhydryl groups, is also thought to impair lipid peroxidation by displacing transition metals such as iron and copper from catalytic sites. In keeping with such a central antioxidant role, this element has a profound effect on the level of oxidative stress experienced by the testes. Thus rats fed a zinc deficient diet experience a decrease in testicular antioxidant potential and a concomitant increase of lipid peroxidation in this tissue.19 Conversely, zinc administration will counteract the oxidative stress created in the testes by exposure to lead.20-21 as well as the peroxidative damage induced by ischemia-reperfusion as a consequence testicular torsion-detorsion.22 Zinc administration has also been shown to attenuate the testicular oxidative DNA damage induced by cadmium as well as the decline in sperm production and testosterone secretion induced by this heavy metal.

Selenium and N-acetyl-cysteine,Selenium is an essential trace element in formation of sperm and testosterone biosynthesis . Selenium (Se) is an important trace mineral having many essential roles at the cellular and organismal levels in animal and human health. The biological effects of Se are mainly carried out by selenoproteins . As an essential component of selenoproteins, Se performs structural and enzymic roles; in the latter context it is well known for its catalytic and antioxidative functions[24].Selenoproteins help maintain normal sperm structure integrity. N-acetyl cysteine is a naturally occurring compound which comes from amino acid L-cysteine, and functionsas a precursor of glutathione peroxidase [25]. Placebo controlled clinical trial carried out in Iran and Tunisia showed that selenium supplementation improved sperm counts, concentration, motility and morphology as well as sperm concentration in infertile men [26].Safarinejad et al investigated the effect of selenium and N-acetyl-cysteine on 468 infertile men with idiopathic oligo-asthenoteratospermia. They were followed by a 30 weeks treatment period. In response to treatment, serum follicle-stimulating hormone decreased but serum testosterone and Inhibin B increased. In addition, all semen parameters significantly improved with selenium and N-acetyl-cysteine

21 Ko EY, Sabanegh ES. The Role of Over-the-Counter Supplements for the Treatment of Male Infertility-Fact or Fiction? J Androl. 2012;33:292–308. [PubMed] [Google Scholar]
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23 Linster CL, Van Schaftingen E. Vitamin C. FEBS J. 2007;274:1–22. [PubMed] [Google Scholar]
24 Keskes-Ammar L, Feki-Chakroun N, Rebai T, Sahnoun Z, Ghozzi H, Hammami S, et al. Sperm oxidative stress and the effect of an oral vitamin E and selenium supplement on semen quality in infertile men. Arch Androl. 2003;49:83–94
25 Mistry HD, Pipkin FB, Redman CW, Poston L. Selenium in reproductive health. Am J Obstet Gyn. 2012;206:21–30. [PubMed] [Google Scholar]
26 Sedigheh Ahmadi, Reihane Bashiri, Akram Ghadiri-Anari, Azadeh Nadjarzadeh.Antioxidant supplements and semen parameters: An evidence based reviewInt J Reprod Biomed (Yazd) 2016 Dec; 14(12): 729–736.
PMCID: PMC5203687

treatment. Administering selenium plus N-acetyl-cysteine resulted in further beneficial effects in semen parameters[27] .

L-carnitine (LC) or 3-aminobutyric acid is a naturally occurring compound and also a semi-essential vitamin like substance required for human metabolism. LC involvement in intermediary metabolism is essential for bioenergetic processes, where it has a major role in the formation of acyl carnitine esters of long-chain fatty acids[28] . The highest concentrations of LC exist in epididymis which is 2000 times higher than whole blood concentration. The high level of LC in epididymis is resulted from an active secretory process . Findings show a positive relationship between initial sperm movement and increased LC in epididymis and L-acetyl in sperm[29] .

CoQ10 also known as ubiquinone is an antioxidant. As a component of the electron transport chain, it participates in aerobic cellular respiration, which generates energy. This oil-soluble, vitamin-like substance is present in cell membrane and lipoproteins[30]. In recent years, the role of this vitamin-like antioxidant in male infertility has been discussed widely. it was found that the concentration of CoQ10 was correlated with key semen parameters such as sperm concentration, motility and morphology because the total antioxidant capacity improves. supplementing infertile men with CoQ10 does not increase live birth or pregnancy rates, but there is a global improvement in sperm parameters such as sperm concentration and motility and CoQ10 concentration in semen[31]-[32] .

VI. SPERM FATTY ACIDS IN FARMED BIRDS

Various studies have shown that the reproductive system is affected by the quality and quantity of fatty acids. Fatty acids have a direct effect on Diameter of spermatozoon tubes, Lumen diameter, number of Leydig cells, number of sertoli cells, number of spermatogonia, number of spermatocytes And the number of spermatids. Fatty acids are playing the significant roles in the maintenance of motility of sperm, membrane integrity of membrane as well as protection of sperm against the cold shock[33].

VII. IMPORTANCE OF ANTIOXIDANTS IN POULTRY DIET ENRICHMENT

27 Safarinejad MR, Safarinejad S. Efficacy of selenium and/or N-acetyl-cysteine for improving semen parameters in infertile men: a double-blind, placebo controlled, randomized study. J Urol. 2009;181:741–751. [PubMed] [Google Scholar]
28 Arduini A, Bonomini M, Savica V, Amato A, Zammit V. Carnitine in metabolic disease: potential for pharmacological intervention. Pharm Ther. 2008;120:149–156. [PubMed] [Google Scholar]
29 Radigue C, Es-Slami S, Soufir J. Relationship of carnitine transport across the epididymis to blood carnitine and androgens in rats. Arch Androl. 1996;37:27–31. [PubMed] [Google Scholar]
30 Ernster L, Forsmark-Andree P. Ubiquinol: an endogenous antioxidant in aerobic organisms. Clin Invest.1993;71:S60–S65. [PubMed] [Google Scholar]
31 Lafuente R, González-Comadrán M, Solà I, López G, Brassesco M, Carreras R, et al. Coenzyme Q10 and male infertility: a meta-analysis. J Assis Reprod Gen. 2013;30:1147–1156. [PMC free article] [PubMed] [Google Scholar]
32 Ahmadi, S., Bashiri, R., Ghadiri-Anari, A., & Nadjarzadeh, A. (2016). Antioxidant supplements and semen parameters: An evidence based review. International journal of reproductive biomedicine (Yazd, Iran), 14(12), 729–736.
33 Robinson J.R., Robinson M.F., Levander O.A. and Thomson C.D. (1985). Urinary excretion of selenium by New Zealand and North American human subjects on differing intakes. Am. J. Clin. Nutr. 41: 1023-1031

Animal health depends on many factors and recently it has been appreciated that diet plays a pivotal role in health maintenance and prevention of various diseases. Among many dietary factors, antioxidants have a special place being major players in the battle for animal survival, maintenance of animal health, productive and reproductive performance. This is largely because of the detrimental effects of free radicals and toxic products of their metabolism on various metabolic processes[34].

The process of fertilisation of ova is very complex and well regulated. For example, in order to be fertile animal spermatozoa should be characterised by high motility and acrosome integrity. Furthermore to be motile the spermatozoa should have intact mitochondria (energy-producing stations in the cell) and high membrane flexibility and fluidity. To maintain those membrane properties there is a need for high level of polyunsaturated fatty acids . In fact spermatozoa from all animal species are characterised by extremely high proportions of those fatty acids and as a result they become very vulnerable to oxidative stress due to overproduction of free radicals. To deal with those dangerous conditions the antioxidant system of the spermatozoa includes fat-soluble and water-soluble chain-breaking antioxidants as well as antioxidant enzymes. Understanding of involvement of selenium in maintenance of semen quality came from data on selenoproteins. In particular, there are several selenoproteins, which are found in spermatozoa. For example, glutathione peroxidases (GSH-Px), a family of 5 important antioxidant enzymes, are responsible for prevention of damaging effects of free radicals and toxic products of their metabolism on spermatozoa. The last (5th) member of this family of selenoproteins, so called sperm nuclear GSH-Px was identified only twoyears ago. It seems likely that thioredoxin reductase (TR) is also involved in antioxidant defence in spermatozoa, but there are no data available at present to confirm this[35]. Furthermore, a specific sperm capsular selenoprotein is located in the midpiece of spermatozoa. Recently it was identified as phospholipid hydroperoxide glutathione peroxidase, a form of Se-dependent GSH-Px. Since mitochondria are the main source of free radicals in the spermatozoa and they are located in midpiece, antioxidant protection there is a crucial factor for sperm motility and fertilizing ability. For example, Sedeficiency caused various sperm abnormality in this region resulting in decreased fertilising ability and organic selenium dietary supplementation is more efficient in improving semen morphology than selenite. Experiments with Sel-Plex in the cockerel diet proved the point: organic selenium also increases duration of fertility[36].

VIII. NATURAL ANTIOXIDANTS AND CHICKEN EMBRYONIC DEVELOPMENT

Chick embryo tissues contain a high proportion of highly polyunsaturated fatty acids in the lipid fraction and therefore need antioxidant defence. Tissues of newly hatched chicks express a range of antioxidant defences including natural antioxidants(vitamin E, carotenoids, glutathione, ascorbic acid) and antioxidant enzymes

34 Surai, Peter. (2007). Natural Antioxidants in Poultry Nutrition: New developments. Conference: 16th European Symposium on Poultry Nutrition

35 Surai, Peter & Cerolini, Silvia & Wishart, G.J. & Speake, B.K. & Noble, R.C. & Sparks, N.H.C.. (1998). Lipid and Antioxidant Composition of Chicken Semen and its Susceptibility to Peroxidation. Avian and Poultry Biology Reviews. 9. 11-23.

36 Agate D.D.. O'Dea E.E. and Rustad M.E. (2018). Effects of dietary selenium on laying hen fertility as assessed by the periviteline sperm hole assay. Proc. Poultry Research and Production Symposium, Alberta Poultry Researchh Centre, pp.1-4

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(superoxide dismutase, glutathione peroxidase and catalase) as well as antioxidant enzyme cofactors(Se, Zn, Mn and Fe)[37] . Of these, vitamin E, carotenoids and metals, including Se, are delivered from the maternal diet via the egg and the others are synthesised in the tissues. There are tissue-specific features in antioxidant composition. For example, the highest level of vitamin E was found in the liver and lowest in the brain. Carotenoids also were concentrated in the liver. However, brain was substantially enriched in ascorbic acid; and it was suggested that in the brain an effective recycling of vitamin E by ascorbic acid could maintain effective antioxidant protection even with low level of vitamin E. Highest Se-GSH-Px was observed in the liver and kidney, but lowest in the brain. In newly hatched

chicks Se concentrations in tissues can be placed in the following order:

liver>kidney>lung> heart>brain>muscle. Therefore, it has been suggested that brain lipid composition and antioxidant concentrations predispose this tissue to be most vulnerable to lipid peroxidation. It might well be that these features of the brain are involved in nutritional encephalomalacia development in the case of low vitamin E and Se supplementation. It is necessary to underline that maternal diet composition is a major determinant of antioxidant system development during embryogenesis and in early postnatal development. Vitamin E, carotenoids and selenium are transferred from feed into egg yolk and further to embryonic tissues. Our observations indicate that an increased antioxidant supplementation of the maternal diet can substantially increase their concentrations in developing chick tissues and significantly decreases susceptibility to lipid peroxidation. There is tissue specificity in Se transfer from egg to the embryo. For example, in contrast to the liver, there was only a trend (non-significant) toward higher Se accumulation in the brain of chickens hatched from the egg enriched in Se. It is important to mention that there was a positive effect of Se supplementation of the maternal diet on the levels of vitamin E in the liver, brain and blood plasma of day old chicks. A positive effect of Se supplementation of the maternal diet was seen at day 5 and 10 post-hatch when vitamin E concentrations in theliver and plasma were significantly elevated compared to controls. The mechanisms for this sparing is not clear, but could be related to Se antioxidant properties. It is also possible that Se can affect other aspects of vitamin E metabolism and transport to target tissues.

GSH-Px activity in the liver of day old chicks depends on Se supplied in the maternal diet. Low dietary Se was associated with decreased Se in egg yolk; and as a result Se-GSH-Px activity in the liver of newly hatched chicks significantly decreased[38] . An efficient carry-over of Se and vitamin E from hens to progeny was indicated by a significant increase in muscle Se, liver GSH-Px activity and vitamin E content at hatching .[39]. There was no difference in Se-GSH-Px activity in the liver in response to further increased Se supplementation (from 0.2 to 0.4 mg/kg), which probably means that inclusion of 0.2 mg/kg Se in the maternal diet provides enough Se to the egg

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and embryonic tissues to meet the requirement for the maximum Se-GSH-Px activity. GSH-Px activity in the liver increased throughout embryonic development

reaching maximum at time of hatching. In the liver of the newly hatched chick, Sedependent GSH-Px is the major form of the enzyme comprising about 61% of total activity. In the majority of the tissues of the newly hatched chick there was a highly significant correlation between Se level and the activity of Se-GSH-Px. In our study it has been shown that the effect of Se in the maternal diet is still apparent at 5 and 10 days of postnatal development. This finding suggests that Se accumulated in the liver of newly hatched chicks is actively used during the first days of postnatal development. It is possible to suggest that Se assimilation from the diet is low just after hatching and the chick relies on the reserves of the element accumulated during embryogenesis. Furthermore, our recent data from quail experiment indicate that increased Se levels in the diet as a result dietary organic Se supplementation are associated not only with increased Se level in tissues of newly hatched quail, but also at day 7 and 14 posthatch[40]. Postnatal development of the chick is associated with changes in the antioxidant defence strategy. The main antioxidant protection of newly hatched chicks is afforded through high concentrations of natural antioxidants, mainly vitamin E and in some cases (wild birds) carotenoids in tissues. However, during the first 10 days post-hatch, vitamin E and carotenoid concentrations in the chicken liver decreased 20-fold; and the same is true for turkeys, ducks and geese. Therefore to compensate for this decrease in antioxidant potency, activity of GSH-Px in the liver significantly increased. As a result, this Sedependent enzyme becomes the major player in antioxidant defence during postnatal development of the chicken.

Our data indicate that Se supplementation of the breeder diet at 0.2-0.4 ppm in the form of organic Se could provide substantial protection against free radicals and associated toxic metabolites to newly-hatched chicks. The benefit of organic Se use in breeder's dietlies in its efficient absorption, transport and accumulation in egg and embryonic tissues.This results in enhanced antioxidant status of the newly hatched chick. Since after hatching the levels of major natural antioxidants (vitamin E and carotenoids) in tissues progressivelydeclines, the antioxidant enzymes are a critical part of antioxidant defence. Therefore enhanced GSH-Px activity in tissues as a result of organic Se supplementation of the maternal diet could be considered an effective means of increasing chick viability posthatch. Enhancing antioxidant system capacity may also enhance immune response, which is extremely important at this period of chicken development. In general there is some evidence to show that in commercial conditions inclusion of organic selenium into the breeder's diet is associated with improved hatchability. Furthermore organic selenium supplementation of the maternal diet decreased chick mortality for the first two weeks posthatch confirming a vitality of the idea of relationship between antioxidant defences and chicken viability[41] .

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IX. CONCLUSIONS

Human, chickens, pigs, cows or any other animal species are exposed to free radical attack , Subsequent to that apoptosis in everyday life and that is why an integrated antioxidant system has been developed in every cell during evolution to prevent damages to biologically relevantmolecules including DNA, proteins and lipids. Some of antioxidants are synthesised inthe body, however, major source of antioxidants is our diet. From many hundred dietarycompounds possessing antioxidant activities selenium and vitamin E are considered tobuild a core of antioxidant defence. It has been appreciated that efficiency of antioxidantsdepends on their form in the diet. For example, in recent years it has been proven that organic selenium, for example in the form of Sel-Plex, has important advantages incomparison to inorganic selenium. It is interesting that the benefit of organic seleniumhas been proven practically for all species, including chicken, pigs, cows, sheeps and fish. It seems likely that an optimal combination of organic selenium and vitamin E in the diet is a key for an effective antioxidant defence. However, there is a need for further research in this field to establish those optimal combinations for each species depending on age, productivity and other relevant technological conditions In summary we can say, Free radicals' life-threatening attacks to the body's different organs can cause arterial occlusion and induction of oxidative stress and subsequently causing serious damage to tissues. Meanwhile, testicular tissue is highly predisposed to activity of free radicals and oxidative stress due to several reasons including high cell division rate, cell competition for oxygen rate, low oxygen pressure due to weakened vessels as well as high levels of unsaturated fatty acids. Furthermore, since the body's antioxidant system, including antioxidant enzymes such as SOD, catalase and GPX produced in the body is not able to neutralize all free radicals, the use of antioxidant supplements is recommended to fight adverse effects of oxidative stress, enhance spermatogenesis and increase enhance fertility.

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