KOREAN J. FOOD SCI. TECHNOL.
Vol. 5, No. 2 (1973)

Some Aspects of Dietary Garlic, Selenium and Tocopherol, in the Nutrition of Animal

by

Sea Yeol Chun

Radiation Biology Division, Korea Atomic Energy Research Institute, Seoul, Korea

(Received March. 15, 1973)

마늘, Se 및 비타민 E가 동물영양에 미치는 효과

全 世 烈

한국원자력연구소 방사선생물학연구실

(1973년 3월 15일 수리)

Abstract

Tocopherol, Se and garlic powder were dieted to hatched chick breeding. The Se content of certain organs is influenced by garlic powder supply. The high Se content for the testis was a function of the vitamin E uptake. Effect of low dose of Se on the growth and survival of rat were examined under the diet of 2 μg/ml of Se in drinking water either in the form of Na_2SeO_3 or Na_2SeO_4. The females were dead in early ages while the males were not influenced by dieting the selenite did not make males dying rapidly at early ages and males were less growth depressed. The previously known fact that garlic act as a tonics may be attributable to its high contents of Se and sulfur-containing amino acids which are closely related to vitamin E. Further details on the dietary mechanisms of the Se, vitamin E, and garlic powder are described in this paper.

Introduction

There has been a considerable interest in the determination of Se since the discovery that this element may have an essential function in mammals. In particular the connection between Se and vitamin E has attracted considerable attention. In contrast to the profound impace that Se has had in animal nutrition, very little is known about its role in human nutrition. The influence of Se[1] on reproduction has prompted research in this element. Se compounds have been found in nature[2] such as selenocysteine and selenomethionine however, the metabolic pathway of Se in animals has not been obtained. Vitamin E deficiencies and related conditions in animals can be conveniently differentiate into three groups disorders[3] which are prevented soley by vitamin E. These responsive to both vitamin E and Se. The biologically active from of Se in animals and its metabolic relationship with vitamin E remain obscure. Investigation on the chemical nature of seleno-compounds of plant origin[4~6] have indicated the presence of selenomethion-

ine. Selenotrisulfides are formed non-enzymatically from Selenite and sulfohydryl compounds[7] and of their possible occurence in animals[8] suggest that these forms of Se may also occur in plant materials. Investigation of the composition of garlic showed the presence of Se and sulfur containing amino acid.[9] Garlics are rich in Se combined sulfur amino acid. Some modern nutritional practices might promate marginal deficiency of an essential trace element on which homeostasis of cholosterol depends[10] Se deficiency syndrome uncomplicated by vitamin E deficiency has been produced in chicks by use of the crystalline amino acid diet of Thompson.[11] It was domonstrated the essential role of Se in the prophyloxis of exudative diathesis in chickens.[12~13] Symptoms of dietary liver necrosis in vitamin E deficient rat are prevented when garlic is fed as a Se and sulfur containing amino acid source, which was the starting point of intensive research on a new nutritive factor. Obviously, good results of vitamin E substitution by Se in these diseaes caused speculation on a similar type of action of the two essential nutritive factors. Organo-seleniated compound, in chick was named factor 3, completing the list of the protective factors, namely cysteine and vitamin E.[13~14] It is even thought today that the protective effect of cysteine is related to Se. Comprehensive study of the kinetics of ^{75}Se in the rat, designed to probe into the metabolic inter-relation between this trace element and vitamin E.[13] The study in normal animals with then be compared to animals in vitamin E deficiency of treated with large amounts of α-tocopherol. Microgram quantities of supplemental Se have been found to exert desirable effect on the growth and development of chicks.[12] Technique of Allway[14] have been combined in the method described here to allow rapid determination of submicrogram Se in rat. The requirement of the chick for Se and vitamin E interrelated. Scott[15] work has shown that the two nutrients spare each other. Se compounds appear to be involved in some unknown way in a carrier system for vitamin E retention.[16] Vitamin E has been postulated as maintaining body Se in an active form.[15]

Experimental Method

The experiments were conducted with white Leghorn one day old obtained from a commercial hatchery. The chicken were housed in incubator. Feed and water were supplied ad libitum. The composition of purified diet Se, vitamin E, and garlic powder has been described previously.[17~8] The group 1, and 2, was fed a standard diet without vitamin E as described by Scott.[18~9] The group 3 the controls were fed semi-synthetic diet without supplementation of selenite tocopherol and garlic. The group 4, 5, 6 both diet 5 ppm, of Se was added in the form of sodium selinite (Na_2SeO_3). The group 7, 8 was added in garlic powder 10 g/100 g feed. A semi-synthetic diet were supplied for up to four weeks according to Bieri[20~1]: This basal (diet Table I) was feed to two lots of 40 chicken.

Table 1. The semi-synthetic basal diet

Group 1.	Semi-synthetic diet+tocopherol 30 μg/kg feed.
Group 2.	Semi-synthetic diet+tocopherol 20 μg/kg feed.
Group 3.	Semi-synthetic diet without supplementation of Selenite or tocopherol, garlic.
Group 4.	Semi-synthetic diet+5 ppm Na_2SeO_3/kg feed.
Group 5.	Semi-synthetic diet+2 mg/Na_2SeO_3/kg feed.
Group 6.	Semi-synthetic diet+2 mg/Na_2SeO_3/kg feed +1.4% $CaHPO_4$.
Group 7.	Semi-synthetic diet+garlic powder 10 g/100 g feed.
Group 8.	Semi-synthetic diet+garlic powder 20 g/100 g feed.

Table 2. Basal diet chicken

Yellow corn	59. 0%
Soybean meal	30. 0%
Fish meal	5. 0%
Alfalfa meal	2. 0%
Calcium carbonate	1. 5%
Calcium phosphate ($CaHPO_4 \cdot 2H_2O$)	1.75%
Sodium chloride	0.45%
Premix	0. 3%

Vitamins minerals and antibiotics added into 100 mg diet: Vitamin: A_1, B_2, B_6, B_{12}, C, D.

Minerals: Mn, Zn, Fe, Cu, Co, I.

Table 3. Composition of basal diet

Dietary constituents	Percent %
Casein	15.00
Gelatin	10.00
Salt mixture	5.17
Vitamin mixture	0.50
Choline chloride 70% solution	0.20
Sucrose	65.21
Lard	1.00

The tissue sample were analyzed for Se by the method of Ewan.[22~23] Each experiment was of three weeks to 12 weeks duration. After the chicken were killed and samples of blood, skin, muscle, liver, kidney testes, ovary.

Material and Method

Sprague-Dawley rats (males and females) were maintained on semisynthetic basal diet and tap water ad libitum the daily in take averaged 10~12 g; Se in the form of Na_2SeO_3 was mixed with surcrose before being added to diet to give supplements of 0.10 0.25, 0.25 and 1.00 ppm. The basal diet was analyzed by neutron activation analysis for Se and found to contain 0.006 ppm. Male rats, weighing about 50 g were used in this study.[23~4] One day after receipt they were randomly placed into dietary group and provided food and tap water ad libitum. Diet fed were basal, 0.10 ppm Se with no vitamin E, 0.25 ppm Se 0.5 ppm Se and 1.00 ppm Se. Animals were weighed weekly. Food intake was not measured. 54 males and 56 females rats were given 2 μg/ml selenite and sodium selenite in drinking water. Each experiment was 30, 90 days duration, shows the weight gain of the group fed dietary levels of Se mice (male and females). Selenite and selenate in drinking water were fed in defferant age (30~90 day) to compare the difference occured in mean body weight. Previous analysis had shown that garlic contained Se. All organ samples produced were analysed by neutron activation. The organ sample were transfered to vial, seal and irradiated for 0.3 min together with Se standards in a neutron flux of approximately 3.8×10^{12} n/cm^2 S. After cooling of period of about 0.2 min, the irradiated samples were transferred to activity determined on the multichannel γ-spectrometry. A detailed description of the procedure has been given elsewhere.[25] Cysteine and methionine were estimated according to the method described in the literature [26].

Results

The Se content of the examined organs are shown in Table 4~6. Se was detected in all the preparation from the Se garlic powder supplemented experimental. Ehlig[28] have indicated that elemental Se may be in this unavailable form. The liver was found to retain the greatest quantity of the oral selenate initially. The total Se content of liver and muscle tissue of lamb fed increasing level of Se as selenite and tocopherol are presented in Table 4~5. In the presence of the generally recommended dietary vitamin E level of 15 to 20 IU/kg of diet, as little as 0.05 ppm of dietary Se prevents dificiency sign.[27] However, 0.1 ppm of dietary Se has been recommended as a more practical level. A procedure was empirically determined, where by sample of tissue could be analyzed for Se in a manner which did not require chemical separations after irradiation. However, dialysis prior to irradiation also enable the determination of Se bound to tissue proteins. Investigations on animals and on human beings carried out with the fresh drug have necessarily led to inconclusive results, in view of the extreme readiness with which alliin and its cleavage products decompose.[29] Garlic, for the alliin content of garlic of preparations made from it can be radily determined. Optimal doses of sodium selenite for chicken for development and weight gain was 1 mg/kg of feed daily during two periods of 10 day. The lethal doses were 80 to 150 mg/kg of feed.[30] The Se content (in ppm) of the examined chick organs are shown in table 5.4. Se was detected in all the preparation from the vitamin E, Se supplementeal experimental group. However, the value for the various tissues were rather different, and in certain organs they were strongly influenced by presence of vitamin E. Garlic diet group showed remarkably higher Se level in the liver, kidney and testes than in the blood, skin and muscle. The concentration of Se in garlic diet was significantly higher in garlic powder diet group aminals than in the vitamin E diet group. Testes and kidney the Se content was found to higher than in the blood or muscle. The Se levels in the testes, overy and kidney were about 0.3~0.6 ppm higher in the garlic diet than

in the diet of the controls. Liver tissue relatively large amount of Se could be detected, as expected due to the well known greater retention effect of the liver. All group in high Se contents were measured in the kidneys. High value were obtained in the testes independent of the vitamin E. supply than for garlic diet. The amount of Se in the kidney and testes was about ten times greater than in muscle. The amount in the ovary was about the same as that determined in the liver.

Table 4. Se content in various organs of chicks(ppm)

	Group 1	Group 2	Group 2 (control)
Blood	0.37	0.42	0.35
Skin	0.28	0.31	0.27
Muscle	0.23	0.26	0.21
Liver	1.32	1.58	1.25
Kidney	3.02	3.86	2.82
Testes	3.10	3.43	2.95
Ovary	0.87	1.12	1.04

The semi-synthetic basal diet was supplemented tocopherol 20 ppm Group 1 or tocopherol 30 ppm, Group 2 feed.

Table 5. Se content in various organs of chicks(ppm)

	Group 4	Group 5	Group 6	Control
Blood	0.38	0.47	0.49	0.36
Skin	0.28	0.33	0.35	0.27
Muscle	0.24	0.26	0.30	0.22
Liver	1.42	1.96	2.06	1.26
Kidney	3.04	3.58	3.75	2.84
Testes	3.60	3.88	3.97	3.21
Ovary	1.54	1.72	1.84	1.35

Group 4. The semi-synthetic basal diet was supplemented 5 ppm Na_2SeO_3/feed.

Group 5. The semi-synthetic basal diet was supplemented 2 mg Na_2Seo_3/feed

Group 6. The semi-synthetic basal diet was supplemented 2 mg Na_2 SeO_3/feed kg+1.4% $CaHPO_4$.

Leghorn chicken supplement of the ration with 0.1 mg of Na_2SeO_4/kg improved the growth of chicks and increased egg production.[31] The Se level in hen egg yolk was determined by neutron activation analysis ^{77m}Se. The Se content was in the range 0.18~1.17, μg per/g dry yolk. The biological variation may be due to differances in Se the chicks from the egg, or they may be caused by biochemical differences due to genetic variation. Changes in the Se concentration in various tissues and organs of chickens depending on tissue content in the diet were detected. Addition of sodium selenite at 0.1 mg/kg of diet increased the egg-laying capacity by 9.3%, increased the Se content in liver, ovary, heart, muscle, which in turn is compesnated by accumulation of Se in the eggs.[31] However, the metabolic pathway of Se in animals has not been obtained. This data showed aid in establishing nutritional requirements and biochemical function of Se in reproduction. After four weeks under experimental conditions chickens fed with vitamin E deficient diet showed symptoms of muscular dystrophy. Some were unable to stand. Some show tumor or paralysis of the legs, general weaknees and heavy breathing. White striation in the chest muscle were the rule. Such symptoms were not found in the group fed with vitamin E supplemented diet.[32] In few cases the above mentioned symptoms were also found in the selenite group. However, no sign of muscular dystrophy were seen in group receiving Se, P supplemented feed. Attempts by later workers to replace dietary cystine by related sulphur compounds led to the recogniton of the fact that methionine rather than cystine is an in dispensable constituent of the diet. The affinity of difference for ^{75}Se was studied after application of $Na_2{}^{75}SeO_3$ in vitro and in vivo. In vitro the relative ^{75}Se incorporation in the tissues increases with increasing $Na_2{}^{75}SeO_3$ concentration.

Table 6. Se content in various of chicks(μg/g)

	Group 7	Group 8	Control
Blood	0.36	0.38	0.35
Skin	0.26	0.29	0.26
Muscle	0.23	0.24	0.21
Liver	1.34	1.36	1.21
Kidney	2.96	3.15	2.72
Testes	3.78	3.94	3.18
Ovary	1.65	1.82	1.38

Group 7. The semi-synthetic basal diet was supplemented garlic powder 10 g/100 g feed.

Group 8. The semi-synthetic basal diet was supplemented garlic powder 20 g/100 g feed.

Table 7. Content of cystine and methionine in chicken organs(μg/g)

	Cysteine	Methionine
Hen egg	2.4	3.8
Chicken meat	0.9	2.5
Bone	1.6	2.1
Brain	1.3	3.0
Heart	3.8	2.3
Kidney	1.4	1.9
Liver	1.2	2.2
Lung	2.5	1.3
Ovary		1.5
Pancreas	3.1	1.8
Spleen	1.8	1.9

The study confirms the observations of Ewan[33] that in most instances increased dietary or carrier Se decreases retention of trace does. Se is an essential nutrient its own right. It is required in metabolic processes which are not protected by vitamin E. Indeed one of the important functions of vitamin E may be cencerned with protection trace of Se in the animal body.[32~34] Se as an essential nutrient for certain animals. Its possible benefit to health. The results of research examining metabolic relation among vitamin E, Se and S-containing amino acid, mainly methionine, are discussed.[35] The conversion of selenite into dimethyl-selenite is enzymic as well as non-enzymic in nature, since boiled microsoms are capable of acting as methyl effectors. The chief methyl donor is S-adenosyl-methienine: however, other methyl doners are effectively, the protein component of the diet may be replaced by a mixture of purified amino acids from which methionine and cystine have been excluded. A sulphinic acid $HO_2S \cdot CH_2 \cdot CH\ (NH)_2 \cdot CO_2H$, derived from cysteine has recently been isolated from rat brain[36] and is an important intermediate in the metabolic oxidation of this amino acid. It appears that in animal metabolism the sulfur undergoes oxidation and Se undergoes reduction. Both elements are required for normal metabolism.[37] It is suggested that vitamin E and the fatsoluble. Antioxidants make the methyl group of methionine more available for Se detoxification.[38] If the total sulfur concentration of an animal diet is constant, the Se contained in high-plants will be of nearly equal value to animals as the Se in the low-S-plant.[39]

1. Growth

Table 8 shows the weight gain of the group fed different dietary levels of Se. The group fed the basal diet had least gain, but such large variations were observed that significance was reached only between the basal and the 1.00 ppm Se group. Dietary Se was increased but satatisical significance was reached only when the 0.10 ppm group was compared with the other group. Animal receiving 0.10 ppm Se, these not fed vitamin E had some whole-body retention as those fed vitamin E.

2. Tissue retention

The influence of dietary Se on tissue retention is given in Fig 1. The 0.10 and 0.50 ppm Se group had two rat and the other had three each. The most striking finding was that, of whole-body ^{75}Se 9.5% per gram was found in the testes of the animals fed the basal diet. The percentage of the ^{75}Se in blood, muscle, liver and kidney. Testes and heart was greatest when the basal diet was fed compared to levels when the Se-Supplemented diets were fed. Abscence of vitamin E from the 0.10 ppm Se diet had no effect organ retention. The pattern of retention in the liver was unique. Animals fed the basal diet had very low liver retention. Rat had a higher Se content in their organ than sick animal (rat). In case, the highest Se level was in kidney and liver.

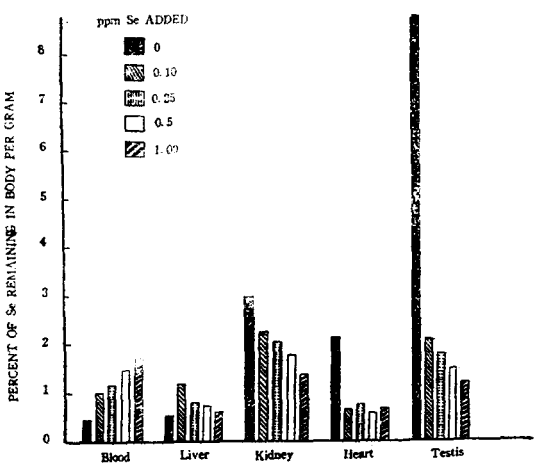

FIG. Effect of dietary Se on tissue retention. The 0.10 and 0.50 groups each had two rats

Table 8. Weight gain of animals fed different dietary levels of SeO_3

Diet (ppm Se added)	No. of animals	Mean wt gain (g)
0	3	164.2
0.10	2	192.7
0.25	3	192.5
0.50	2	193.7
1.00	3	194.0

70 day period.
Includes only animals receiving a submicrogram amo unt of intake Se.

Table 9. Mean weight of rats given Selenite and Selenate in drinking water

Age day	Controls	Selenite	Selenate
Males			
30	55.3 g	33.2 g	49.6 g
60	170.3	86.1	180.5
90	232.4	203.2	248.2
Females			
30	66.2	38.2	51.3
60	140.8	81.3	134.7
90	186.6	120.6	176.4

* 2 ppm Se element.
* 54 males and 56 females rats were given 2 μg/ml Se as sodium selenite in top water, drinking water.

Male mice given 2 ppm Se as selenite in drinking water grew more rapidly than their controls up to 90 day of age (Table 8). Female mice give selenite grow less rapidly than their controls after the 90 day interval. Effect of selenite and selenate on growth of rats. Selenite at 2 ppm Se depressed growth of rats of both sexes (Table 8). The same dose as selenate had no effect, except at the 30 day interval. Liver of the rats selenite and dying were grossly abnormal, with irregular means of fatty degeneration. Livers of the longest-lived female rats given selenite were less affected than were those of younger animals. There has been considerable discussion on the toxicity of Se, especially in respect to its action as an essential trace element for mammals.[39] These data, male mice tolerated selenite well, growth being enchanced. Females less Se growth being slightly depressed. Male rat tolerated selenite very poorly, females somewhat better. The dosage given in drinking water and may not be applicable to administration of the element in food. The intake of Se by these rate can be roughly calculated from the average measured dialy intake of water and the estimated intake of food, 6 g/100 g/day. Animals given selenate at 2 ppm Se drink 10.21±1.73 μg Se/100 g body weight/day. Those given selenite drink 11.5±1.13 μg Se/100 g/day. An additional 9.9 μg came from food. Contol consumed 7.5 g H_2O/100 g/day. Hopkins[40] reported low rates of growth in rat fed various purified diets containing 5 ppm Se as selenite for two weeks, but no depression of growth when a crude ration was fed. The present data suggest that selenite in water is more toxic to young rats than is selenite mixed with food, especially with a crude diet. Morality was higher in the selenite-fed animals, but both forms of Se depressed growth.

Table 10. Mean weights of mice Selenite in drinking water

Age	Controls	Selenite
30	24.4 g	25.3 g
Males 60	36.7	41.2
90	43.1	47.8
30	21.0	19.3
Females 60	27.3	29.2
90	35.3	34.5

Table 11. Se in animal tissue, wet weight. (μg/g)

Mammals	Heart	Lung	Liver	Kidney	Spleen
Rabbit	0.39	0.32	0.68	0.78	
Mouse	0.24	0.30	0.41	0.72	0.14
Rat	0.26	0.31	0.39	0.74	0.16

Animal from this area showed considerable Se in five tissue. The concentration in heart and spleen were generally similar, but that in liver was greater and that in kidney about three time higher than the others. Species difference appeared. Se was observed in the following organs, listed in decreasing order of content kidney, testes, liver, ovary, blood muscles. In the test group slight deviations from this order were

found. These result agree well with Grants[41] experiments with pig. All human blood and all sample of human tissue were analyzed. Miller[42] found high concentrations of Se in kidney and liver tissue of pigs. Se content of 5 mg/kg in food or 0.5 mg/kg in milk or drinking water may be toxic for man. High contents of Se are found in kidney and in fishes. Daily human intake on a standard diet was 62 μg. The calculated human body burden of Se was 14.6 mg. Wild animals contented two three times the human levels. Kindeys had the highest amounts. ^{75}Se as Na_2SeO_3 was adminstered orally to rats under different nutritional conditions. The active form of Se may be selenite form of Se may be selenite form part of the active center of an uncharacterized class of catalytically active nonheme-Fe proteins that are protected from oxidation vivo by vitamin E.

Discussion

The present need is to determine which biochemical reactions would be consistent with known chemical properties of sulfo and seleno-amino acid or vitamin E. Metabolism and function of Se in animals reviewed the evidence that animal can reduce selenates and selenites, from seleno-amino acid incorporate these seleno-amino acid into proteins.[3] According to Schwarz[3] rats deprived of Se but fed adequate tocopherol fail to grow, adrenal atrophy. On the other hand, rats deficient in both tocopherol and Se develop hepatic necrosis. An organo-seleniated compound, different from selenomethionine or selenocystine, has a highly protective effect against liver necrosis in vitamin E deficient rat: In seleniated compounds test Schwartz[43] indicated that the 7-7-diseleno-divaleric acid exhibited the highest protective effect in animals and also yielded promising results in clinical trials in children. This provides only a partial parallel in the physiological effect of Se and α-tocopherol. Deficiency of each in the rat produces decreased tissue levels of ubiquinone (coenzyme Q).[44~5] Ubiquinone synthesis has two alternate biochemical pathways, one dependent on the tocopherol and one on Se, or that Se functions as a lipid antioxidant.[45] Although under the similarity exist, it is becoming increasingly apparent that where are biochemical difference in the responses to coenzyme Q and vitamin E. Much of the Se in the animals is tightly bound in the proteins.[46] The clinical success of the application in many different diseases caused by vitamin E deficiency complicated the picture of the biochemical mechanism for the Se and vitamin E function. The syndrome is characterized by poor growth, poor feathering, and mortality. Vitamin E absorption is impaired simultaneously, causing a fall in plasma tocophenol level. Vitamin E deficiency alone has been shown to cause no pancreatic alterations in the chick.[15] Both nutrients also may function in the control of peroxidation and of free radical formation.[27,49]

It is concluded that Se deficiency alone was responsible for the pancreatic pathology.[47] When Se was deficient, the lesions appeared. Two Se and vitamin E dificiency diseases bovine nutritional liver necrosis.[47] These two nutrients have been postulated to function in intracellular oxidation.[48] This ultrastructural pathology is prevented by feeding vitamin E or Se.[49] Hegsted[50] have reported whole-body studies on rats given ^{75}Se selenomethionine. They found that the administration of methionine did not influence retention of the ^{75}Se but that inorganic Se did. A recent paper has shown that several forms of organic Se, including selenomethionine, share the same urinary metabolites with selenite.[51] These studies plus the conclusion of Waterlow[52] that ^{75}Se administered as ^{75}Se-selenomethionine is extensively reutilized suggest that selenomethonine is extensively broken down and that Se then enters other metabolic pathways which are influenced by the Se status of the infant or the rat. The recent identification of a major urinary metabolite of Se by two groups[53~4] as trimethylselenonium together with the present data and previous work[55] suggest that the activity of the pathway for the production of trimethylselenonim is directly related to dietary Se intake. Dimethyl selenide is an intermediate in the pathway and that the conversion of dimethyl selenide to trimethylselenonium is rate-limiting under conditions of Se excess, methanethiol, together with dimethyldisulphide, is a metabolic product of the wood-rotting fungus, Schizophyllum Communo[61], and there are indications that it may be a catabolite of methionine in animals.

Calombetti[56] have reported comparative study of the radioprodective efficiency of sulfur and Se on aliphatic chains. Cystine. methionine selenocystine, and

Table 12. Typical radioprotective effect of chemical substances

		Substance name	Effective objectives	Effective amounts (mg/kg body wt)
Sulfur contain Substance	Cysteine	Cysteine	Above molecular level	950~1200 ip
	Cysteamine	Cysteamine		75~ 250 ip
	Group	Aminoethyliso-thiouronium bromide-hydrobromide (AET)		
		Glutathione		800~1000 ip
		Dithiocarbamate		500~ 600 ip
		Thiourea		2500 ip

ip : Intraperitoneal injection.

Selenomethionine were submitted to X-ray irradiation and E.S.R. spectra were analyzed. Chemicals radioprotection afforded by Se-contaning compound in biological and chemical systems. The order of reactivity of Se compounds with radicals appears to parallel their known radioprotective ability in biological systems and emphasizes the need for an under standing of radiation chemical process to aid the understanding or radiobiological ones.[57] The presence of high amount of Se compounds in garlic suggest that these compound have a powerful radioprotective action processes to aid the understanding. Desai[58] found the same binding of selenite to the gamma globulin fraction, in chicks treated with 500 μg selenite. The authors advance the hypothesis of a seleno-lipoprotein migrating in the gamma zone and acting as a carrier of vitamin E. The protective effect of cysteamine (mercaptoethlamine, MEA) against whole-body irradiation has been well established by several groups[59~60]. We have tried to find out by a series of experiments how the protective efficiency of MEA is affected when mice are exposed to partial body irradiation. Since the allin content of the drug runs approximately paralled to its sulfur content, it was possible to determine, by performing sulfur analysis on samples of various origins, which specimen of the drug was most suitable for obtaining alliin.[61] We still know very little about the enzymes responsible for the formation and decomposition of methionine, which plays an important part in the process of transmethylation.[62] All living organisms require sulphur in some suit able form, and in higher animals this need is met by the amino acids L-cysteine, L-cystine and L-methionine, together with the heterocyclic compoundes biotin and thiamine. Mosts and micro-organisms are less exacting that animals in their sulphur requirements, for these can often be satisfied by the provision of inorganic sulphate. In most living organisms the amount of energy contributed by sulphur compounds is small by comparison with that derived from other sources. Growth experiments have also been used in the study of compounds which compete with normal, sulphur-containing metabolites, or which deprive an animal of a particular sulphur compound by combining with it. The Se requirements of animals are normally conveted by conventional feeds. Solution mixtures of the radiation protectors cystine or Se-cystine or protamine were preperation. A roview Se is an essential nutrient in its own right. It is required in metabolic process which are not protected by vitamin E. Indeed, one of the important function of Vitamin E may be concerned with protection of traces of Se in the animal body. Further the data obtained by Lee[62] indicate that the garlic being about increase of body weight and release the hyperpiesia when garlic was administe to rat. When garlic was adminstered to rat. It was observed that the semen producibility in spermatozoin was much accelerated than tn spermate-gonien comparing the control. It means that the germ cell in spermaduct become tenic to act semen vigourousy. It is known that vitamin E acts not only a role of preventing sterility but also a role of nourishing the wall of the capillary vessels to promote blood circulation. The previeusly known fact that garlic acts as a tonics may be attributable to its high contents of Se and sulfur-

containing amino acids which are closely related to vitamin E.

요 약

마늘은 강장(强壯), 강정(强精) 식품으로 인정되어 왔으나 그 원인은 아직 미지이다. 여기에는 비타민 E와 밀접한 연관성이 있는 Se 함유아미노산에 기인됨을 가정하고 병아리에 비타민 E, Se 및 마늘을 투여하여 각 장기에 대한 Se의 함량을 조사하였다. 마늘을 투여한 동물은 Se 및 비타민 E와 Se투여군보다 Se함량이 많으며 특히 정소에는 수배이나 축적됨으로 정력제로 될수 있는 요인이 된다고 본다. 쥐에 Se를 2 μg/ml (Na_2SeO_3 Na_2SeO_4) 투여하여 성장 및 생존에 미치는 영향을 조사하였다. Se 투여군에서 숫쥐는 성장 및 치사에 영향이 없지만 암쥐는 성장이 훨씬 저하되었다. 본 실험결과 마늘이 정력제에 작용을 하는 것은 Se함유 아미노산 함량이 풍부함으로 이것이 비타민 E의 작용과 같이 불임(不姙)을 막고 노쇠한 모세혈관을 회복시키는 역할을 상승시켜 주는 것으로 간주된다. 여기서는 Se, 비타민 E, 마늘에 대한 동물의 기능에 영향을 관찰 한 것을 보고 한다.

◇

The author is deeply indebted to Dr Sang-Joou Shinn and Dr Keung-Shik Park, in carrying out this work.

Referance

1) Nesheim, M. C. and Scott, M. L.: *Fed proc.*, **20**, 674 (1961).
2) McConnel, K. P. and Wabnitz, C. H.: *Biochem. Biophy. Acta.*, **86**, 182 (1964).
3) Schwarz, K.: *Fed. Proc.*, **24**, 58 (1965).
4) Peterson, P. J. and Butler, G. W.: *Aust. J. Biol. Sci.*, **15**, 126 (1962).
5) Schrift, A.: *Nature*, **201**, 1304 (1964).
6) Jenkins, K. J. and Hidiroglou, H.: *Can. J. Biochem.*, **45**, 1027 (197).
7) Gather, H. E.: *Biochemistry*, **46**, 1417 (1968).
8) Jenkins, K. J.: *Can. J. Biochem.*, **46**, 1417 (1968).
9) Sea Yeol Chun: *Korean J. Food Sci Technol.*, **5**, 55 (1973).
10) Schroeder, H. A.: *J. Nutr.*, **94**, 475 (1968).
11) Thompson, J. N. and M. L. Scott.: *J. Nutr.* **100**, 797 (1970).
12) Patterson, E. L. Milstry, R., and Stokstad, E. L.: *Proc, Soc. Exptl. Biol. Med.*, **95**, 617 (1957).
13) Imbach, A. and Sternberg, J.: *Int. J. Appl. Radiat., & Isotope*, **18**, 545 (1967).
14) Allaway, W. H. and Cary, E. E.: *Anal. Chem.*, **36**, 1359 (1964).
15) Scott, M. L.: *Int. Z Vitaminforsch.*, **40**, 334 (1970).
16) Desai I. D. and M. L. Scott.: *Arch. Biochem. Biophy.*, **110**, 309 (1965).
17) Jekins, K. J., Hill, D. C., Hutocheson, L. M. and Branion, H. D.: *Poultry Sci.*, **41**, 61 (1962).
18) Scott, M. L., Bieri, J. G. and Schwarz, K.: *Poultry Sci.*, **36**, 1155 (1957).
19) Scott, M. L., Hill, P. W., and Norris, L. C.: *J. Nutr.*, **56**, 387 (1955).
20) Bieri, J. G.: *Am. J. Clin. Nutr.*, **9**, 89 (1961).
21) Bieri, J. G., DAM, H., Prange, J. and Sonpregaard, E.: *Acta Physiol. Scand.*, **52**, 36 (1961).
22) Ewan, R. C., Baumawn, C. A. and Pape, A. L.: *J. Nutr.*, **9**, 549 (1967).
23) Ewan, R. C., Baumawn, C. A. and Rape, A. L.: *J. Agr. Food Chem.*, **16**, 212 (1968).
24) Peterson, P. J. and Spedding, D. J.: *New Zealand. J. Agr. Res.*, **6**, 13 (1963).
25) Chun, Sea Yoel: *Korean J. Food Sci Technol.*, **4**, 61 (1972).
26) Hegazi,S. M. and Salem, S. A.: *J. Sci. Fd Agr.*, **23**, 497 (1972).
27) Thompson, J. N. and M. L. Scott.: *J. Nut.* **97**, 335 (1969).
28) Ehling, C. F., Hogue, D. E., Allaway, W. H.: *J. Nutr.*, **92**, 121 (1967).
29) Patrick, H.: *Radioisotopes in animal nutrition & phsiyology*, Amsterdam, (1965).
30) Andreev, M., Minina, L. and Bronmikova, K.: *Vet. Med. Nauki.*, **7**, 19 (1970). *In Chem, Abstr* (Se). 1626 (1971).
31) Cals, I. Pelekis, Vestn, Sel. Skokhoz, Nauki., **16**, 23(1971). *In Chem Abstr.*, (Se)No. 19863 (1972).
32) Giese, W. Cappelle, W., and Hill, H.: *Trace Mineral Studies with Isotopes in Domestic Animals.* Proc. Intern. Atom Energy Agency: Vienna, Austria, p.97
33) Ewan, R. C. Pope, A. L. and Baumann, C. A.: *J. Nutr.*, **91**, 547 (1967).
34) Scott, M. L.: *Fat-Soluble Vitam. in Proc. Symp..* (1969) Pub. 359 (1970).
35) Frost, Douglas, V.: *Feedstuff.*, **43**, 12 (1971).
36) Paulson, G. D., Beumann, C. A. and Pope, A. L.:

J. Animal Sci., **25**, 1054 (1966).
37) Wanger Philip. D.: *Sulphur Inst., J.* **6**, 6 (1970).
38) Levander, O. A. and Morris, V. C.: *J. Nutr.*, **100**, 1111 (1970).
39) Rosenfeld, I. and O. A. Beath,: *Selenium*, (1964). Academic press New York.
40) Hopkins, L. L. Jr., Pope, A. L. and Baumann. C. A.: *J. Nutr.*, **88**, 123 (1966).
41) Grant, C. A.,A., Thafvelin, B. and Christell, R.: *Acta. Pharmacol., et Toxicol.*, **18**, 285 (1961).
42) Miller, W. T. and Schoenig, H. W.: *J. Agr. Res.*, **56**, 831 (1938).
43) Schwarz, K.: *Proc. Fedn. Am. SOCS Exp. Biol.*, **24**, 58 (1965).
44) Olson, R. E.: *Fed. Proc.*, **24**, 85 (1965).
45) Green, J., Diplock, A. J. Bunyan, J., Edwin, E. E. and Mchale, D.: *Nature*, **189**, 318 (1961).
46) Underwood, E. J.; *Trace Element in Human and Animal Nutrition* 2nd Ed., New York, Academic Press (1962).
47) Cheville, N. F.: *Pathol Vet.*, **3**. 208 (1966).
48) Tappel. A. A. Caldwell,: First Int. *Symp. Se in Biomed.*, Oregon State Univ. P. 345
49) Todd, G. C. and L. Krook: *Pathol Vet*, **3**, 379 (1966).
50) Hegsted, D. M. and Said, A. K.: *Proc. Soc. Exp. Biol. Med.*, **133**, 1388 (1970).
51) Palmer, I. S. R. P., Gunsalas, Halverson, A. W. and Olson, O. E.: *Biochim. Biophys. Acta.*, **208**, 260 (1970).
52) Waterlow, J. C., Garrow, J. S. and Millward, D. J.: *Clin. Sci.*, **36**, 489 (1969).
53) Palmer, I. S., Gunsalas, R. P. and Millward, D. J.: *Biochim. Biophys Acta.*, **177**, 336 (1969).
54) Byard, J. L.: *Arch. Bioch. Biophys.*, **130**, 556 (1969).
55) Ganther, H. E., Levander, O. A. and Baumann, C. A.: *J. Nutr.*, **88**, 55 (1966).
56) Calombetti, G. and Falcone, G.: *Quad. Ric. Sci.*, **57**, 136 (1968).
57) Badiello, R., Breciia, A.: *Radiat. Prot. Sensitization*, (Italy). Proc Int. Symp. 2nd. (1968).
58) Dessi, I. D. and Scott, M. L.: *Archs Biochem. Biophys.*, **10**, 309 (1965).
59) Baco, Z. M.: *Chemical Protection against Ionizing Radiation*, Thomas, C., Springfield III (1965).
60) Melching, M. J. and Streffer, D.: *Advance in Drug Research* (Jucker, E. Ed) 9:10 Birchauser, Basel (1966).
61) Arthur, Stoll. and Ewald. Seebeck: *Advance enzymol*, **11**, 377 (1951).
62) Meyer, A. and Vigneaud, V. du; *J. Biol. Chem.*, **143**, 375 (1942).
63) Zin-Soon Lee: *Seoul University Papers*, **5**, 144 (1957).