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Effects of Various Levels of Dietary Selenium and Arsenic on Chickens

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EFFECTS OF VARIOUS LEVELS OF DIETARY SELENIUM AND ARSENIC ON CHICKENS

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AND ARSENIC ON CHICKENS

This thesis is approved as a creditable and independent investigation by a candidate for the degree, Doctor of Philosophy, and is acceptable as meeting the thesis requirements for this degree, but without implying that the conclusions reached by the candidate are necessarily the conclusions of the major department.

EFFECTS OF VARIOUS LEVELS OF DIETARY SELENIUM
AND ARSENIC ON CHICKENS

Abstract

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Various levels of selenium and arsenic additions were made to several types of chicken diets to study their effects on growth and reproduction criteria. Three experiments were conducted. The first study involved day-old chicks and terminated at 64 weeks of age. These yearling hens were then used for the second experiment which terminated when the hens were 104 weeks old. The third study was initiated with 20 week old pullets and terminated when they were 52 weeks old.

Feeding purified diets resulted in slower chick growth and lower mature body weights of hens. Selenium additions of 2.0 ppm resulted in hens weighing slightly less throughout the production periods. Those fed 8 ppm weighed even less. This weight difference due to 8 ppm Se was not evident when 15 ppm As was added to a corn-soy diet or with 8 ppm As added to the purified diets.

Lower levels of selenium additions (0.1, 0.2 and 2.0 ppm) were not detrimental to egg production. In one experiment a significant ($P < 0.01$) improvement in egg production was obtained by adding 2 ppm Se to a corn-soy diet. Egg production was lowered when 8 ppm Se was added. Usually this depression was overcome by including arsenic. Purified diets resulted in significantly ($P < 0.01$) lower egg production. A purified type diet composed of glucose and a combination of isolated

soy protein and Torula yeast allowed for performance superior to that obtained with either of the protein sources used individually.

Addition of 2 ppm or 8 ppm Se to corn-soy diets resulted in significantly ($P < 0.01$) smaller eggs. Lower additions (0.1 and 1.0 ppm) of Se did not significantly reduce egg size, but in many cases eggs from hens fed 1.0 ppm Se were somewhat smaller. Egg size was also lowered when the higher Se levels were used in the purified diets.

Mortality was higher when purified diets were fed. Two ppm Se allowed for lower mortality during the laying periods of two experiments. Highest death loss occurred when 8 ppm Se was fed. Arsenic additions usually restored mortality to a level similar to that with the unsupplemented basal diets. No significant effect on mortality was observed with 0.1 and 1.0 ppm Se.

Eight ppm Se reduced fertility which could be restored by including arsenic. Although a slight reduction in egg fertility occurred in one experiment with 2 ppm Se, there was no effect on fertility with lower Se additions.

There was no adverse effect on hatchability of fertile eggs when 2 ppm or less Se was added. However, when 8 ppm Se was included there was a dramatic toxic effect on the embryos. This effect was only partly overcome by adding arsenic with the 8 ppm selenium.

Egg and tissue selenium increased as dietary additions were increased. Feathers, kidney and liver contained the highest concentrations of selenium. Lower amounts were found in thigh and breast muscle.

In most cases, arsenic lowered tissue selenium levels. Eggs did not accumulate higher levels of selenium as the hens aged. When 0.1, 0.2 and 2.0 ppm Se were added to purified diets containing little selenium, the resulting eggs contained selenium levels similar to those found in eggs produced by hens receiving the unsupplemented corn-soy diet.

Selenium added to a low Se diet increased egg or tissue selenium, whereas Se additions to a diet containing fair amounts of selenium had little effect on the amount deposited in tissues and eggs.

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INTRODUCTION

Selenium is a trace element regarded by most authorities as necessary for proper animal nutrition. Historical accounts as early as 1856 indicate that cattle or horses consuming excessive amounts either died or were adversely affected by loss of hair and sloughing of hooves. Franke (1934) describes the symptoms of excessive organic selenium consumption and the economic losses due to "Alkali Disease". Areas of the Great Plains and Rocky Mountain states have soils which contain 20 ppm or more of selenium. (Moxon et al., 1939). Certain seleniferous soils do, however, vary in their available selenium. (Olson et al., 1942). Plants such as Astragalus racemosus serve as indicators of high soil selenium. These indicator plants may take up and accumulate selenium to levels of 1000-4000 ppm.

Various means have been used to overcome the problems encountered in areas of high soil selenium or with high selenium feed. Among the somewhat successful methods used are administration of arsenic, use of high protein diets, use of linseed oil meal in the diet, or pasture management which allows grazing of high selenium areas in late season.

More recently Schwarz and Foltz (1957) showed selenium to be a part of an essential activity called Factor 3 and necessary to prevent liver necrosis in rats. Subsequent investigations have shown low levels (0.05-0.10 ppm) of selenium to be desirable and indeed essential.

Thompson and Scott (1969) demonstrated that selenium was required for growth of chicks even in the presence of ample amounts of vitamin E. Selenium and/or vitamin E deficiency are related to several nutritional diseases. These are nutritional muscular dystrophy, encephalomalacia and exudative diathesis. Perhaps current practices of high temperature grain drying and confinement housing lower vitamin E intake by swine and poultry and thus deficiency problems are aggravated.

The purposes of the present study were to further establish the influence of arsenic using marginally toxic selenium diets fed to chicks and laying hens in a life cycle study. Effects of lower selenium supplements on reproduction, production, liveability, and tissue and egg deposition were also studied.

LITERATURE REVIEW

Chemical and Physical Properties of Selenium

Selenium, a non-metal, was discovered in 1817 by Berzelius in flue dust of pyrite burners. It is a naturally occurring substance found in rocks, soil, plants, animals and manure in four different valences or oxidation states; -2, selenides; 0, elemental Se; +4 selenites; and +6, the selenates (Allaway, 1968). Selenites readily accept electrons and are reduced to elemental Se or to organic selenides. Organic selenides donate electrons and their Se is oxidized to higher valences. Selenites are tightly bound in insoluble complexes by hydrous oxides of iron at pH 4 to 8.5.

The positions of phosphorus and arsenic, and those of sulfur and selenium in the periodic table indicate how similar they might be chemically. Arsenicals are known to catalyze phosphorus reactions. Perhaps selenium and sulfur may be related in the same way. Phosphorus and arsenic are known to protect against selenium toxicity if given at a proper level as reviewed by Frost (1967).

Organic chemistry of selenium and sulfur are quite similar. However, organic selenium compounds are less stable than their sulfur analogs. Selenium has many volatile forms at low temperatures. Animals fed high levels of selenium, seleniferous plants and organic selenium compounds give off characteristic odors which are believed to be volatile selenium (Allaway, 1968).

Selenium is toxic to animals in its oxidized or reduced forms but not in its reddish colored elemental form. Animals injected with selenate reduce the Se and give off methyl selenides in their breath (McConnell, 1941) and reviewed by Ganther (1965). It is doubtful that oxidized forms of selenium are converted to seleno-amino acids as evidenced by studies with rabbits (Cummins and Martin, 1967).

Soil selenium is quite water soluble. Selenates and organic selenium are readily taken up by plants but the selenites are tightly bound to iron. Soils in seleniferous areas may vary considerably in their available selenium (Olson et al., 1942). Plants can convert inorganic selenium to organic or organic selenium to inorganic forms (Hamilton and Beath, 1963). Allaway et al. (1967) suggests that 75% of plant selenium is in the form of selenomethionine with 20% in other soluble forms. Animals have about 10% of their total selenium in liver, 10% in blood, 20% in muscle and 25% in skin including hair, fleece or feathers.

Of the various grains tested, wheat was the most efficient selenium absorber (Hamiliton and Beath, 1963). Selenium contents of corn, rape, flax, and safflower grain were relatively low in these greenhouse studies. Sunflowers absorbed relatively large amounts of selenium. Other workers (Ehlig et al., 1968) report little differences in crop plant selenium if grown in low selenium soil.

Selenium-Relationships to Other Nutrients

In laboratory experiments, selenium responsive diseases have been

produced when the diet was deficient in selenium and vitamin E. Rarely have these diseases been produced when animals received a normal amount of vitamin E. Vitamin E apparently has a sparing effect on the selenium requirement (Scott, 1969). The selenium requirement for prevention of exudative diathesis in chicks is spared by vitamin E.

The mechanism by which vitamin E spares selenium is unknown. Table 1 in the Appendix illustrates the various inter-relationships among selenium, vitamin E, polyunsaturated fatty acids, cystine and antioxidants. In order to explain the mode of action of selenium one must take into consideration the relationships to vitamin E and other nutrients. Several suggestions by Schwarz (1965) may explain the interchangeability of selenium and vitamin E: perhaps one is a precursor of the other's metabolic activity, one protects or spares the other, they truly substitute for each other, they catalyze different alternate pathways of metabolism, or finally they catalyze closely adjacent steps in a chain of reactions.

Vitamin E is known to serve as a biological antioxidant. Either vitamin E or ethoxyquin (or other natural or synthetic fat-soluble antioxidants with properities similar to vitamin E) can function in prevention of encephalomalacia, steatitis, erythrocyte hemolysis and of rat incisor depigmentation (Scott, 1969). Severity of these vitamin E deficiency diseases can be increased by adding linoleic acid to the diet. Tappel (1962) found that in absence of suitable antioxidants, tissues are damaged by proliferating free radicals and lipid

peroxides. Sulfhydryl enzymes, labile vitamins and lysosomal membranes are thought to be destroyed. Therefore if the chemical composition of a tissue is such that it is more prone to autoxidation, there is a greater need for antioxidants (Witting, 1965).

Tappel and Caldwell (1967) proposed that some selenium compounds have unique oxidation-reduction properties. They suggested mechanisms by which selenium metabolites might inhibit autoxidation. For example, if unsaturated lipids produce peroxy or other radicals during peroxidation these radicals are capable of initiating a chain reaction leading to extensive decomposition. Perhaps the free radicals could be destroyed when they are first formed by an antioxidant such as a selenoprotein.

Certain other conditions illustrate the sparing effect of vitamin E on the selenium requirement. However, selenium appears to be the primary factor required to prevent myopathies in the gizzard and heart of turkeys, to prevent liver necrosis in rats and to prevent white muscle disease in lambs and calves (Scott, 1969). The amount of selenium needed to prevent the above conditions is not increased by linoleic acid or decreased by antioxidants. The condition of exudative diathesis in chicks can be alleviated by selenium; thus sparing the requirement for vitamin E. In selenium deficient diets, vitamin E does not prevent or cure exudative diathesis in chicks. The addition of 0.05 ppm selenium (as sodium selenite) to the diet prevents the disease.

Thompson and Scott (1969) found that the chick's quantitative need for selenium could not be arrived at until the vitamin E levels were set at

specific levels. When vitamin E was left out of a diet or replaced by an antioxidant, chicks required at least 0.05 ppm selenium in the diet. If inefficient absorption occurred, 0.1 ppm selenium was needed. When diets contained 100 ppm vitamin E, the selenium requirement was less than 0.01 ppm, with 10 ppm vitamin E the requirement was more than 0.02 ppm and with no vitamin E 0.05 ppm selenium was needed by chicks.

Other work has shown that selenium increases blood and tissue uptake of vitamin E as much as 100 X more than with chicks not given selenium in their amino acid diet. (Scott, 1969). Earlier work by Desai and Scott (1965) showed that activities of labeled selenium and tocopherols followed each other quite closely in the serum proteins. Indications were that vitamin E may be carried by a selenolipoprotein fraction which is associated with serum gamma globulin. This compound may function in the absorption, retention, prevention of destruction and transfer across cell membranes of d-alpha-tocopherol and thereby enhance its biological activity in the blood and body cells.

Sulfur amino acids may also be related to selenium and vitamin E. Nutritional muscular dystrophy in chicks has been shown to be prevented by either vitamin E or cystine. Many cystine supplies have been found to be contaminated with selenium, however. Apparently, the sulfur amino acid has no effect on muscular dystrophy in other animals. Selenium spares the vitamin E requirement but by itself won't prevent muscular dystrophy. The complexity of the problem was shown by Calvert et al. (1964), in that by adding graded levels of linoleic acid the incidence

and severity of muscular dystrophy increased. The vitamin E requirement for chicks was increased with additions of linoleic up to 0.5% of the diet. No further increase in vitamin E requirement was found when linoleic was increased from 0.5% to 2.5%. Oleic acid had no effect in producing muscular lesions.

Earlier work by Scott et al. (1955) in developing a chick diet to study uncomplicated vitamin E deficiency showed that the methionine and cystine levels in the diet were more closely related to erosion of the gizzard lining than was vitamin E. Other work (Scott and Calvert, 1962) found cystine but not methionine to be effective in preventing muscular dystrophy in chicks. By increasing the arginine level of the diet, more methionine was needed for certain metabolic reactions and thereby reduced conversion of methionine to cystine. Perhaps in a high arginine diet more methionine is needed to prevent muscular dystrophy. Other evidence of a sulfur amino acid-selenium-vitamin E relationship was shown by Supplee (1966). He found a characteristic feather abnormality in poults fed a diet low in selenium and vitamin E. Feathers normally contain relatively high levels of sulfur amino acids. The incidence of the feather abnormality was reduced by high levels of several antioxidants.

From the above discussion one can readily see that nutrient sparing effects between vitamin E and selenium, requirement stressing effects of linoleic acid, and antioxidant and sulfur amino acid effects can affect the nutritional health of the animal. One should be aware of these interrelationships when deciding on particular levels of these nutrients to include in diets.

Selenium-Arsenic Relationships

Early observations by Moxon (1938) showed that arsenic salts could be used to partially overcome harmful effects of excess selenium consumed by swine. Several workers used sodium arsenite administered through water at 2.5 or 5 ppm arsenic to overcome problems with high selenium fed to rats (Moxon and DuBois, 1939) and laying hens (Moxon and Wilson, 1944).

Various forms of arsenic have been used. Both sodium arsenate and sodium arsenite but not arsenic sulfides were effective against seleniferous wheat, sodium selenite and seleno-cystine in studies by DuBois et al. (1940). Organic arsenicals, arsanilic acid and 3-nitro-4-hydroxyphenylarsonic acid were also effective (Hendrick et al., 1953). Kamstra and Bonhorst (1953) injected selenium and arsenic into rats and observed that the antagonism was effective for selenite. Perhaps orally ingested selenate is converted to the selenite form before it becomes toxic.

The exact mechanism by which arsenic overcomes selenium toxicity has not been determined. Apparently, arsenic does not result in lower selenium absorption (Moxon et al., 1945; Peterson et al., 1950). Other workers report increased gastrointestinal excretion and higher kidney levels of selenium when arsenic is used (Kamstra and Bonhorst, 1953; Ganther and Baumann, 1962; Levander and Baumann, 1966a). In some instances where arsenic was used there was no increase in tissue deposition of selenium (Peterson et al., 1950). Arsenic actually decreased blood,

liver and carcass selenium (Ganther and Baumann, 1962; Levander and Baumann, 1966a). Palmer and Bonhorst (1957) observed higher blood selenium and lower liver selenium when 1.36 mg Se and 2.0 mg As/kg body weight were injected subcutaneously into rats. They postulated the existence of a blood barrier which reduced incorporation of selenium into liver within a given range. Within this restricted range, selenium and arsenic antagonized one another and form a volatile selenium substance provided arsenic was injected within one hour of selenium. When excess selenium and arsenic are present they may have additive toxicity. A few hours post injection, selenium and arsenic may be incorporated into protein in a relatively non-toxic form if proper levels have been used. Cummins and Martin (1967) concluded that there was no pathway for *in vivo* synthesis of seleno-cystine or selenomethinine, at least in the rabbit.

An excretory route which should be considered is bilary excretion. Levander and Baumann (1966b) observed that normally little selenium is lost via bile but a greatly increased amount is lost when arsenic is injected into rats. Perhaps the increased gastrointestinal selenium levels seen when arsenic is administered is due largely to the loss in the bile.

Olson *et al.* (1963) observed that volatilization of selenium through the lungs was not affected by arsenic when low levels of selenium were given to rats. However, when high levels (2 mg Se/kg body weight) are injected, exhalation plays a much greater role. These workers found

that both arsenite and arsenate were effective in increasing selenium exhalation when the high levels selenite or selenate were ingested. Apparently, the rat is more efficient in converting selenite to the volatile form than selenate. Several investigators have observed that inorganic forms of arsenic which alleviate selenium toxicity actually inhibit volatilization (Kamstra and Bonhorst, 1953; Ganther and Baumann, 1962). However, arsenic alleviates selenium toxicity even when selenium volatilization does not occur to any significant extent (Olson et al., 1963).

Urinary and fecal excretion of selenium are other pathways by which excess selenium may be lost. Ganther and Baumann (1962) reported that arsenic increased kidney selenium levels but did not change the amount excreted in the urine.

Levander and Baumann (1966a) observed that arsenic increased urinary selenium in only one isolated experiment and other investigators have not been able to demonstrate any change in urinary excretion due to arsenic treatment (Olson et al., 1963; Peterson et al., 1950). Apparently, the mechanism by which arsenic overcomes excessive levels of selenium is not involved with increased urinary loss.

Some reports show arsenic to increase fecal excretion of selenium. Ganther and Baumann (1962) and Levander and Baumann (1966a) observed an increase in gastrointestinal excretion which could include unabsorbed as well as absorbed-secreted selenium. Olson et al. (1963) and Peterson et al. (1950) observed no increase in fecal selenium when arsenic was given.

Arsenic has been used by several workers to overcome harmful effects of excess selenium in poultry diets. Moxon and Wilson (1944) describe work with the selenium-arsenic antagonism. Other reports of the use of arsenic in poultry diets include turkeys (Carlson, 1951), laying hens (Krista, 1961; Thapar, 1964; Carlson et al., 1969; Thapar et al., 1969) and chicks (Carlson et al., 1954 and 1962; Thapar, 1964; Thapar et al., 1969).

Arsenic fed to hens receiving high selenium caused less selenium to be deposited in eggs (Krista, 1961) and more to be deposited in chick tissue and liver (Carlson et al., 1962). In later studies, As supplements reduced egg and liver selenium (Carlson et al., 1969), and increased liver selenium in chicks. However, with older chickens arsenic supplements decreased liver and egg selenium (Thapar et al., 1969).

Carlson et al. (1962) found that when sodium selenite was included in starting diets fed to male chicks, a level of 10 ppm inhibited growth. The growth inhibition could be overcome by adding 15 ppm arsenic (as sodium arsenite). However, when selenium from seleniferous wheat was included to provide 8 ppm dietary selenium, the growth depression could not be overcome by adding sodium arsenite. Perhaps this suggests that the organic selenium of the wheat may be of more biological potency than that of sodium selenite.

Selenium as a Carcinogen

A few investigators have reported development of tumors in the liver of rats fed selenium in long term studies. Nelson et al. (1943) fed

selenium in the organic form with corn and wheat and also as inorganic solutions of ammonium potassium sulfide and ammonium potassium selenide. Eleven of 53 rats surviving the dietary treatments for 18-24 months developed hepatocellular adenomas. Five of the eleven tumors were considered to be low grade carcinomas which did not metastasize. Tscherkes et al. (1961) reported that hepatic tumors were observed in laboratory rats fed sodium selenate. Forty heterozygous rats were fed 4.3 ppm Se as sodium selenate for periods up to 32 months. Of the 23 rats that survived more than 18 months, 3 had hepatic carcinomas, 3 hepatic adenomas and four had lesions considered precancerous. Other workers (Harr et al., 1967) have been unable to produce hepatic tumors by feeding selenium to rats. The carcinogenicity of selenium is an unanswered question; however, the major portion of research in this area has not indicated selenium to be carcinogenic. Recently, there has been considerable interest in use of selenium as an anti-cancer or anti-tumor substance (Allaway, 1970).

Effect of Selenium on Egg Hatchability

Studies by Franke and Tully (1935) and Poley et al. (1937) first showed the dramatic effects of excess selenium on depressing hatchability of eggs. Poley and Moxon (1938) found that 2.5 ppm Se included in chicken breeder diets was not harmful. Levels of more than 5 ppm Se lowered hatchability and 10 ppm Se provided by seleniferous wheat gave zero hatchability. The eggs themselves contained 1-3 ppm Se. Later, Poley et al. (1941) noted improved hatchability with 2 or 4 ppm Se

provided by seleniferous wheat. Fertility of eggs was somewhat lower for the selenium treatments but this was not believed to be due to the rations fed but to different breeding males. Hatchability for the 0, 2 and 4 ppm groups was 70.7, 81.4 and 75.1 percent, respectively. Most embryonic death occurred during the third week of incubation and only one embryo from 212 eggs which failed to hatch showed evidence of a "wiry down" condition characteristic of selenium poisoning. Neck edema was common among dead embryos. Moxon and Wilson (1944) fed hens a diet containing 10 ppm Se provided by seleniferous wheat. They observed that the detrimental effect of selenium on hatchability was partially overcome by including arsenic in the drinking water at levels of 2.5 and 5 ppm. Thapar (1964) observed a significant decrease in hatchability of eggs from hens fed a corn-soy diet supplemented with 8 ppm Se as selenious acid (H_2SeO_3). Supplying added Se at 2 ppm or at 8 ppm plus 15 ppm As (as sodium arsenite) had no adverse effect on hatchability.

Carlson et al. (1969) used a glucose-isolated soybean protein diet supplemented with 0, 2, 8 ppm Se and 8 ppm Se plus 8 ppm As for a long term life cycle with laying hens. The 8 ppm Se (as sodium selenite) reduced egg production and hatchability.

Two ppm Se improved hatchability somewhat and the arsenic completely overcame adverse effects of 8 ppm on hatchability. Thapar et al. (1969) reviewed the two previously described experiments using the corn-soy and the purified type diet with the various sub-toxic and marginally toxic addition of selenium.

Effects of Dietary Selenium on Chick Performance

Poley and Moxon (1938) found that the growth and mortality of chicks hatched from hens receiving 5 ppm selenium in their ration were not affected when the chick rations contained no supplemental selenium. Poley et al. (1941) reported a growth response with male and female chicks when their diets were supplemented with 2 ppm selenium from seleniferous grain. When chicks were fed a diet without added selenium the ones hatched from dams receiving 4 ppm dietary selenium grew more slowly than those from dams receiving 0 or 2 ppm added selenium. However, when chicks were fed higher levels of selenium (5 and 8 ppm) the ones hatched from dams receiving 4 ppm dietary selenium grew just as well as those hatched from dams receiving lower levels. This suggests some mechanism by which chicks may have acquired from their dam an ability to metabolize excess selenium. These workers also reported that a level of 10 ppm selenium markedly decreased chick growth and 14 ppm was much more toxic as evidenced by a further reduction in growth and increased mortality.

In a more recent report Carlson et al. (1962) found that 10 ppm selenium (as sodium selenite) inhibited male chick growth to four weeks of age. The growth inhibition was partly overcome by adding 15 ppm arsenic (as sodium arsenite). When seleniferous wheat was used a similar growth inhibition occurred, but arsenic failed to counter-act this effect from a natural selenium source. Thapar (1964) found that when 0 or 2 ppm selenium (as selenious acid) were added to a corn-soy chick starter there was no significant difference in growth. A higher

level (8 ppm Se) significantly decreased growth, but this effect was successfully counteracted using 15 ppm arsenic (sodium arsenite). Male and female chicks responded similarily to the selenium supplements. Male chicks weighed 20-30 grams more than the females at four weeks of age. Thapar et al. (1969) found that 2 ppm selenium (as sodium selenite) supplementing a glucose-isolated soy protein diet had no adverse effect on four week old female chick growth. When 8 ppm Se or 8 ppm Se plus 8 ppm As (as sodium arsenite) were included in the diet chick growth was reduced somewhat.

Nesheim and Scott (1958) were probably the first investigators to show a growth response with chicks fed minute levels of selenium. A Torula yeast diet (0.056 ppm Se) and an isolated-soy protein diet (0.09 ppm Se) were fed to chicks. Addition of 0.04 ppm Se to the Torula yeast diet which contained 50 IU vitamin E per pound resulted in maximal growth. When vitamin E was not present 0.04 ppm Se was not adequate for growth. They concluded that 0.10 ppm selenium (as sodium selenite) was necessary for maximal growth in the presence of adequate vitamin E. Apparently, the isolated-soy diet contained sufficient selenium so that no growth effect was noted. These workers had difficulty in confirming this work in later studies due to the use of ingredients contaminated with selenium.

Probably one of the most interesting recent reports concerning the necessity of selenium for growth of chicks is that by Thompson and Scott (1969). A purified crystalline amino acid diet (no cystine) containing less than 0.005 ppm selenium was used to test various levels of added

selenium and vitamin E. When 100 ppm vitamin E was given the selenium requirement was less than 0.01 ppm. However, with 10 ppm vitamin E the selenium requirement increased to more than 0.02 ppm. When no vitamin E was included the selenium requirement for chicks increased to 0.05 ppm.

Effects of Dietary Selenium on Growing Pullets

Poley et al. (1941) found that pullets fed 0, 5 or 8 ppm selenium during the growing period of 8-24 weeks had equal growth rates. The selenium was provided in the diet by seleniferous wheat and barley. The pullets were examined at 8, 16 and 24 weeks of age for gross pathological lesions. No differences that could be attributed to level of selenium were observed. Thapar (1964) observed that pullets fed 2 ppm selenium (as selenious acid) were significantly heavier than unsupplemented groups at 8 weeks of age. Feather formation was impaired by 8 ppm selenium. Birds fed 8 ppm selenium had a yellowish tint in their feathers and a garlic-like odor typical of dimethyl selenide was present in the confinement area. Pullets fed 2 ppm selenium weighed the same as unsupplemented groups at 12 weeks. At 16, 20 and 24 weeks the 2 ppm groups weighed slightly more than those fed no additional selenium. Selenium fed at 8 ppm reduced the growth rate of pullets at all ages (8-24 weeks) but this effect was completely counteracted by the addition of 15 ppm arsenic (as sodium arsenite). Mortality was not increased in the 8 ppm Se or the 8 ppm Se plus 15 ppm As treatments. The 2 ppm Se level had 3-4 percent higher death loss than the supplemented group.

In the more recent report (Thapar et al., 1969), pullets fed a glucose-isolated soy protein diet were similar at 20 weeks with regard

to weight whether fed the 0, 2, 8 ppm Se or the 8 ppm Se plus 8 ppm As treatments. Selenium was provided by sodium selenite and arsenic by sodium arsenite in this study.

Effects of Dietary Selenium on Laying Hens

Selenium provided in a laying hen diet at a level of 15 ppm (from seleniferous grains) caused hens to lose weight and produce smaller eggs (Poley et al., 1937). The total number of eggs produced and fertility of those eggs were unaffected. Hatchability was zero after seven days on the high selenium diet. Poley and Moxon (1938) found that only 10 ppm Se was markedly toxic in laying hen diets supplemented with 0, 2.5, 5 and 10 ppm selenium (from seleniferous grain). Later Poley et al. (1941) found no differences in body weight, mortality, feed consumption or egg production of laying pullets fed selenium in a ten-week trial. Selenium levels of 0, 2 and 4 ppm were provided by normal and seleniferous wheat, corn and barley.

Recent studies with laying hens (Thapar, 1964) show that 2 ppm Se (as selenious acid) in a corn-soy diet was not harmful. Body weights at 32 weeks of age were slightly higher for those fed 2 ppm Se than for the unsupplemented groups. Little difference in body weight was found at 76 weeks of age except that hens fed 8 ppm Se weighed 100 grams less than the other treatments. Egg size was unaffected by 2 ppm Se and lowered 2 grams with 8 ppm Se. Arsenic at 15 ppm partly overcame the decrease in egg size due to the high selenium treatment. In the twelve-

month laying trial, hen-day egg production average 63.7, 63.3, 43.8 and 57.3 percent for the 0, 2, 8 and 8 ppm Se plus 15 ppm As treatments, respectively. Feed conversion favored the 2 ppm Se treatment (2.4 vs. 2.2 kg/Doz. standard 56 gram eggs). Mortality from 24-76 weeks of age was lowest for the 2 ppm Se supplemented group. (9 percent less). Seven percent more hens were lost when fed 8 ppm selenium as compared to the group fed the basal diet. Arsenic slightly lowered the death loss from 8 ppm selenium.

Thapar et al. (1969) fed a glucose-isolated soybean protein diet to hens and added 0, 2, 8 and 8 ppm Se + 8 ppm As. In this study sodium selenite and sodium arsenite were used. Body weights of the hens at 72 and 105 week of age were similar. However, there may have been a tendency for hens fed 8 ppm selenium to weigh slightly less. Mortality through 105 weeks was 81.5, 66.4, 86.0 and 66.6 for the respective treatments. Hen-day egg production through the 72 week portion of the study was lowered by the 8 ppm selenium but unaffected by the lower selenium or the arsenic-selenium treatment.

To the author's knowledge studies using lower levels of selenium and arsenic or selenium alone have not been reported. It appears that there is a need to pinpoint more clearly a level of selenium supplementation which might improve performance of laying hens.

Several workers have reported a reduced feed intake when laying hens were fed added selenium. Poley et al. (1941) found that sodium selenite added at 2 ppm to a laying ration reduced feed intake. Thapar

(1964) observed that 2 ppm added selenium (selenious acid) improved feed efficiency because less feed was needed to produce the same number of standard 56 gram eggs than was required with the unsupplemented diets.

Effect of Dietary Selenium on Tissue Deposition

Selenium is known to be deposited in tissue and eggs. The amount which is deposited can be related to the dietary intake. Carlson et al. (1962) found that selenium from high selenium wheat (20 ppm) was deposited in both liver and muscle (pectoralis major) to a greater extent than it was from sodium selenite. Arsenic supplied as arsanilic acid or sodium arsenite increased the liver selenium and increased the muscle selenium to a lesser degree in the four-week old chicks. Using a corn-soy diet it was also observed that both arsanilic acid and arsenite increased liver selenium levels. A level of 30 ppm arsenic caused a greater amount of selenium to be deposited in chick liver than did the 15 ppm.

A recent report (Scott and Cantor, 1971) demonstrated that selenium could be increased in the blood and tissues of young chicks by increasing the dietary level. To a corn-soy chick starter containing 0.07 ppm they added 0.0, 0.1, 0.2, 0.4, 0.6 and 0.8 ppm Se as sodium selenite. At four weeks of age Se levels in the blood were 0.08, 0.13, 0.19, 0.20, 0.23, 0.24; in muscle 0.06, 0.07, 0.10, 0.11, 0.13, 0.16, and kidney 0.39, 0.34, 0.80, 0.56, 0.62 and 0.71 ppm. Blood levels were slightly higher at eight weeks of age but tended to plateau with dietary intakes of 0.27 to 0.87 ppm Se.

McFarland et al. (1970) studied the distribution of selenium in adult male and female chickens. For adult laying hen tissues, the selenium concentrations were in decreasing order as follows: pineal, pituitary, kidney, spleen, egg yolk, liver, pancreas, magnum, cerebrum, diencephalon, cerebellum, blood, ovary and pectoral muscle. They concluded that selenium tends to concentrate in glandular organs and those tissues associated with detoxifying and excreting selenium. Often the organs in which selenium is highest are those associated with protein synthesis. It was noted the selenium in the blood of females was lower than that of males. Work by Thapar et al. (1969) found that hens fed corn-soy diets with selenium added at 0, 2, 8 and 8 ppm Se plus 15 ppm As, deposited 0.41, 0.86, 3.21, and 1.41 ppm Se in liver and 0.36, 0.52, 0.45, and 0.50 ppm Se in breast muscle for the respective treatments. When a glucose-isolated soy protein diet was used with 0, 2, 8 ppm Se and 8 ppm Se plus 8 ppm As, the liver values for selenium were 0.48, 1.09, 3.47 and 1.34, respectively. In their work the hens were fed selenium in a long term life cycle study.

It has also been well established that eggs are a route of excretion for excess selenium. Krista et al. (1961) found that hens fed a corn-soy diet with 10 ppm added Se deposited 10.6 ppm Se in their eggs. In their studies, the egg contents were precipitated by acetone and the resulting precipitate analysed for selenium. By including 15 ppm As the selenium was reduced to 7.9 ppm. In longer term studies, Thapar et al. (1969) found that 2 ppm added dietary selenium increased egg

selenium only slightly (0.1-0.2 ppm), that 8 ppm Se increased it further and that arsenic lowered egg selenium. Selenium is found to be concentrated in the yolk portion of the egg. Hadjimarkos and Bonhorst (1964) observed the ratio between yolk and albumen selenium to be 6.3:1. McFarland et al. (1970) found a similar ratio (5.1:1) indicating the yolk to be the richest source of selenium.

EXPERIMENTAL PROCEDURE

These studies were conducted over the two year period from May 1969 to June 1971. Basically, three experiments were conducted and will be identified as Experiment One, Two and Three.

Experiment One

Fertile eggs obtained from Regional Control Single Comb White Leghorn (SCWL) stock maintained at the Poultry Research Center, South Dakota State University (PRC-SDSU) were incubated in Jamesway incubators. On May 9, 1969 the chicks were hatched and 38 mixed sex chicks were placed randomly into each of 39 pens in electrically heated, stainless steel battery brooders. All individual chicks of each group were identified by wing bands. The experiment was a completely random design. Each of thirteen dietary treatments was replicated three times making up the total of 39 groups of chicks.

Criteria studied in Experiment One include body weight changes, mortality, feed consumption, egg production, egg quality, egg shell thickness, egg fertility, egg hatchability, progeny performance, and selenium content of eggs, feathers, liver, thigh muscle, breast muscle, kidney and heart. Male chicks were killed at four weeks of age. Kidney and liver samples from five male chicks from each of the 39 groups were obtained for the selenium analysis. Female chicks were maintained in the battery brooders until eight weeks of age when they were placed at

various densities of 5-8 pullets into 40.6 cm by 45.7 cm layer cages in a windowless, environmentally controlled cage house located at the PRC-SDSU. They were maintained in these same cages throughout Experiment One during which time eggs and tissues were obtained for Se analysis. Three cages of pullets were considered as one replicate group. Analysis of variance and F tests were computed on the data obtained. Treatment means were separated using Duncan's New Multiple Range Test (Steel and Torrie, 1960).

Feed and water were supplied ad libitum to the chicks in stainless steel pans and to the caged birds with Hart water cups and galvanized feeding troughs. Three types of diets were fed in Experiment One from day-old to 64 weeks of age. Ethoxyquin was added to all diets at 110 mg per kg of diet and dl-alpha-tocopheryl acetate at 10 mg per kg of diet in this experiment.

Composition of the first type of diet is shown on Table 1. Note that during the growing period (12-24 weeks) a high fiber oats diet was used. Selenium was supplemented to these practical type diets at 0, 2, and 8 parts per million (ppm) selenium (Se) and 8 ppm Se plus 15 ppm arsenic (As). Selenium was supplied in the form of sodium selenite and arsenic in the form of sodium arsenite. The 2 ppm Se is a sub-toxic level and 8 ppm Se a marginally toxic level. Arsenic at 15 ppm was previously shown to be a desirable level to counteract the toxic effects of selenium in practical type diets (Thapar et al., 1969).

The second diet used in Experiment One was a purified type diet based on glucose and isolated soybean protein. Composition of this diet

Table 1. Practical Diets Used in Experiments One, Two and Three.

Ingredient	Percent of Diet		
	Starter	Grower	Layer
Yellow corn, grd.	61.45	-----	67.26 (67.44)[4]
Oats, grd.	-----	80.76	-----
Soybean meal (50)	27.50	-----	20.0
Soybean meal (44)	-----	2.0	-----
Wheat middlings	-----	5.0	-----
Meat scraps (50)	2.0	2.0	-----
Alfalfa meal (17)	2.0	2.0	2.0
Fish meal (60)	2.0	1.0	-----
Dried whey	2.0	2.0	-----
Yellow grease	-----	-----	3.0
Dicalcium phosphate	2.0	3.0	2.0
Limestone	0.25	1.5	5.0
MHA	0.05	-----	-----
Minerals[1]	0.51	0.5	0.5
Vitamins[2,3]	0.24	0.24	0.24 (0.06)[4]

[1]Supplies per kilogram of diet: NaCl, 4.75 gm; Ca, 37.5 mg; P, 12.5 mg; Mn, 12.5 mg; Cu, 1.65 mg; Zn, 15 mg; Fe, 12.5 mg; Co, 0.5 mg; I, 0.35 mg; and S, 15 mg. Starter diet also contained additional Mn, 30 mg and Zn, 15 mg supplied in the sulfate monohydrate forms.

[2]Supplies per kilogram of diet: Vitamin A palmitate, 2500 IU; vitamin D, 1000 ICU; dl-alpha-tocopheryl acetate, 10 IU; menadione, 0.5 mg; riboflavin, 2 mg; pantothenic acid, 4 mg; niacin, 20 mg; choline, 200 mg; vitamin B_{12}, 4 mcg; folic acid, 0.5 mg; biotin, 50 mcg; ethoxyquin, 110 mg; and oxytetracycline, 22 mg.

[3]Experiment Three layer diet vitamins supply per kilogram of diet: Vitamin A palmitate, 4000 IU; vitamin D_3, 1000 ICU; menadione sodium bisulfite, 1 mg; riboflavin, 2 mg; pantothenic acid, 4 mg; niacin, 20 mg; choline chloride, 500 mg; vitamin B_{12}, 8 mcg; folic acid, 0.5 mg; biotin, 50 mcg; and ethoxyquin, 50 mg.

[4]Experiment Three.

Table 2. Glucose-Isolated Soybean Protein Diets Used in Experiment One.

Ingredients	Percent of Diet		
	Starter	Grower	Layer
Isolated soy protein[1]	24.0	15.55	17.77
Glucose monohydrate[2]	64.17	73.0	67.28
Cellulose[3]	5.0	5.0	5.0
Corn oil	2.0	2.0	2.0
Dicalcium phosphate	2.0	2.0	1.0
Limestone	1.0	1.0	5.8
MHA	0.27	0.3	0.2
Glycine	0.2	0.2	-----
Minerals[4]	1.13	0.73	0.73
Vitamins[5]	0.23	0.22	0.22

[1]Purina Assay Protein RP-100, Ralston Purina Company, St. Louis, Mo. 63199.

[2]Cerelose, Corn Products Company, Argo, Ill. 60501.

[3]Solka floc, BW-20, Brown Company, Berlin, N. H. 03570.

[4]Supplies per kilogram of diet: NaCl, 0.5% (0.35%); KCl, 0.3% (0.15%); $MgSO_4$, 0.25% (0.15%); $MnSO_4 \cdot H_2O$, 261 mg; $ZnSO_4 \cdot H_2O$, 220 mg; $FeSO_4$, 163 mg; $CuSO_4 \cdot 5H_2O$, 39.5 mg; $CoCl_2 \cdot 6H_2O$, 12 mg; KI, 11 mg; $Na_2MoO_4 \cdot 2H_2O$, 11 mg; H_3BO_3, 11 mg; $KAl(SO_4)_2 \cdot 12H_2O$, 11 mg; Na_2SiO_3, 44 mg; and NaBr, 2 mg. Values in parenthesis indicate reduced levels used in the grower and layer diets. $MgCO_3$ was substituted for $MgSO_4$ in the grower and layer rations.

[5]Supplies per kilogram of diet: ( ) indicates reduced levels used in the grower and layer. Vitamin A, 6000 IU; vitamin D_3, 1000 ICU; dl-alpha-tocopheryl acetate, 10 mg; menadione sodium bisulfite, 11 mg; riboflavin, 22(11) mg; folic acid, 4(3) mg; pyridoxine·HCl, 22(20) mg; thiamine·HCl, 22(20) mg; calcium pantothenate, 44(30) mg; niacin, 88(55) mg; choline chloride, 1295 mg; ascorbic acid, 22 mg; vitamin B_{12}, 30(20) mcg; biotin 440(220) mcg; ethoxyquin, 110 mg; and oxytetracycline, 22 mg.

Table 3. Glucose-Torula Yeast Diets Used in Experiment One.

Ingredient	Percent of Diet		
	Starter	Grower	Layer
Torula yeast[1]	42.4	27.45	31.4
Glucose monohydrate[2]	45.94	61.1	53.7
Cellulose[3]	5.0	5.0	5.0
Corn oil	2.0	2.0	2.0
Dicalcium phosphate	2.0	2.0	1.0
Limestone	1.0	1.0	5.8
MHA	0.1	0.3	0.2
Glycine	0.2	0.2	----
Minerals[4]	1.13	0.73	0.68
Vitamins[5]	0.23	0.22	0.22

[1]Lake States Division, St. Regis Paper Company, Rhinelander, Wisc.

[2]Cerelose, Corn Products Company, Argo, Ill. 60501.

[3]Solka floc, BW-20, Brown Company, Berlin, N. H. 03570.

[4]Similar to that shown in Table 2 except here $MnSO_4 \cdot H_2O$, 230 mg; $ZnSO_4 \cdot H_2O$, 96 mg; and $FeSO_4$, 41 mg per kilogram of layer diet was used.

[5]Similar to that shown in Table 2.

is shown in Table 2. To this diet 0, 2, and 8 ppm Se and 8 ppm Se plus 8 ppm As were added. Previous studies at this laboratory had shown 15 ppm arsenic to be rather toxic in a purified diet and that a level of 8 ppm was much more desirable to counteract toxic effects of selenium.

The third type of diet used in Experiment One was a purified type composed of glucose and Torula Yeast. Composition of this diet is shown

in Table 3. Selenium was supplemented to this diet at 0, 0.2, 2, and 8 ppm and 8 ppm Se plus 8 ppm As. Torula yeast was used because of its relatively low selenium content and ability to produce selenium deficiency signs (Schwarz and Foltz, 1957). The additional level of selenium supplementation (0.2 ppm) was selected because this is twice the suggested requirement for chicks (Thompson and Scott, 1969), and yet much lower than the 2 ppm level used in this and the other previous studies.

Mortality, feed consumption and body weight records were summarized at 28-day intervals throughout the entire experimental period (0-64 weeks of age). The experiment was divided into a starting period (0-12 weeks), growing period (12-24 weeks) and laying period (24-64 weeks). At 24 weeks the surviving pullets were redistributed among replicates of each given treatment so as to equalize bird density. Five pullets were allowed per 40.6 cm by 45.7 cm cage with a lath perch installed approximately 15 cm above the cage bottom and 15 cm from the rear of each cage.

During the egg producing periods additional information on hen-day egg production (number of hens x number of days divided by number of eggs), egg weight, shell thickness, interior egg quality, egg fertility, egg hatchability, progeny performance, feed efficiency (kg feed per doz.) and selenium content of eggs was obtained. The above parameters were summarized at 28-day intervals except for egg fertility, egg hatchability and selenium analysis of eggs and tissues.

The egg weight average of each replicate group for each 28-day period was obtained by weighing ten eggs per week for each of four weeks

and dividing total egg weight by total number of eggs (40). Shell thickness and egg quality determination were made from ten eggs per replicate group per 28-day period. Interior egg quality was determined according to the method of Haugh (1937). Shell thickness was measured in millimeters using a micrometer. Artificial insemination was used to obtain fertility, hatchability and progeny information. Males of the same strain were used. Progeny were grown to four weeks of age on a diet similar to their dams or fed a special chick starter (Table 4).

Whole egg samples were collected from each of the 39 replicate groups at 32, 42, 52, and 62 weeks of age which would correspond to 10, 20, 30 and 40 weeks into egg production. In all cases the whole egg samples were prepared by homogenizing five eggs per sample (yolk and albumen). All selenium analyses were conducted in the laboratory of Dr. O. E. Olson (Station Biochemistry, South Dakota State University) using the Watkinson fluorometric technique as modified by Olson (1969).

Experiment One continued for 64 weeks. At the end of 64 weeks hens fed the purified diets were sacrificed; and feathers, liver, kidney, thigh, breast and heart samples were taken from five hens per replicate group. Each type of tissue from the five hens per group were combined in order to make one selenium determination for each replicate group. (i.e. 5 livers = one sample). Because of a recent report concerning the ability of selenium to decrease liver fat (Jensen *et al.*, 1970) the percentage fat was determined in the liver samples according to the method for meats described in Official Methods of Analysis of the

Table 4. Glucose-Torula Yeast-Isolated Soybean Protein Diets.

Ingredients	Percent of Diet	
	Starter	Layer
Torula yeast[1]	22.5	17.5
Isolated soy protein[2]	12.5	9.75
Glucose monohydrate[3]	53.24	55.31
Cellulose[4]	5.0	5.0
Corn oil	2.0	4.0
Dicalcium phosphate	2.0	2.0
Limestone	1.0	5.5
MHA	0.2	0.2
Glycine	0.2	----
Minerals	1.13[5]	0.58[7]
Vitamins	0.23[6]	0.16[8]

[1]Lake States Division, St. Regis Paper Co., Rhinelander, Wisc. 54501.

[2]Purina Assay Protein RP-100, Ralston Purina Co., St. Louis, Mo. 63199.

[3]Cerelose, Corn Products Company, Argo, Ill. 60501.

[4]Solka floc, BW-20, Brown Company, Berlin, N. H. 03570.

[5]Similar to that shown in Table 2.

[6]Similar to that shown in Table 2 except dl-alpha-tocopheryl was reduced to 5 mg. per kilogram of diet.

[7]Similar to that shown in Table 2 for the layer diet except here these changes were made; NaCl, 3 mg; $MgCO_3$, 1 mg; $MnSO_4 \cdot H_2O$, 77 mg; $ZnSO_4 \cdot H_2O$, 69 mg; and $FeSO_4$, 15.8 mg per kilogram of diet.

[8]Similar to the values listed in Table 2 for the layer diet but with the following changes: 2 mg per kilogram of diet dl-alpha-tocopheryl acetate, 50 mg per kilogram of diet ethoxyquin and no oxytetracycline.

Association Official Analytical Chemists (1970). Feather samples were obtained from a region anterior to the thigh. After the hens were killed by dislocating their cervical vertebrae, sampling of feathers was conveniently done by dry picking. To obtain the other tissues each hen was partly skinned and the body cavity exposed so that the heart, one kidney and 3-5 grams of a liver lobe could be removed. Hens fed the corn-soy diet were not sacrified at 64 weeks of age but used in Experiment Two.

Experiment Two

Hens which survived Experiment One and had been fed the corn-soy diet (Table 1) supplemented with 0, 2, and 8 ppm Se and 8 ppm Se plus 8 ppm As were used in Experiment Two. A randomized complete block design was used with two blocks in smaller cages and one block in larger ones. F tests and analysis of variance were calculated on data obtained.

The hens that at the time were 64 weeks old were maintained on the same treatments for an additional four weeks and then force molted. The force molt was accomplished by removing the hens from the cages and placing them on slat floors in another poultry house. Water was not allowed for 48 hours and feed not given for 96 hours. These changes caused an immediate cessation of egg production and initiated a molt. When the hens were re-fed they were given the same diets as they had been fed previously. After the hens molted they were replaced in laying cages with three replicates for each of four treatments. Two sizes of cages were used because of physical limitations. One replicate (block) of all

four treatments was placed in 40.6 x 45.7 cm cages. Two replicates of all treatments were placed in 30.5 x 45.7 cm cages. Four hens per cage were used in the larger size cages and three per cage in the smaller type. No perches were provided inside the cages in this experiment.

Feed and water were provided as described for Experiment One. Only the corn-soy diet was used in this experiment and similar criteria were studied as in Experiment One. Egg fertility and hatchability were observed only once at 102 weeks of age. Artificial insemination was used to obtain fertile eggs. No progeny studies were made in this experiment. Egg samples were taken for Se analysis at 96 and 104 weeks. At 104 weeks of age 4 hens per replicate group were sacrificed with an electric stunning knife and their tissues removed as described in Experiment One. The remaining hens were fed the corn-soy basal diet without any added selenium for a depletion study. Egg samples were taken for selenium analysis at two, four, six and eight weeks after the diets were changed. After four weeks (108 weeks of age) 3 hens per replicate group were sacrificed for selenium tissue analysis. The remaining hens were maintained for an additional four weeks (to 112 weeks of age) and then they were sacrificed and the tissues were sampled for selenium content.

Experiment Three

This study was initiated on October 2, 1970. Twenty-week old pullets from Regional Control SCWL stock obtained from the PRC-SDSU and DeKalb 131 stock grown at PRC-SDSU but originally obtained from Sunshine State

Hatchery, Brookings, South Dakota were used. Three hundred thirty six pullets of each strain were selected randomly and placed into 192 cages of two sizes. The experiment was arranged in a factorial design having strain, cage size, selenium supplementation, vitamin E supplementation, diet type, replicates and periods as factors. Analysis of variance and F tests were computed in the normal manner for a factorial design. When missing values occurred, a least squares analysis was used (Steel and Torrie, 1960).

Cage sizes used were the 30.5 x 45.7 cm and 40.6 x 45.7 cm cages. Selenium was supplemented at three levels (0, 0.1, 1.0 ppm) to a corn-soy diet (Table 1) and to a glucose-isolated soy-Torula yeast (Table 4) diet. Ethoxyquin was added at 50 mg/kg of diet. Both a high and low level of vitamin E were used. The low level in the corn-soy diet was that provided only by the diet itself and the high level being 10 ppm added dl-alpha-tocopheryl acetate. The low level in the glucose-isolated soy-Torula yeast diet was 2 ppm of added dl-alpha-tocopheryl acetate and the high level being 10 ppm of the same form of vitamin. It was believed that the purified diet might be so low in natural tocopherols that good performance might not be obtained without supplementing vitamin E. Four replicates were used with each cage of pullets (three individual pullets in the small cages and four in the large cages) constituting a replicate. Therefore, 56 birds were used for each of twelve diet treatments. Data calculation and replicate averages were made as the combined total or average of the group of pullets in that particular cage. Each cage was considered an experimental unit. Eight 28-day periods were included in

Experiment Three. Due to very low egg production during the 20-24 week period, the values obtained for egg production were not recorded. Production data were obtained for the seven subsequent 28-day periods to 52 weeks of age. Thus these pullets received the 12 experimental diets for 32 weeks.

Hen-day egg production, egg yield (grams of egg per hen per day), average egg size, interior egg quality, average body weight, feed consumption, mortality, egg fertility, and egg hatchability, progeny performance, and selenium contents of tissues and eggs were observed. All criteria were summarized at 28-day intervals except selenium content data and reproduction studies. The egg production was expressed on the hen-day basis (as described in Experiment One) and as egg yield. The basis for egg yield was determined from average egg weight, which was calculated by weighing all eggs produced on fourteen days of the 28-day period and dividing by the total number weighed. Egg yield was then calculated by average egg weight multiplied by total number of eggs divided by number of hen-days. Interior egg quality was determined by the method of Haugh (1937). Average body weight determinations were made by weighing all hens of the cage (experimental unit) and dividing by the number of hens weighed. Mortality on the hen-housed basis is calculated by expressing number of hens present at the end of a 28-day period divided by the number initially at the start of the experiment X 100. Feed consumption was recorded as grams per hen per day. This was determined by observing the total amount of feed consumed of each of the

twelve diets divided by number of hen-days from groups fed that particular diet. This method did not allow one to observe strain or cage effects on feed consumption.

Various selected tissues and eggs were sampled for subsequent selenium analysis at several times during the experiment. Eggs were obtained for selenium analysis at 32 weeks of age (12 weeks after pullets were first fed the additional selenium), again at 52 weeks of age (32 weeks on selenium supplementation) and after a two-week and four-week withdrawal period (at 54 and 56 weeks of age). During the withdrawal period which began at 52 weeks of age, hens were fed the basal diets (corn-soy or purified type) without selenium added. Tissues were sampled for selenium analysis at 52 weeks and again after a one-month withdrawal period. This was accomplished by sampling tissues and eggs from hens in the smaller cages at the 52 week sampling and those from the larger cages at the 56 week sampling. The manner of sample collection, preparation and analysis of selenium was the same as described for Experiment One. Tissues studied were feathers, heart, kidney, breast muscle, thigh muscle and liver. Each type of tissue from three hens was combined to form a single sample for the analysis.

At several intervals hens were bred by artificial insemination to obtain fertility, hatchability, and progeny data. The methods used to calculate fertility and hatchability were the same as described previously (Experiment One). Progeny were fed the diet shown in Table 4.

RESULTS AND DISCUSSION

Experiment One

Body weights of the chickens in Experiment One are listed in Table 5 by four-week intervals. Each value represents the average of three replicate groups of birds. At four weeks, the males were removed from the experiment and their weights are given separately from the females. Chicks fed the practical type corn-soy diet weighed the most during the growing stages and remained the heaviest throughout the laying periods. In general, 2 ppm of selenium had no significant adverse effect on body weights and in some cases it appeared to improve early chick growth. During the laying periods the 2 ppm selenium prevented hens from becoming as heavy as those fed basal diets. Eight ppm selenium depressed body weight slightly but this effect was partially overcome in the corn-soy diet by including arsenic. There would be no advantage to feed more than 2 ppm Se as evidenced by the body weight values shown in Table 5. Since 2 ppm is 20 times the amount shown by Thompson and Scott (1969) to be the requirement, this would surely be the maximum amount that anyone should normally add. By 20 weeks there was little difference in body weights within any given type of diet; although, pullets fed the purified diets weighed somewhat less than those fed the corn-soy diets.

Table 6 shows the kilograms of feed required to produce a dozen eggs during each of the ten laying periods. According to the F tests

Table 5. Body Weights of Chickens by Four Week Intervals (Experiment One).

Age (wks.)	Treatment (ppm Se)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15[1]	0	2	8	8-8[2]	0	0.2	2	8	8-8[2]
4[3]	260	278	266	259	200	210	196	199	203	195	194	188	191
4[4]	246	251	229	236	185	196	175	184	184	181	178	170	162
8[5]	588	609	564	556	503	504	462	489	410	421	427	384	360
12	985	991	965	966	882	879	878	882	894	868	868	815	799
16[6]	1.20	1.24	1.26	1.24	1.16	1.17	1.18	1.15	1.17	1.16	1.12	1.08	1.12
20	1.48	1.44	1.48	1.44	1.41	1.42	1.44	1.44	1.39	1.39	1.39	1.34	1.34
24	1.74	1.75	1.68	1.65	1.61	1.57	1.62	1.56	1.58	1.55	1.56	1.53	1.50
28	1.94	1.84	1.85	1.85	1.73	1.71	1.65	1.64	1.67	1.65	1.65	1.64	1.58
32	2.00	1.92	1.88	1.93	1.72	1.70	1.70	1.67	1.71	1.69	1.69	1.62	1.63
36	2.03	1.96	1.85	1.96	1.77	1.72	1.71	1.67	1.78	1.77	1.72	1.50	1.63
40	2.10	2.01	1.86	1.97	1.71	1.74	1.73	1.66	1.79	1.76	1.64	1.64	1.60
44	2.15	2.05	1.92	2.00	1.78	1.78	1.71	1.67	1.84	1.84	1.77	1.72	1.73
48	2.14	1.99	1.90	2.00	1.79	1.80	1.73	1.66	1.87	1.90	1.75	1.73	1.77
52	2.18	2.03	1.86	2.03	1.83	1.83	1.74	1.68	1.98	1.92	1.94	1.82	1.82
56	2.16	2.02	1.93	1.92	1.73	1.87	1.74	1.71	1.96	1.98	1.94	1.78	1.72
60	2.10	1.99	1.95	2.02	1.77	1.87	1.72	1.68	1.89	1.90	1.91	1.78	1.79
64	2.10	2.02	1.91	2.02	1.68	1.80	1.63	1.68	1.85	1.80	1.81	1.74	1.73

[1]Eight ppm selenium and 15 ppm arsenic.

[2]Eight ppm selenium and 8 ppm arsenic.

[3]Body weights of male chicks at four weeks of age (gm).

[4]Body weights of female chicks at four weeks of age (gm).

[5]Eight and twelve week body weights for females in grams.

[6]Sixteen through sixty-four week body weights in kilograms.

Table 6. Feed Conversion (kg/Doz.) During the Laying Period (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	3.44	2.72	3.77	4.21	6.80	5.60	6.55	7.62	7.78	9.17	7.18	9.48	10.98
28-32	2.10	1.81	2.17	2.06	2.96	3.48	3.39	3.11	3.40	3.64	3.69	2.81	3.91
32-36	2.45	1.79	2.23	2.12	3.16	3.66	4.19	3.85	5.80	6.70	5.43	5.84	4.06
36-40	2.71	2.15	2.76	2.34	2.67	2.04	2.87	2.81	6.96	9.32	5.88	9.03	8.60
40-44	2.38	1.85	1.89	2.08	4.02	3.82	4.37	4.66	8.29	8.29	9.87	10.34	14.10
44-48	2.37	2.43	2.94	2.21	4.14	5.00	5.04	4.15	6.45	9.77	6.89	11.08	8.83
48-52	2.69	2.05	2.71	2.41	4.49	5.25	4.72	5.41	7.07	7.39	7.48	7.98	8.09
52-56	2.53	2.28	3.04	2.03	4.42	4.06	4.08	5.09	5.91	6.06	4.79	6.72	6.91
56-60	3.12	2.40	3.24	2.89	5.49	4.31	5.48	5.81	5.87	5.24	5.21	7.15	7.67
60-64	3.05	2.29	3.16	2.96	6.39	4.38	6.57	5.99	7.56	6.94	7.86	9.95	8.34
Aver.	2.68A[1]	2.18A	2.79A	2.53A	4.45B	4.16B	4.73B	4.85B	6.51C	7.25CD	6.43C	8.04D	8.15D
Duncan 5[2]		1.07	1.13	1.17	1.19	1.22	1.23	1.25	1.26	1.28	1.29	1.29	1.29
Dunc										1.66	1.67	1.67	1.67

[1] Mean … rent.

[2] Indi… ignificantly ($P < 0.05$) different by D…

[3] Indi… ignificantly ($P < 0.01$) different by D…

Table 6. Feed Conversion (kg/Doz.) During the Laying Period (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	3.44	2.72	3.77	4.21	6.80	5.60	6.55	7.62	7.78	9.17	7.18	9.48	10.98
28-32	2.10	1.81	2.17	2.06	2.96	3.48	3.39	3.11	3.40	3.64	3.69	2.81	3.91
32-36	2.45	1.79	2.23	2.12	3.16	3.66	4.19	3.85	5.80	6.70	5.43	5.84	4.06
36-40	2.71	2.15	2.76	2.34	2.67	2.04	2.87	2.81	6.96	9.32	5.88	9.03	8.60
40-44	2.38	1.85	1.89	2.08	4.02	3.82	4.37	4.66	8.29	8.29	9.87	10.34	14.10
44-48	2.37	2.43	2.94	2.21	4.14	5.00	5.04	4.15	6.45	9.77	6.89	11.08	8.83
48-52	2.69	2.05	2.71	2.41	4.49	5.25	4.72	5.41	7.07	7.39	7.48	7.98	8.09
52-56	2.53	2.28	3.04	2.03	4.42	4.06	4.08	5.09	5.91	6.06	4.79	6.72	6.91
56-60	3.12	2.40	3.24	2.89	5.49	4.31	5.48	5.81	5.87	5.24	5.21	7.15	7.67
60-64	3.05	2.29	3.16	2.96	6.39	4.38	6.57	5.99	7.56	6.94	7.86	9.95	8.34
Aver.	2.68A[1]	2.18A	2.79A	2.53A	4.45B	4.16B	4.73B	4.85B	6.51C	7.25CD	6.43C	8.04D	8.15D
Duncan 5[2]		1.07	1.13	1.17	1.19	1.22	1.23	1.25	1.26	1.28	1.29	1.29	1.29
Duncan 1[3]		1.42	1.48	1.52	1.55	1.57	1.60	1.61	1.63	1.66	1.67	1.67	1.67

[1]Means followed by unlike letters are significantly ($P < 0.05$) different.

[2]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.05$) different by Duncan's test.

[3]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.01$) different by Duncan's test.

both periods and treatments were significantly different. When the overall average is observed it appears that there were differences among diets but not within any given diet. There did appear to be a slight (not significant) improvement in feed conversion by feeding 2 ppm Se as shown in the data for the corn-soy and glucose-isolated soy diets.

Table 7 shows the hen-day egg production for ten periods. Note that a significant ($P < 0.01$) response was obtained by feeding 2 ppm Se to the corn-soy diet. This difference was quite consistent throughout all periods. Eight ppm Se lowered egg production slightly and arsenic completely overcame the adverse effects of this higher level of selenium.

Hens fed 2 ppm added Se to the glucose-isolated soy diet had slightly higher (not significant) egg production than the unsupplemented groups. Arsenic failed to overcome toxic effects of the 8 ppm Se in the purified diets. The addition of 0.2 ppm Se to the Torula yeast diet did not improve egg production, however, performance was quite poor with both of the purified-type diets.

Average egg weights are listed in Table 8. The hens fed the control corn-soy diet produced the largest eggs. The unsupplemented and 8 ppm Se plus 15 ppm As diets fed to hens allowed them to produce the 56 gm Large size eggs sooner (44-48 weeks) than did hens which received either 2 or 8 ppm Se. Although a 56 gram egg average was produced in later periods by hens receiving the corn-soy diet with higher selenium

Table 7. Percent Hen-Day Egg Production by Four-Week Intervals for 40 Weeks (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	39.45	47.46	35.09	32.59	13.15	16.00	12.67	10.95	16.59	11.50	14.76	11.19	8.96
28-32	75.37	77.46	67.41	67.24	37.85	35.05	30.89	32.83	26.33	24.16	32.70	27.82	31.96
32-36	67.05	76.91	62.90	70.21	39.04	32.25	27.55	31.24	27.65	20.61	25.19	23.47	21.32
36-40	64.00	73.65	55.76	68.26	31.15	36.86	23.94	26.42	24.58	17.18	25.45	19.59	16.56
40-44	64.23	73.49	58.95	65.58	31.89	37.09	29.28	28.41	19.93	14.75	11.56	12.89	7.67
44-48	64.69	70.10	61.49	62.13	29.39	30.01	26.36	24.56	24.16	16.19	23.15	15.28	17.89
48-52	60.57	68.93	59.12	64.89	30.90	26.43	28.53	23.88	25.60	21.71	21.14	19.87	19.87
52-56	64.09	69.57	61.34	65.17	32.59	37.86	33.34	24.07	29.69	26.26	34.28	22.55	22.61
56-60	57.66	68.77	60.96	60.22	26.88	35.71	25.65	23.14	29.97	30.26	30.55	21.35	21.17
60-64	55.07	69.35	57.29	54.71	19.32	32.22	17.34	19.76	23.51	21.74	17.68	14.79	19.23
Aver.	61.22E[1]	69.57F	58.03E	61.10E	29.21CD	31.95D	25.56BC	24.53BC	24.80BC	20.44AB	23.65AB	18.88A	18.72A
Duncan 5[2]		3.80	4.00	4.14	4.23	4.31	4.37	4.42	4.46	4.50	4.53	4.56	4.58
Duncan 1[3]		5.03	5.23	5.40	5.51	5.57	5.66	5.71	5.76	5.82	5.86	5.90	5.93

[1]Means followed by unlike letters are significantly ($P < 0.05$) different.

[2]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.05$) different by Duncan's test.

[3]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.01$) different by Duncan's test.

Table 8. Egg Weight (Grams) by Four-Week Intervals for 40 Weeks (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	49.13	47.80	46.60	47.50	41.97	42.60	41.44	42.47	47.61	45.82	43.80	42.65	43.20
28-32	51.23	50.07	49.93	49.47	47.23	46.20	43.83	44.27	47.65	46.43	45.80	44.90	45.03
32-36	53.73	52.83	52.27	53.23	49.30	49.50	48.53	48.10	48.82	49.07	49.33	47.67	47.89
36-40	54.60	53.90	52.33	54.17	50.56	50.80	46.97	48.16	49.67	49.28	48.30	46.31	47.49
40-44	54.83	55.93	54.10	55.33	50.83	51.77	49.14	52.65	49.15	51.12	48.21	48.77	47.42
44-48	57.13	55.00	54.30	56.13	51.75	53.67	50.32	51.90	50.13	50.10	50.27	49.97	50.33
48-52	57.97	57.07	55.98	57.57	52.26	53.00	52.38	51.80	53.01	52.61	51.00	50.70	51.11
52-56	58.87	56.43	56.37	58.80	53.63	54.47	52.94	53.38	53.27	51.69	50.77	51.20	49.70
56-60	59.43	56.07	56.77	58.07	52.74	53.83	52.87	51.94	52.59	53.90	50.89	51.65	49.59
60-64	58.67	56.17	57.23	58.97	51.71	53.64	51.84	50.22	51.83	54.22	51.56	49.33	49.98
Aver.	55.56G[1]	54.13EF	53.59E	54.92FG	50.20BCD	50.95D	49.03AB	49.49ABC	50.37BCD	50.42CD	48.99AB	48.32A	48.18A
Duncan 5[2]		0.90	0.95	0.98	1.00	1.02	1.03	1.05	1.06	1.06	1.07	1.08	1.08
Duncan 1[3]		1.19	1.24	1.28	1.30	1.32	1.34	1.35	1.36	1.38	1.39	1.40	1.40

[1]Means followed by unlike letters are significantly ($P < 0.05$) different.

[2]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.05$) different by Duncan's test.

[3]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.01$) different by Duncan's test.

levels, there were many smaller eggs produced which would not be Large Grade A eggs. This would be undesirable from a commercial point of view. Note that egg size decreased as selenium levels increased. A significant ($P < 0.01$) reduction occurred with 8 ppm Se in all three diets. Two ppm added Se reduced egg size significantly ($P < 0.01$) in the corn-soy. A level as high as 2 ppm selenium is probably to high to allow for optimum egg size.

Table 9 lists interior egg quality measured as Haugh Units. There was a general decline in interior egg quality as the hens grew older. Perhaps this is involved with a larger egg size and a tendency for more thin albumen production. Hens fed practical diets also tended to have lower Haugh Unit values than those fed the purified type diets. Again this difference is probably due to egg size differences and also to the lower rate of production with purified diets. No explanation can be given for the significantly ($P < 0.01$) improved interior quality of eggs from hens fed the 8 ppm Se and 15 ppm As. The higher average values listed for the remaining treatments are probably due to smaller egg size.

Table 10 shows egg shell thickness values in millimeters X 10^2. There appears to be no adverse effects of selenium or arsenic addition on shell thickness. There was a slightly thicker (not significant) average shell thickness produced when 2 ppm Se was added in each of the two purified diets.

The glucose-isolated soy fed hens produced eggs that had significantly ($P < 0.01$) thinner shells. Perhaps this is characteristic of a

Table 9. Interior Egg Quality (Haugh Units) by Four-Week Intervals for 40 Weeks (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	82.05	80.83	81.37	84.05	85.45	87.57	85.11	86.23	85.83	82.81	83.35	83.19	86.80
28-32	83.77	80.03	80.03	83.10	83.63	83.40	85.13	87.10	86.50	85.63	83.77	85.20	85.70
32-36	77.17	77.13	77.63	81.23	82.60	81.70	84.73	85.70	84.57	87.80	83.13	83.20	83.19
36-40	67.17	68.37	65.94	73.40	75.38	75.07	78.14	80.53	76.97	77.70	78.37	77.96	78.68
40-44	73.57	72.30	71.93	77.60	78.10	79.93	82.24	79.63	80.97	77.02	81.45	77.94	80.10
44-48	69.47	70.23	69.23	75.10	78.06	76.73	83.04	84.25	83.36	81.45	82.33	83.12	85.57
48-52	64.07	70.07	65.75	75.40	75.40	76.40	80.44	79.38	80.57	80.04	80.10	77.63	81.75
52-56	63.00	66.70	65.17	69.70	70.83	71.83	78.77	74.89	76.27	76.29	76.77	75.90	76.07
56-60	71.37	75.43	71.90	77.03	77.60	80.67	80.92	84.43	82.71	81.73	82.36	82.82	83.06
60-64	68.67	69.53	67.70	73.73	76.63	79.91	83.29	81.44	81.82	81.68	81.70	83.35	83.42
Aver.	72.03A[1]	73.06A	71.67A	77.04B	78.37B	79.32BC	82.18D	82.36D	81.96CD	81.22CD	81.33CD	81.03CD	82.43D
Duncan 5[2]		1.84	1.94	2.00	2.05	2.09	2.11	2.14	2.16	2.18	2.19	2.20	2.22
Duncan 1[3]		2.43	2.53	2.61	2.66	2.70	2.74	2.76	2.79	2.82	2.83	2.85	2.87

[1]Means followed by unlike letters are significantly ($P < 0.05$) different.

[2]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.05$) different by Duncan's test.

[3]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.01$) different by Duncan's test.

Table 10. Egg Shell Thickness (10^{2} mm) by Four-Week Intervals for 40 Weeks (Experiment One).

Period (wks.)	Treatments (ppm Se and As)												
	Corn-Soy				Glucose-Isolated Soy				Glucose-Torula Yeast				
	0	2	8	8-15	0	2	8	8-8	0	0.2	2	8	8-8
24-28	39.53	38.13	38.73	38.90	32.10	31.70	32.10	32.17	38.37	38.07	37.03	37.33	37.20
28-32	36.80	37.80	38.07	38.03	33.17	34.60	33.60	33.17	37.00	36.03	36.23	38.03	37.23
32-36	37.47	37.47	38.43	38.80	34.07	34.10	33.37	33.87	40.30	39.40	38.90	36.80	37.20
36-40	38.07	35.03	36.37	36.73	32.30	33.27	33.37	30.73	36.77	37.70	40.03	38.43	36.87
40-44	38.40	38.67	39.03	37.87	33.83	37.47	34.20	36.70	39.67	38.67	42.27	39.90	40.60
44-48	38.47	37.73	38.13	38.57	32.30	34.87	35.27	36.17	40.97	39.10	40.90	39.97	39.70
48-52	37.97	36.00	36.17	37.37	30.93	33.37	32.63	33.47	39.43	37.90	39.10	38.40	38.13
52-56	36.60	35.03	34.73	36.07	29.00	30.03	31.67	33.17	35.10	35.50	35.03	37.13	35.67
56-60	36.97	35.83	36.50	37.03	31.10	31.63	32.10	31.40	34.70	36.27	34.83	35.40	37.83
60-64	35.40	32.83	33.90	37.83	28.40	29.57	27.40	28.47	33.17	35.83	35.83	33.97	33.77
Aver.	37.57B[1]	36.45B	37.01B	37.72B	31.72A	33.06A	32.57A	32.93A	37.55B	37.45B	38.02B	37.54B	37.42B
Duncan 5[2]		1.41	1.49	1.54	1.57	1.61	1.63	1.65	1.68	1.70	1.71	1.71	1.72
Duncan 1[3]		1.86	1.94	1.99	2.03	2.06	2.08	2.11	2.13	2.14	2.16	2.17	2.19

[1]Means followed by unlike letters are significantly ($P < 0.05$) different.

[2]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.05$) different by Duncan's test.

[3]Indicates Least Significant Ranges (LSR) of means required to be significantly ($P < 0.01$) different by Duncan's test.

diet containing isolated soy protein or calcium and vitamin D_3 levels were not adequate.

Percent mortality for the growing and laying periods of Experiment One is shown in Table 11. The rather high mortality experienced with both of the purified type diets (but especially the glucose-isolated soy) was due in part to a disease condition called Thrush or Candidiasis. Addition of a mold inhibitor (Nystatin) partly alleviated the problem. Nystatin was included in the purified diets at 65 mg per kg of diet for the remainder of the experiment. This drug is used to prevent crop mycosis and mycotic diarrhea.

The addition of 2 ppm Se to the diets resulted in the lowest mortality of any of the treatments. When selenium was increased to 8 ppm mortality increased in most instances. Arsenic only partially counteracted the toxic effects of 8 ppm selenium. However, arsenic included with 8 ppm selenium in the corn-soy diet seemed quite effective in reducing toxic effects of the selenium during the laying period. Perhaps additions of 2 ppm or less of selenium may exert a beneficial effect which results in lower mortality.

Eggs were incubated at four intervals during Experiment One to study fertility and hatchability and to obtain chicks for progeny studies. Results are summarized in Table 12. The values include the total number of eggs used from all four hatches. Fertility was not recorded for the first hatch so one of the hatchability rows represents the percent of eggs hatched for three individual hatches where

Table 11. Percent Mortality During the Growing and Laying Periods of Experiment One.

Growing Period (0-24 weeks)

Treatment	Diet Type		
	Corn-Soy	Glucose-Isolated Soy	Glucose-Torula Yeast
Basal	5.4 (3/55)[1]	17.3 (9/52)	27.3 (15/55)
0.2 ppm Se	------	------	22.6 (12/53)
2.0 ppm Se	0.0 (0/56)	9.8 (6/61)	18.3 (11/60)
8.0 ppm Se	0.0 (0/55)	20.0 (10/50)	31.5 (17/54)
8.0 ppm Se + As[2]	7.0 (4/57)	17.6 (9/51)	30.3 (20/66)

Laying Period (24-64 weeks)

Basal	17.8 (9/45)	56.8 (25/44)	25.7 (9/35)
0.2 ppm Se	------	------	27.9 (12/43)
2.0 ppm Se	15.6 (7/45)	51.1 (23/45)	20.0 (9/45)
8.0 ppm Se	46.7 (21/45)	60.0 (27/45)	8.6 (8/40)
8.0 ppm Se + As[2]	28.9 (13/45)	57.1 (24/42)	28.9 (13/45)

[1]Indicates number of birds which diet out of starting number.

[2]Arsenic at 15 ppm in first column, 8 ppm in the others.

fertility was determined and the other row the overall hatchability observed in all four hatches. Results indicate that selenium at 8 ppm has a dramatic effect on hatchability as evidenced by the poor performance obtained. In most cases this high level of selenium was toxic to the embryo during late stages (20-day embryos). It seems that edema noted in the posterior of the neck in the high selenium treatment may be due to a factor which manifests itself in disturbed fluid balance. Perhaps the embryos developed but fail to hatch because of energy depletion or nerve transmission problems which do not allow

Table 12. Percent Fertility and Hatchability of Eggs from Hens Fed Various Levels of Selenium and Arsenic.

Treatment (ppm Se and As)

Corn-Soy

0	2	8	8-15
80.9 (225/278)[1]	85.1 (296/348)	68.8 (141/205)	85.3 (220/258)
92.0 (207/225)[2]	93.9 (278/296)	33.3 (47/141)	73.6 (162/220)
91.3 (253/277)[3]	91.8 (390/425)	27.8 (55/198)	65.3 (241/369)

Glucose-Isolated Soy

0	2	8	8-8
59.5 (97/163)[1]	61.6 (162/263)	56.8 (79/139)	61.6 (90/146)
79.4 (77/97)[2]	76.5 (124/162)	5.1 (4/79)	31.1 (28/90)
82.4 (117/142)[3]	77.0 (164/213)	8.0 (9/113)	33.6 (41/122)

Glucose-Torula Yeast

0	0.2	2	8	8-8
60.5 (104/172)[1]	59.6 (130/218)	71.4 (132/185)	52.8 (65/123)	68.0 (85/125)
88.5 (92/104)[2]	92.3 (120/130)	89.4 (118/132)	49.2 (32/65)	75.3 (64/85)
90.1 (127/141)[3]	93.4 (142/152)	88.3 (143/162)	50.0 (42/84)	77.7 (93/120)

[1]Values in this row represent percent of total eggs set which were fertile in three hatches. Parentheses include number of fertile eggs over number set.

[2]Values in this row represent percent of fertile eggs which hatched in three hatches. Parentheses include number of eggs hatched over number of fertile eggs.

[3]Values in this row represent percent of fertile eggs which hatched in all four separate hatches. Parentheses include number of eggs hatched over number of fertile eggs.

the embryo to pip or break open the egg shell. No lack or deformity of extremities or the characteristic "wiry down" was observed among dead embryos.

The high level (8 ppm) of selenium appeared to reduce fertility. Due to the fact that many of the hens on the high selenium treatments layed poorly, it seems possible that problems with training of artificial insemination occurred. With these difficulties in mind, one whould probably attach more importance to the hatchability data.

Selenium included in the diets at 2 ppm did not seem to adversely affect hatchability of eggs from hens fed the corn-soy diet. However, when a similar level of selenium was fed in the purified diets there was a trend towards lower hatchability which was further evidenced by increasing selenium to 8 ppm. Selenium at 8 ppm had a particularly powerful influence on eggs from hens fed the glucose-isolated soy diet. Arsenic only partly overcame the toxicity observed with 8 ppm Se. Egg hatchability is, therefore, a rather sensitive measure of selenium toxicity. Results from individual hatches were quite consistent with the averages shown in Table 12.

Chicks hatched from several of the fertility-hatchability studies were used to study progeny performance. From one of the four hatches, chicks were saved from those hens which had received purified diets with 0, 0.2 and 2.0 ppm selenium. Equal numbers of mixed sex chicks hatched from eggs from dams receiving each of the diets were distributed into four replicate groups of ten chicks. The chicks were fed the yeast-soy chick starter shown in Table 4. Various low levels

of selenium (0, 0.05, 0.10, 0.20 and 0.40 ppm) were added to the starter diet in order to observe any growth response from selenium supplementation to a relatively low selenium diet (0.06 ppm). After four weeks, the four replicates of chicks averaged 166, 171, 182, 175 and 184 grams for the respective added selenium levels of 0, 0.05, 0.01, 0.20 and 0.40 ppm. The differences may indicate a trend toward improved growth with low level supplementation but one should recall that these were mixed sex chicks hatched from dams receiving varying levels of selenium in their diets.

In a later study chicks which were hatched in a fertility-hatchability study were fed the same starter diet (Table 4) supplemented with 0, 0.05 and 0.10 ppm Se. Again, these were mixed sex chicks hatched from dams receiving the different purified diet treatments. The chicks were equally distributed into replicate groups for the dietary treatments. At the end of two weeks the four replicate groups of eight chicks averaged 91, 91, and 91 grams for the 0, 0.05 and 0.10 ppm selenium treatments. After four weeks the chicks averaged 185, 203 and 203 grams for the respective treatments. It appears that in order to measure a growth response to added dietary selenium it would be desirable to use a basal diet extremely low in selenium and vitamin E. One would want to use chicks of the same sex and from dams receiving the same diet.

Chicks that hatched from eggs obtained from hens receiving the corn-soy diets were not used to study effects of low level selenium supplementation. These chicks were fed the yeast-soy chick starter

(Table 4) without additional selenium to observe any carry-over effects from their dam's dietary treatments. In the first study four replicates of ten chicks were obtained from each of the four hen treatments. Chick body weights after four weeks averaged 210, 208, 220, 211 grams respectively for the hen diet treatments of 0, 2, 8 ppm Se and the 8 ppm Se plus 15 ppm arsenic combination. In a similar second study, six chicks were used in each of four replicates from each maternal treatment. Four week body weights were 184, 168, 149 and 182 grams for the respective hen treatments of 0, 2 ppm, 8 ppm Se and 8 ppm Se plus 15 ppm As. In these studies mixed sex chicks were used so one should be cautious in making any conclusions. It seems that the 8 ppm Se diet had a detrimental effect on subsequent chick performance. No substantial reason can be offered to explain the differences in four week body weight between the two studies or the difference in performance of chicks from dams receiving 8 ppm Se.

Tissues and eggs were analysed for selenium at various intervals during Experiments One and Two. Results of these analyses are presented in Tables 13, 14 and 15.

Data taken from Thapar et al. (1969) are included in the table for comparative purposes. Note that in the present study, arsenic included in the corn-soy diet reduced liver selenium slightly but increased liver selenium in each of the purified type diets. This contrasts with the data of Thapar where the arsenic increased liver selenium in the corn-soy diet but not in the glucose-isolated soy

Table 13. Liver Selenium of Four-Week Old Male Chicks Fed Various Dietary Levels of Selenium (Experiment One).

Treatment (ppm Se)	Type of Diet		
	Corn-Soy (ppm Se)	Glucose-Isolated Soy (ppm Se)	Glucose-Torula Yeast (ppm Se)
0	0.75 (0.63)[2]	0.30 (0.35)[2]	0.29
0.2	-----	-----	0.55
2.0	1.34 (0.89)	1.11 (0.79)	1.01
8.0	2.90 (4.69)	2.77 (2.46)	3.60
8.0 + As[1]	2.55 (9.45)	3.60 (2.04)	7.16

[1]Arsenic at 15 ppm in first column and 8 ppm in the others.

[2]Data from Thapar et al. (1969) where similar diets and selenium additions were used.

diet. Liver selenium analysis from chicks on the basal diets for the two studies agree quite well, although the corn-soy diet used in the present study may have contained slightly more selenium.

Table 14 lists the values obtained for selenium analysis of the various tissues taken at 64 weeks of age. Only hens receiving the purified diets were sacrificed at this time, so the data does not include information from the corn-soy treatments. Each value given includes the average of three replicates of a pooled sample from five hens.

Increasing the dietary selenium resulted in increased tissue deposition in all cases. The rate of increase was not linear (8 ppm did not cause four times the deposition of that encountered with 2 ppm). Liver and kidney were especially high in selenium compared to the other

Table 14. Effect of Selenium Supplementation on Tissue Selenium Deposition (Experiment One).

Tissue[1]	Treatment								
	Glucose-Isolated Soy[2] (ppm Se)				Glucose-Torula Yeast[3] (ppm Se)				
	0	2	8	8-8[4]	0	0.2	2	8	8-8[4]
Liver	0.33	0.93	2.53	2.12	0.28	0.55	1.00	2.45	2.88
Kidney	0.37	0.94	2.31	1.98	0.40	0.71	1.15	2.46	2.37
Heart	0.16	0.32	0.95	0.83	0.18	0.27	0.43	1.23	1.05
Breast	0.09	0.18	0.26	0.30	0.06	0.10	0.16	0.23	0.29
Thigh	0.11	0.17	0.30	0.32	0.10	0.15	0.18	0.30	0.35
Feathers	0.27	1.36	3.32	4.97	0.33	0.37	1.11	3.42	3.10

[1]Value given in ppm wet weight basis.

[2]Basal diet contains 0.02 ppm selenium.

[3]Basal diet contains 0.07 ppm selenium.

[4]Selenium added at 8 ppm and arsenic at 8 ppm.

tissues. Arsenic had little effect on liver and kidney selenium deposition.

The other tissues showed varied responses to the arsenic but showed increased selenium content as the dietary selenium supplements were increased. Feathers contained rather high levels of selenium.

Recently, Jensen (1970) reported that dietary additions of selenium decreased liver fat of laying hens. Determinations of liver fat of hens fed the glucose-isolated soy diet in this study showed livers contained 22.8 percent fat on the dry weight basis. The same diet with 2 ppm Se added produced livers containing 26.5 percent fat. Livers

from hens fed the glucose-Torula yeast diet contained 25.7, 25.3 and 25.0 percent fat for the 0, 0.2 and 2 ppm Se treatments, respectively.

Table 15. Effect of Dietary Selenium Supplementation on Deposition of Selenium in Whole Eggs (Experiment One).

Diet	Treatment (ppm Se)	Age of Hens (weeks) 32	42	52	62	96	104
Corn-Soy	0	0.44[1]	0.53	0.47	0.48	0.40	0.46
	2	0.56	0.77	0.71	0.64	0.59	0.57
	8	1.46	1.83	1.82	1.86	1.70	1.50
	8-15[2]	1.88	1.54	1.46	1.77	1.21	1.28
Glucose-Isolated Soy	0	0.23	0.13	0.17	0.14	----	----
	2	0.44	0.61	0.58	0.53	----	----
	8	1.22	1.74	1.76	1.64	----	----
	8-8[3]	1.14	1.33	1.40	1.47	----	----
Glucose-Torula Yeast	0	0.15	0.13	0.14	0.11	----	----
	0.2	0.31	0.38	0.40	0.28	----	----
	2	0.59	0.57	0.69	0.60	----	----
	8	1.52	1.89	1.96	1.99	----	----
	8-8[3]	1.21	1.29	1.34	1.56	----	----

[1]Values on wet basis as ppm selenium.

[2]Selenium at 8 ppm and arsenic at 15 ppm.

[3]Selenium at 8 ppm and arsenic at 8 ppm.

The egg selenium contents responded to dietary supplements of selenium. Again the increase observed was not a linear response because a lower percentage of selenium from high levels was deposited than from lower levels. It is of interest that there was more selenium

deposited in the eggs from the corn-soy basal diet than those from the glucose-Torula yeast diet containing the 0.2 ppm added selenium. In most cases, adding 2 ppm to either of the purified diets resulted in a selenium level in eggs only slightly higher than those observed with the unsupplemented corn-soy basal.

There was no consistent tendency for egg selenium levels to increase with time for hens receiving the corn-soy diet. It appeared that arsenic tended to increase egg selenium with time for hens on the two purified diets. The corn-soy basal contained an average of 0.40 ppm Se, the glucose-isolated soy diet 0.07 ppm Se and the glucose-Torula yeast diet 0.02 ppm selenium.

Experiment Two

Hens that had been fed the practical type corn-soy diet in Experiment One were used in this study. Body weights of the means of the three replicate groups of hens on each treatment are shown in Table 16. Levels of selenium fed were the same as those used in Experiment One. Hens fed the basal diet (0 ppm Se added) were heaviest throughout the study. Those fed the selenium-arsenic combination weighed nearly the same as the basal group. When 2 ppm Se was fed there was a slight reduction in body weight as with the 8 ppm Se fed groups. Note that at 72 weeks, all hens weighed the least, which was due to the weight loss during the forced molt period.

Table 16. Body Weights of Yearling Hens (kg) by Four Week Intervals (Experiment Two).

Age (weeks)	Treatment (ppm Se) 0	2	8	8-15[1]
68	2.11	2.00	1.96	2.02
72	1.94	1.78	1.83	1.77
76	1.85	1.85	1.85	1.81
80	2.11	2.01	2.00	2.00
84	2.18	2.09	2.03	2.07
88	2.27	2.21	2.15	2.27
92	2.24	2.17	2.16	2.19
96	2.27	2.19	2.22	2.25
100	2.21	2.14	2.15	2.24
104	2.22	2.14	2.15	2.27

[1]Eight ppm selenium and 15 ppm arsenic.

Mortality encountered is shown in Table 17. Values enclosed in parenthesis indicate the number of hens lost out of number of hens at the start of Experiment Two. The values listed for 64-76 weeks include the molt period during which time one-half the total mortality occurred in the basal and 2 ppm supplemented groups. The highest losses were in the basal group. Note that selenium fed at 2 ppm lowered mortality. However, when selenium was increased to 8 ppm, more of the hens died and arsenic had little effect on protecting against this toxicity. Values listed for 24-104 weeks include mortality throughout the laying periods of Experiment One and Two.

Hen-day egg production was recorded for seven 28-day periods after the hens molted. This included the time interval from 76-104 weeks of

Table 17. Percent Mortality of Yearling Hens (Experiment Two).

Time Interval (weeks)	Treatment (ppm Se) 0	2	8	8-15[1]
64-76	13.5 (5/37)	5.3 (2/38)	4.2 (1/24)	6.4 (2/31)
64-104	27.0 (10/37)	10.5 (4/38)	16.7 (4/24)	18.8 (6/32)
24-104	40.0 (18/45)	24.5 (11/45)	57.8 (26/45)	42.2 (19/45)

[1]Eight ppm selenium and 15 ppm arsenic.

age. Results are shown in Table 18. Each value given is the average for each treatment calculated from total eggs from all three replicates divided by total hen days from the three replicates.

It should be noted that a significant F test was obtained for periods. This was due to the lower production during the first period. Other than during the first period, production was not different from period to period within any given treatment.

The average egg weights are shown in Table 19. Each figure in the table represents the average of three replicates of forty eggs. Although no significant F tests were observed, the eggs from the hens fed 2 and 8 ppm Se were consistently 1.5 to 2 grams lighter. Periods did not differ because hens were fully mature when Experiment Two was initiated.

Interior egg quality measured by Haugh Units were also observed. Values listed in Table 20 represent the average of three replicates

Table 18. Percent Hen-Day Egg Production and Analysis of Variance (Experiment Two).

	Treatment (ppm Se)			
Age (weeks)	0	2	8	8-15[1]
68	57.6	64.1	47.3	49.3
Molt				
80	24.6	27.6	24.8	24.5
84	51.9	55.3	39.8	46.8
88	56.9	53.3	42.2	46.9
92	47.3	47.3	40.8	44.6
96	47.4	52.8	40.5	43.9
100	51.2	51.4	47.7	49.3
104	48.0	52.1	40.6	46.3
Average	46.7	48.6	39.5	43.2

Analysis of Variance

Source	df	SS	MS	F
Treatment	3	629.16	209.72	0.54
Period	6	5596.17	932.70	22.01**
Block	2	1549.75	774.88	
T X P	18	367.36	20.41	0.70
T X B	6	2349.60	391.60	
P X B	12	508.47	42.37	
T X P X B	36	1044.94	29.03	

[1]Eight ppm selenium and 15 ppm arsenic

**Significantly different at ($P < 0.01$).

of ten eggs each for each period. There was a decrease ($P < 0.01$) in interior quality as the hens aged. Perhaps this is due to a slightly larger egg. Selenium or arsenic showed no adverse effect on interior egg quality.

Table 19. Egg size and Analysis of Variance (Experiment Two).

Age (weeks)	Treatment (ppm Se) 0	2	8	8-15
	gm	gm	gm	gm
80	61.3	58.4	59.0	59.6
84	61.6	59.7	58.8	59.7
88	61.2	60.0	59.7	60.3
92	61.0	59.5	60.2	60.7
96	60.4	59.1	58.9	60.3
100	61.2	59.2	59.6	61.7
104	61.6	59.8	59.9	61.8
Average	61.0	59.4	59.4	60.9

Analysis of Variance

Source	df	SS	MS	F
Treatment	3	49.46	16.49	0.65
Period	6	13.62	2.27	1.25
Block	2	24.78	12.39	
T X P	18	14.02	0.78	0.04
T X B	6	152.72	25.45	
P X B	12	21.91	1.82	
T X P X B	36	633.78	17.60	

Egg shell thickness was measured so as to observe any influence of high levels of selenium or arsenic on the egg shell quality. The values shown on Table 21 are averages of three replicates of ten eggs per treatment for each period. There was a significant decrease ($P < 0.01$) in shell thickness as hens ages. One could expect this was a normal

Table 20. Interior Egg Quality (Haugh Units) and Analysis of Variance (Experiment Two).

	Treatment (ppm Se)			
Age (weeks)	0	2	8	8-15
80	74	74	73	79
84	68	72	71	70
88	76	74	75	78
92	72	71	70	72
96	72	72	71	71
100	67	64	67	68
104	71	68	71	71
Average	71	71	71	73

Analysis of Variance

Source	df	SS	MS	F
Treatment	3	61.14	20.38	0.32
Period	6	707.50	117.92	32.75**
Block	2	96.29	48.15	
T X P	18	126.38	7.02	0.93
T X B	6	382.79	63.80	
P X B	12	43.19	3.60	
T X P X B	36	271.20	7.53	

**Significantly different at ($P < 0.01$).

change associated with aging. The shells become thinner with increasing bird age except during the last period (100-104 weeks). Perhaps this was due to lower egg production during the last period.

Feed conversion was slightly improved when 2 ppm Se was added. Table 22 expresses fed conversion as kilograms of feed per dozen of eggs and as grams of feed required per gram of egg. The values represent

Table 21. Egg Shell Thickness (mm X 10^2) and Analysis of Variance (Experiment Two).

Age (weeks)	Treatment (ppm Se) 0	2	8	8-15
80	41.4	37.2	38.0	36.7
84	38.0	37.1	38.8	37.5
88	38.9	37.9	39.9	38.3
92	37.4	36.4	37.4	37.8
96	38.4	34.7	37.5	37.3
100	36.3	33.2	39.3	35.6
104	38.8	37.4	39.3	39.4
Average	38.4	36.3	38.1	37.5

Analysis of Variance

Source	df	SS	MS	F
Treatment	3	45.79	15.26	1.03
Period	6	103.66	17.28	12.08**
Block	2	8.33	4.16	
T X P	18	22.67	1.26	0.82
T X B	6	87.97	14.66	
P X B	12	17.20	1.43	
T X P X B	36	55.39	1.54	

**Significantly different at ($P < 0.01$).

a replicate average for all seven production periods. Although less feed was required to produce a dozen eggs when hens were fed 2 ppm added selenium, the eggs were slightly smaller (see Table 19.). Replicate C of the group fed 8 ppm Se contained only six hens which layed at a fairly good rate. Therefore, the 2.70 kilograms of feed required per dozen eggs would probably not be representative. Arsenic appeared to overcome the toxic effects of feeding 8 ppm Se as evidenced by feed conversion values similar to the unsupplemented treatment. Due to

Table 22. Feed Conversion Expressed as Kilograms Feed Per Dozen Eggs and Grams of Feed Per Gram of Egg (Experiment Two).

Method	Replicate	Treatment (ppm Se) 0	2	8	8-15
Kg/Doz.	A	3.55	2.90	4.56	2.70
	B	3.89	3.17	3.93	3.61
	C	3.09	2.70	2.70	4.01
	Average	3.49	2.89	3.72	3.37
Gm Feed/ Gm Egg	A	4.73	4.14	6.38	3.92
	B	5.35	4.44	5.45	4.98
	C	4.30	3.72	3.84	5.26
	Average	4.79	4.04	5.21	4.66

variation among replicates these treatment difference were not significantly different from one another.

Eggs were sampled for whole egg selenium determination at 96 and 104 weeks. Results of these analysis were listed in Table 15 of Experiment One. Feeding selenium at 2 ppm for 104 weeks increased egg selenium only 0.1 ppm over the basal diet. Eight ppm Se essentially tripled egg selenium but this increased deposition was lowered by feeding arsenic.

Hens were inseminated near the end of Experiment Two (102 weeks). Eggs were saved for incubation starting 48 hours post insemination and for six subsequent days. Hens were reinseminated four days after the first insemination. Table 23 shows the number of eggs obtained from each replicate and the results for fertility and egg hatchability.

The unsupplemented hens produced eggs with the highest fertility. The adverse effect on fertility by feeding 2 ppm Se in Experiment One was not observed in this experiment. There were no significant differences in fertility due to treatments.

Egg hatchability was not adversely affected by 2 ppm Se or the 8 ppm Se plus 15 ppm arsenic treatments. However, 8 ppm Se significantly ($P < 0.01$) reduced egg hatchability. Dead embryos exhibited edema in the posterior portion of their head and neck perhaps due to the toxic treatment effects.

Table 23. Effects of Selenium and Arsenic on Percent Egg Fertility and Percent Hatchability (Experiment Two).

Fertility

	Treatment (ppm Se)			
Replicate	0	2	8	8-15
A	100.0 (16/16)[1]	67.6 (25/37)	86.7 (13/15)	73.7 (14/19)
B	89.7 (26/29)	88.2 (30/34)	82.4 (14/17)	83.3 (20/24)
C	96.7 (29/30)	82.9 (34/41)	72.7 (16/22)	86.4 (19/22)
Average	94.7	79.5	79.6	81.5

Analysis of Variance

Source	df	SS	MS	F
Total	11	986.62		
Treatment	3	510.91	170.30	2.30
Block	2	32.09	16.05	0.22
Error	6	443.62	73.94	

Table 23. Effects of Selenium and Arsenic on Percent Egg Fertility and Percent Hatchability (Experiment Two). (Con't.)

Hatchability

Replicate	Treatment (ppm Se) 0	2	8	8-15
A	81.2 (13/16)[2]	78.0 (18/25)	38.5 (5/13)	78.6 (4/14)
B	72.0 (18/25)	80.0 (24/30)	35.7 (5/14)	77.8 (14/18)
C	80.0 (20/25)	87.9 (29/33)	18.8 (3/16)	79.0 (15/19)
Average	77.3	80.7	30.2	78.4

Analysis of Variance

Source	df	SS	MS	F
Total	11	5636.64		
Treatment	3	5303.08	1767.69	33.75**
Block	2	19.24	9.62	0.18
Error	6	314.31	52.38	

[1]Values represent number of fertile eggs over number of eggs incubated.

[2]Values represent number of chicks hatched over number of fertile eggs transferred.

**Significantly different at ($P < 0.01$).

Experiment Three

Throughout the presentation and discussion of results of Experiment Three the following symbols will be used to identify the various terms as indicated: S(1) = Regional Control strain, S(2) = Dekalb 131 strain, D(1) = corn-soy diet, D(2) = glucose-isolated soy-Torula yeast diet,

C(1) = 30.5 X 45.7 cm cage, C(2) = 40.6 X 45.7 cm cage, E(1) = zero or 2 ppm vitamin E, E(2) = 10 ppm added vitamin E, L(1) = no added selenium, L(2) = 0.1 ppm added selenium, L(3) = 1.0 ppm added selenium and P(1) - P(7) are four week production periods beginning at 24 weeks of age through 52 weeks.

The averages for the seven four-week production periods are shown in Table 24. Hen-day egg production for the corn-soy diets was significantly ($P < 0.01$) higher than that obtained with the purified type diet. There were no significant differences due to vitamin E, level of selenium or cage size. There was a significant S X P interaction which indicates that one strain did not consistently produce higher than the other throughout all periods. Table 25 lists significant main effects, interactions and main effect means for the entire experiment.

Average egg weights were significantly ($P < 0.01$) different between strains and also between diet types. DeKalb 131 hens produced heavier eggs than did the Regional Control strain throughout the entire experiment. This was true for both diet types. There appeared to be a trend towards increased egg weight as the level of selenium was increased in the purified diets although the reverse was true for the corn-soy diets. Perhaps the higher level of selenium in the corn-soy diets (0.48 ppm) plus the additions were detrimental to the hens; whereas, with the lower selenium content in the purified diet (0.055 ppm) the additions merely provided a more optimum selenium level in the diet.

Table 24. Average Hen-Day Egg Production, Egg Weight, Body Weight and Hen-Housed Mortality for 28 Weeks (Experiment Three).

	Corn-Soy Diet						Glucose-Isolated Soy-Torula Yeast Diet					
	0 ppm Vitamin E			10 ppm Vitamin E			2 ppm Vitamin E			10 ppm Vitamin E		
	Se (ppm)			Se (ppm)			Se (ppm)			Se (ppm)		
	0	0.1	1.0	0	0.1	1.0	0	0.1	1.0	0	0.1	1.0
Hen Day Egg Production (Percent)												
Strain 1	73.5	69.1	69.1	69.4	74.3	71.7	43.1	43.2	38.6	42.0	40.2	38.9
Strain 2	71.1	70.5	66.0	70.6	67.8	70.6	36.6	42.2	41.1	40.5	35.1	38.0
Average Egg Weight (Gm)												
Strain 1	53.9	53.3	52.2	54.0	53.9	52.8	48.6	47.9	49.0	48.3	48.7	49.0
Strain 2	56.7	57.1	56.4	56.2	56.6	55.6	50.8	52.2	53.9	52.2	52.8	52.5
Average Body Weight (Kg)												
Strain 1	1.96	2.06	1.94	1.96	1.94	1.94	1.59	1.62	1.55	1.61	1.55	1.61
Strain 2	1.82	1.81	1.74	1.78	1.73	1.71	1.53	1.53	1.49	1.48	1.54	1.57
Hen-Housed Mortality (Percent)												
Strain 1	15.6	7.3	9.4	13.5	0	3.1	33.3	29.2	26.0	28.1	24.0	27.1
Strain 2	7.3	11.5	15.6	19.8	17.7	25.0	15.6	40.6	25.0	28.1	21.9	20.8

Table 25. Significant Main Effects and Interactions for Various Parameters Measured for 28 Weeks (Experiment Three).

Source	df	MS	F
Percent Hen-Day Egg Production			
Diet (D)	1	308996.0	772.76**
Periods (P)	6	25387.56	198.86**
D X P	6	3842.24	41.91**
Strain (S) X P	6	1009.67	20.97**
Average Egg Weight (gm)			
D	1	6354.48	253.19**
Vitamin (E) X D	1	11.26	12.26*
S	1	3849.56	39.25**
D X S	1	51.23	11.63*
Level Se (L) X D	2	148.83	18.45**
P	6	2714.35	923.28**
D X P	6	64.77	19.74**
D X S X P	6	7.99	2.68*
Average Body Weight (kg)			
Cage (C)	1	0.66	115.75**
E	1	0.14	12.99*
D	1	32.32	2249.13**
E X D	1	0.22	14.18*
S	1	6.25	182.21**
D X S	1	1.65	42.62**
C X E X D X L	2	0.49	5.50*
P	6	0.33	33.23**
D X P	6	0.38	40.27**
C X D X P	6	0.02	2.71*
D X S X P	6	0.02	3.49*

Table 25. Significant Main Effects and Interactions for Various Parameters Measured for 28 Weeks (Experiment Three). (Con't.)

Hen-Housed Mortality

Source	df	MS	F
D	1	13903.68	19.83*
P	6	7468.69	63.14**
D X P	6	2272.53	28.72**

Hen-Day Egg Production (Gm)

D	1	115493.12	1491.06**
P	6	9100.07	260.44**
D X P	6	1641.33	66.68**
S X P	6	298.36	21.93**

Haugh Units

E	1	61.12	24.34*
D	1	13082.47	1282.69**
S	1	947.04	123.06**
C X D X S X L	2	39.38	5.17*
P	6	2032.85	279.96**
D X P	6	673.28	103.25**
S X P	6	39.16	10.92**
C X S X P	6	11.11	3.88*

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

A significant ($P < 0.05$) E X D interaction indicated that vitamin E did not affect egg weight similarily for both diets. It appeared that vitamin E at 10 ppm was desirable in the purified diet but of no advantage in the corn-soy diet. The significant ($P < 0.05$) D X S interaction indicated that both strains did not perform the same on each respective diet. Likewise, the significant ($P < 0.05$) D X L interaction indicated that increasing levels of selenium did not give a similar response with both diets.

A significant ($P < 0.01$) difference in egg weight was obtained for periods. This could be expected because hens tend to lay heavier eggs as they get older. A significant D X P interaction indicated that there was not the same difference in egg size throughout the entire experiment.

Cage size had a significant ($P < 0.01$) effect on body weights of the hens. Those hens in C(1) averaged 1.73 kg and those in the larger C(2) cages 1.68 kg. Vitamin E had a significant ($P < 0.05$) effect on body weight with those fed E(1) averaging 1.72 kg compared to 1.70 kg for E(2). Diets also produced significantly ($P < 0.01$) different body weights with the corn-soy hens averaging 310 grams more than those fed the purified diet.

Strains also differed significantly ($P < 0.01$) in body weight with S(1) averaging 1.77 kg and S(2) only 1.64 kg. Periods also were significantly different ($P < 0.01$) in regard to body weight but hens tend to gain as they mature during time intervals such as used here. Significant

interactions regarding body weight include E X D ($P < 0.05$), D X S ($P < 0.01$), C X E X D X L ($P < 0.05$), D X P ($P < 0.01$), C X D X P ($P < 0.05$) and D X S X P ($P < 0.05$). With the confounding due to interaction it is not possible to discuss main effect differences in such a way as to make valid recommendations (see Table 25).

Values for hen-housed mortality are also included in Table 24. These figures represent the cumulative total mortality for the 32-week experiment. Mortality on the two diets differed significantly ($P < 0.05$). Mortality was higher in groups fed the purified diet. Any trends towards lower mortality due to selenium and vitamin E treatment could not be detected.

Results of body weight averages obtained at four-week intervals are shown in Table 26. The Analysis of Variance and means for significant main effects and significant interactions are shown in Table 27. For Period One, the cages, diets and strains were significantly different.

However, a D X S and C X L interaction was present so the means for the various levels of each factor are also shown in Table 27. During P (2), cages, diets and strains were significantly different, and there was a D X S interaction. Period (3) had significant cage, vitamin E, diet, and strain effects as well as significant D X S, C X D X L, and C X E X D X L interactions. In P(4) cage, diet, and strains differed and D X S, C X D X L and C X E X D X L interactions were present. With P(5) and P(6) only significant diet, strain and D X S effects were noted. During the last period, significant diet,

Table 26. Main Effect Means of Body Weights of Hens by Four-Week Intervals (Experiment Three).

Main Effect	Age (weeks) 28 kg	32 kg	36 kg	40 kg	44 kg	48 kg	52 kg
C(1)	1.70[1]	1.69[2]	1.70[2]	1.72[2]	1.73	1.75	1.80
C(2)	1.65	1.65	1.64	1.68	1.68	1.72	1.77
E(1)	1.68	1.68	1.69[1]	1.71	1.71	1.74	1.79
E(2)	1.67	1.65	1.65	1.69	1.70	1.73	1.78
D(1)	1.76[2]	1.78[2]	1.82[2]	1.89[2]	1.91[2]	1.92[2]	1.95
D(2)	1.60	1.55	1.52	1.52	1.50	1.56	1.62
S(1)	1.74[2]	1.74[2]	1.74[2]	1.77[2]	1.77[2]	1.81[2]	1.86
S(2)	1.61	1.60	1.61	1.64	1.64	1.66	1.71
L(1)	1.68	1.67	1.68	1.70	1.70	1.74	1.79
L(2)	1.68	1.67	1.68	1.72	1.73	1.75	1.79
L(3)	1.66	1.65	1.67	1.68	1.68	1.72	1.77
Aver.	1.68	1.67	1.67	1.70	1.70	1.74	1.78

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

strain, D X S, D X E X L and C X E interactions were present. Throughout all periods diets and strains were significantly different. The D X S interaction was due to a strain difference in degree of response to the diets. In several cases it appeared that L(3) was detrimental in D(1) but gave the optimum body weight in D(2). This was noted in P(1) P(4) and P(6), but was not completely true in P(3). Due to the number of complex interactions other trends are difficult to interpret.

Table 27. Significant Main Effects and Interactions of Body Weight (Experiment Three).

Period	Source	df[1]	MS	F	P
P(1)	Cage	1/3	0.14	14.32	*
	Diet	1/3	1.24	81.63	**
	Strain	1/3	0.72	75.37	**
	D X S	1/3	0.05	16.37	*
	C X L	2/6	0.03	7.65	*

Means (kg)

C(1) = 1.70	D1S1 = 1.84	C1L1 = 1.72
C(2) = 1.65	D1S2 = 1.69	C1L2 = 1.73
	D2S1 = 1.65	C1L3 = 1.67
D(1) = 1.76	D2S2 = 1.56	C2L1 = 1.65
D(2) = 1.60		C2L2 = 1.65
		C2L3 = 1.67
S(1) = 1.74		
S(2) = 1.61		

P(2)	Cage	1/3	0.07	53.54	**
	Diet	1/3	2.43	255.25	**
	Strain	1/3	0.96	199.68	**
	D X S	1/3	0.13	11.97	*

Means (kg)

C(1) = 1.69	D1S1 = 1.88
C(2) = 1.65	D1S2 = 1.69
	D2S1 = 1.61
D(1) = 1.78	D2S2 = 1.51
D(2) = 1.55	
S(1) = 1.74	
S(2) = 1.60	

Table 27. Significant Main Effects and Interactions of Body Weight (Experiment Three). (Con't.)

P(3)	Cage	1/3	0.16	68.54	**
	Vitamin E	1/3	0.08	21.55	*
	Diet	1/3	4.34	520.24	**
	Strain	1/3	0.77	99.82	**
	D X S	1/3	0.24	16.89	*
	C X D X L	2/6	0.09	10.67	*
	C X E X D X L	2/6	0.07	10.99	**

Means (kg)

C(1) = 1.70	C1D1L1 = 1.90	C1E2D1L1 = 1.84
C(2) = 1.64	C1D1L2 = 1.91	C1E2D1L2 = 1.75
	C1D1L3 = 1.77	C1E2D1L3 = 1.79
E(1) = 1.69	C1D2L1 = 1.55	C1E2D2L1 = 1.51
E(2) = 1.65	C1D2L2 = 1.55	C1E2D2L2 = 1.54
	C1D2L3 = 1.59	C1E2D2L3 = 1.62
D(1) = 1.82		
D(2) = 1.52	C2D1L1 = 1.78	C2E1D1L1 = 1.78
	C2D1L2 = 1.78	C2E1D1L2 = 1.76
S(1) = 1.74	C2D1L3 = 1.84	C2E1D1L3 = 1.87
S(2) = 1.61	C2D2L1 = 1.50	C2E1D2L1 = 1.50
	C2D2L2 = 1.51	C2E1D2L2 = 1.56
D1S1 = 1.93	C2D2L3 = 1.48	C2E1D2L3 = 1.46
D1S2 = 1.73		
D2S1 = 1.56	C1E1D1L1 = 1.96	C2E2D1L1 = 1.78
D2S2 = 1.50	C1E1D1L2 = 2.06	C2E2D1L2 = 1.79
	C1E1D1L3 = 1.75	C2E2D1L3 = 1.82
	C1E1D2L1 = 1.59	C2E2D2L1 = 1.50
	C1E1D2L2 = 1.55	C2E2D2L2 = 1.46
	C1E1D2L3 = 1.56	C2E2D2L3 = 1.50

P(4)	Cage	1/3	0.09	42.50	**
	Diet	1/3	6.55	327.50	**
	Strain	1/3	0.82	217.13	**
	D X S	1/3	0.35	92.93	**
	C X D X L	2/6	0.06	5.30	*
	C X E X D X L	2/6	0.08	7.03	*

Table 27. Significant Main Effects and Interactions of Body Weight (Experiment Three). (Con't.)

Means (kg)

C(1) = 1.72	C1D2L1 = 1.54	C1E2D1L1 = 1.92
C(2) = 1.68	C1D2L2 = 1.55	C1E2D1L2 = 1.82
	C1D2L3 = 1.53	C1E2D1L3 = 1.86
D(1) = 1.89	C2D1L1 = 1.84	C1E2D2L1 = 1.52
D(2) = 1.52	C2D1L2 = 1.86	C1E2D2L2 = 1.56
	C2D1L3 = 1.90	C1E2D2L3 = 1.56
S(1) = 1.77	C2D2L1 = 1.50	C2E1D1L1 = 1.85
S(2) = 1.64	C2D2L2 = 1.53	C2E1D1L2 = 1.82
	C2D2L3 = 1.48	C2E1D1L3 = 1.94
D1S1 = 2.00		C2E1D2L1 = 1.50
D1S2 = 1.78	C1E1D1L1 = 1.97	C2E1D2L2 = 1.56
D2S1 = 1.54	C1E1D1L2 = 2.12	C2E1D2L3 = 1.47
D2S2 = 1.50	C1E1D1L3 = 1.80	C2E2D1L1 = 1.84
	C1E1D2L1 = 1.56	C2E2D1L2 = 1.90
C1D1L1 = 1.94	C1E1D2L2 = 1.54	C2E2D1L3 = 1.87
C1D1L2 = 1.97	C1E1D2L3 = 1.50	C2E2D2L1 = 1.50
C1D1L3 = 1.82		C2E2D2L2 = 1.50
		C2E2D2L3 = 1.48

P(5)	Diet	1/3	8.20	1169.61	**
	Strain	1/3	0.79	44.29	**
	D X S	1/3	0.41	16.23	*

Means (kg)

D(1) = 1.91	S(1) = 1.77	D1S1 = 2.02
D(2) = 1.50	S(2) = 1.64	D1S2 = 1.80
		D2S1 = 1.51
		D2S2 = 1.48

P(6)	Diet	1/3	6.25	586.72	**
	Strain	1/3	1.05	190.32	**
	D X S	1/3	0.19	17.49	*
	C X L	2/6	0.07	6.71	*

Table 27. Significant Main Effects and Interactions of Body Weight (Experiment Three). (Con't.)

		Means (kg)			
	D(1) = 1.92	D1S1 = 2.03		C1L1 = 1.78	
	D(2) = 1.55	D1S2 = 1.80		C1L2 = 1.78	
		D2S1 = 1.61		C1L3 = 1.69	
	S(1) = 1.81	D2S2 = 1.51		C2L1 = 1.70	
	S(2) = 1.66			C2L2 = 1.72	
				C2L3 = 1.74	
P(7)	Diet	1/3	5.33	1042.86	**
	Strain	1/3	1.05	114.92	**
	D X S	1/3	0.31	48.48	**
	D X E X L	2/6	0.04	8.69	*
	C X E	1/3	0.04	10.51	*
		Means (kg)			
	D(1) = 1.95	C1E1 = 1.82		D1E2L1 = 1.97	
	D(2) = 1.61	C1E2 = 1.78		D1E2L2 = 1.90	
		C2E1 = 1.75		D1E2L3 = 1.90	
	S(1) = 1.86	C2E2 = 1.77		D2E1L1 = 1.62	
	S(2) = 1.71			D2E1L2 = 1.63	
		D1E1L1 = 1.96		D2E1L3 = 1.57	
	D1S1 = 2.07	D1E1L2 = 2.06		D2E2L1 = 1.61	
	D1S2 = 1.83	D1E1L3 = 1.93		D2E2L2 = 1.61	
	D2S1 = 1.65			D2E2L3 = 1.66	
	D2S2 = 1.58				

[1]Numerator degrees of freedom over degrees of freedom of factor used testing significance.

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

Results of hen-housed mortality are shown in Table 28. The various significant main effects and interactions are listed in Table 29. For P(1) only the C X L interaction was significant. In P(2) the E X D X S and C X D X S X L interaction were significant. In P(3) only the C X E interaction was significant. Diets were significantly different in their effect on mortality in P(4), P(5), P(6) and P(7). The only other significant interaction noted was E X L in P(5).

Table 28. Percent Hen-Housed Cumulative Mortality by Four-Week Intervals (Experiment Three).

Main Effects	Age (Weeks) 28	32	36	40	44	48	52
C(1)	3.1	6.6	8.7	13.2	16.7	17.5	19.2
C(2)	2.6	5.5	8.1	11.7	14.4	15.6	17.4
E(1)	2.4	5.9	7.6	12.8	14.9	17.3	18.2
E(2)	3.3	6.2	9.1	12.1	16.2	15.9	18.4
D(1)	3.7	6.7	7.5	8.5[1]	10.9[1]	11.5[2]	12.1[2]
D(2)	2.0	5.3	9.2	16.4	20.2	21.7	24.0
S(1)	2.3	4.7	7.4	11.8	14.2	15.0	16.6
S(2)	3.4	7.4	9.4	13.1	16.8	18.2	20.0
L(1)	4.8	7.5	9.8	14.2	16.5	17.8	19.1
L(2)	3.0	5.3	8.5	11.6	15.9	16.7	18.1
L(3)	0.8	5.2	6.9	11.6	14.1	15.3	17.7
Aver.	2.9	6.0	8.4	12.4	15.5	16.7	18.3

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

Table 29 Significant Main Effects and Interaction of Cumulative Hen-Housed Mortality (Experiment Three).

	Source	df	MS	F
P(1)	C X L	2/6	235.02	5.41*

Means (percent)

C1L1 = 7.3	C2L1 = 2.4
C1L2 = 2.1	C2L2 = 3.9
C1L3 = 0	C2L3 = 2.4

P(2)	E X D X S	1/3	494.40	14.94*
	C X D X S X L	2/6	347.14	5.27*

Means (percent)

E1D1S1	7.3	C1D1S1L1 = 12.5	C2D1S1L1 = 6.2
E1D1S2	4.9	C1D1S1L2 = 4.2	C2D1S1L2 = 3.2
E1D2S1	3.8	C1D1S1L3 = 0	C2D1S1L3 = 6.2
E1D2S2	7.6	C1D1S2L1 = 8.3	C2D1S2L1 = 3.2
E2D1S1	3.5	C1D1S2L2 = 8.4	C2D1S2L2 = 6.3
E2D1S2	11.5	C1D1S2L3 = 8.3	C2D1S2L3 = 6.2
E2D2S1	4.2	C1D2S1L1 = 4.2	C2D2S1L1 = 3.2
E2D2S2	5.6	C1D2S1L2 = 0	C2D2S1L2 = 6.3
		C1D2S1L3 = 4.2	C2D2S1L3 = 3.2
		C1D2S2L1 = 8.3	C2D2S2L1 = 6.2
		C1D2S2L2 = 5.6	C2D2S2L2 = 3.2
		C1D2S2L3 = 0	C2D2S2L3 = 6.3

P(3)	C X E	1/3	225.98	10.94*

Means (percent)

C1E1 = 9.0	
C1E2 = 8.3	
C2E1 = 6.3	
C2E2 = 9.9	

Table 29 Significant Main Effects and Interaction of Cumulative Hen-Housed Mortality (Experiment Three). (Con't.)

P(4)	Diet	1/3	2991.73	16.41*	
		Means (percent)			
		D(1) = 8.5	D(2) = 16.4		
P(5)	Diet	1/3	6390.61	19.44*	
	E X L	2/6	765.48	5.16*	
		Means (percent)			
	D(1) = 9.7	E1L1 = 13.0	E2L1 = 20.0		
	D(2) = 21.3	E1L2 = 18.5	E2L2 = 13.3		
		E1L3 = 16.4	E2L3 = 11.7		
P(6)	Diet	1/3	4770.64	47.99	**
		Means (percent)			
		D(1) = 11.5	D(2) = 21.7		
P(7)	Diet	1/3	6589.25	180.75	**
		Means (percent)			
		D(1) = 12.1	D(2) = 24.0		

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

During P(1) increasing levels of selenium lowered mortality for hens in C(1) but not those in C(2). No consistent trends were noted from interactions observed in P(2), P(3) and P(5). The practical corn-soy diet had significantly lower mortality than that encountered with the purified diet.

Table 30 lists results of the main effects of percent hen-day egg production by four week intervals in Experiment Three. The significant

Table 30. Percent Hen-Day Egg Production by Four Week Intervals (Experiment Three).

Main Effects	Age (weeks) 28	32	36	40	44	48	52
C(1)	30.1	65.6	61.8	59.8	56.3	56.2	58.2
C(2)	29.5	63.5	59.7	58.0	56.4	56.3	59.6
E(1)	30.1	65.2	60.6	59.0	56.5	56.6	58.8
E(2)	29.5	63.8	60.9	58.8	56.3	55.9	59.1
D(1)	38.1[2]	77.1[2]	80.6[2]	77.7[2]	75.6[2]	72.3[2]	70.5[2]
D(2)	21.5	52.0	40.9	40.1	37.2	40.2	47.4
S(1)	34.3[2]	67.7[1]	62.3	59.9	55.7	54.5	57.7
S(2)	25.3	61.3	59.2	57.9	57.1	58.0	60.1
L(1)	29.4	65.2	62.1	59.7	57.2	56.6	60.6
L(2)	29.7	65.2	59.8	58.0	57.2	57.6	59.1
L(3)	30.3	63.1	60.3	59.0	54.8	54.6	57.0
Aver.	29.8	64.5	60.7	58.9	56.4	56.3	58.9

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

main effects, interactions and their means are listed in Table 31. There was a significant difference between diets in all periods. Strains had significantly different percent hen-day production in the early two periods with the Regional Control strain having the higher rate of production. No consistent trends were noted with the C X D X L interaction in P(1) or the D X S X L interaction in P(6).

Results of hen-day egg production expressed as grams of egg per hen per day are listed in Table 32. Table 33 lists the significant main effects and interactions for hen-day production expressed in grams per hen per day. Diets produced a significant ($P < 0.01$) difference in production in all seven periods. Strains differed ($P < 0.05$) only during period one. The only interaction noted was D X E X L but no consistent trend was noted from the means (see Table 33).

Main effect means from egg weight are shown in Table 34. Type of diet consistently produced a significantly ($P < 0.01$) different egg weight. A D X L interaction was noted in five of seven periods due to a decreased egg size when selenium levels were increased in corn-soy diets but a reverse effect with the glucose-isolated soy-Torula yeast diet. Strains produced significantly ($P < 0.01$ or $P < 0.05$) different egg weights in all periods of this study. The significant main effects and interactions are listed in Table 35.

Main effect means of Haugh Units are given in Table 36. With consistency diets and strains showed significantly ($P < 0.01$ or $P < 0.05$)

Table 31. Significant Main Effects and Interactions of Percent Hen-Day Egg Production (Experiment Three).

	Source	df	MS	F
P(1)	Diet	1/3	13132.41	97.28**
	Strain	1/3	3847.60	35.26**
	C X D X L	2/6	320.57	5.21*

Means (percent)

D(1) = 38.1	C1D1L1 = 33.6	C2D1L1 = 40.4
D(2) = 21.5	C1D1L2 = 23.5	C2D1L2 = 19.4
	C1D1L3 = 38.8	C2D1L3 = 37.8
S(1) = 34.3	C1D2L1 = 23.4	C2D2L1 = 20.3
S(2) = 25.3	C1D2L2 = 21.6	C2D2L2 = 19.3
	C1D2L3 = 20.3	C2D2L3 = 24.6

P(2)	Diet	1/3	30290.67	325.21**
	Strain	1/3	1973.75	16.54*

Means (percent)

D(1) = 77.1	S(1) = 67.7
D(2) = 52.0	S(2) = 61.3

P(3)	Diet	1/3	75815.12	413.96**

Means (percent)

D(1) = 80.6	D(2) = 40.9

P(4)	Diet	1/3	68086.12	433.85**

Table 31 Significant Main Effects and Interactions of Percent Hen-Day Egg Production (Experiment Three). (Con't.)

			Means (percent)	
			D(1) = 77.7 D(2) = 40.1	
P(5)	Diet	1/3	68097.15	358.10**
			Means (percent)	
			D(1) = 75.6 D(2) = 37.6	
P(6)	Diet	1/3	47917.27	341.45**
	Diet X Strain X Level	2/6	348.17	5.46*
			Means (percent)	
	D(1) = 72.3		D1S1L1 = 68.8	D2S1L1 = 42.1
	D(2) = 40.2		D1S1L2 = 72.9	D2S1L2 = 39.5
			D1S1L3 = 70.4	D2S1L3 = 33.4
			D1S2L1 = 75.1	D2S2L1 = 40.5
			D1S2L2 = 76.2	D2S2L2 = 41.6
			D1S2L3 = 70.4	D2S2L2 = 44.2
P(7)	Diet	1/3	23571.36	141.88**
			Means (percent)	
			D(1) = 70.5 D(2) = 48.0	

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

Table 32. Grams of Egg Produced Per Hen Per Day by Four Week Intervals (Experiment Three).

Main Effects	Age (weeks) 28	32	36	40	44	48	52
C(1)	14.3	32.4	32.3	32.2	31.3	32.0	33.6
C(2)	13.9	31.4	31.4	31.3	31.3	32.0	34.4
E(1)	14.4	32.3	31.9	31.8	31.4	32.1	33.9
E(2)	13.8	31.5	31.8	31.8	31.2	31.9	34.1
D(1)	18.2[2]	39.4[2]	43.7[2]	43.2[2]	43.2[2]	42.5[2]	41.7[2]
D(2)	10.0	24.4	20.0	20.3	19.4	21.6	26.3
S(1)	15.8[1]	32.5	31.6	31.1	29.9	30.3	32.4
S(2)	12.4	31.4	32.2	32.5	32.6	33.7	35.6
L(1)	13.9	32.2	32.6	32.3	31.8	32.2	35.2
L(2)	14.1	32.3	31.5	31.3	31.9	33.0	34.0
L(3)	14.3	31.2	31.4	31.8	30.1	30.8	32.7
Aver.	14.1	31.9	31.9	31.8	31.3	32.0	34.0

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

different Haugh Unit values. Dekalb 131 hens produced eggs with superior interior quality during all periods of the experiment. Hens fed the purified type diet had higher Haugh Unit values. This perhaps was due to the smaller eggs obtained from hens fed these treatments.

Several other interactions were noted (see Table 37). Differences observed from the means of the interactions were minor and do not indicate important trends.

Table 33. Significant Main Effects and Interactions of Hen-Day Production Expressed as Gm Egg Per Hen Per Day (Experiment Three).

	Source	df	MS	F
P(1)	Diet	1/3	3285.91	127.79**
	Strain	1/3	566.67	13.65*

Means (Gm Egg/Hen/Day)

D(1) = 18.24 S(1) = 15.82
D(2) = 9.96 S(2) = 12.38

P(2)	Diet	1/3	10823.25	607.40**

Means (gm)

D(1) = 39.42 D(2) = 22.31

P(3)	Diet	1/3	27122.02	813.01**

Means (gm)

D(1) = 43.75 D(2) = 19.98

P(4)	Diet	1/3	25126.12	709.27**
	D X E X L	2/6	109.96	5.24*

Means (gm)

D(1) = 43.22
D(2) = 20.34

D1E1L1 = 45.08	D2E1L1 = 18.88
D1E1L2 = 41.88	D2E1L2 = 22.71
D1E1L3 = 40.66	D2E1L3 = 21.36
D1E2L1 = 43.95	D2E2L1 = 21.14
D1E2L2 = 43.01	D2E2L2 = 17.71
D1E2L3 = 44.76	D2E2L3 = 20.26

Table 33. Significant Main Effects and Interactions of Hen-Day Production Expressed as Gm Egg Per Hen Per Day (Experiment Three). (Con't.)

	Source	df	MS	F
P(5)	Diet	1/3	26113.04	598.72**
		Means (gm)		
		D(1) = 43.2	D(2) = 19.6	
P(6)	Diet	1/3	19518.67	401.73**
		Means (gm)		
		D(1) = 42.5	D(2) = 22.0	
P(7)	Diet	1/3	10771.31	218.33**
		Means (gm)		
		D(1) = 41.7	D(2) = 26.5	

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

Table 34. Main Effect Means of Egg Weights by Four-Week Intervals (Experiment Three).

Main Effects	Age (Weeks) 28	32	36	40	44	48	52
C(1)	46.7	49.0	51.4	52.9	54.5	56.1	57.2
C(2)	46.8	49.1	51.8	53.3	54.6	56.0	57.4
E(1)	46.7	49.1	51.6	53.0	54.6	55.9	57.3
E(2)	46.8	49.0	51.5	53.2	54.4	56.2	57.2
D(1)	47.8[2]	51.1[2]	54.3[2]	55.6[2]	57.0[2]	58.6[1]	59.2[2]
D(2)	45.7	47.0	48.9	50.7	52.0	53.5	55.4
S(1)	45.2[1]	47.5[2]	49.7[2]	51.0[1]	52.8[2]	54.6[2]	55.8[2]
S(2)	48.4	50.7	53.4	55.2	56.3	57.5	58.7
L(1)	46.5	48.9	51.5	53.1	54.6	55.8	57.5
L(2)	46.8	49.2	51.7	53.2	54.9	56.5	57.1
L(3)	47.0	49.1	51.5	53.1	54.1	55.9	57.2
Aver.	46.8	49.1	51.6	53.1	54.5	56.1	57.3

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

Table 38 lists the percent fertility and hatchability summaries from two separate hatches. The treatment average is also given, as calculated from the combined totals of both hatches. Fertility was significantly different ($P < 0.01$) for hens on the two types of diets. The eggs from hens fed the corn-soy diet had 85.7 percent fertility compared to 37.7 percent fertility for eggs from hens fed the purified type diet. No significant effect of selenium or vitamin E was detected.

Table 35. Significant Main Effects and Interactions of Egg Weight (Experiment Three).

	Source	df	MS	F
P(1)	Diet	1/3	200.08	58.67**
	D X E	1/3	33.33	189.39**
	Strain	1/3	499.23	20.38*
	D X L	2/6	24.62	7.93*
	C X E X S X L	2/6	5.66	5.32*

Means (gm)

D(1) = 47.8
D(2) = 45.8

D1E1 = 48.2
D1E2 = 47.4
D2E1 = 45.4
D2E2 = 46.2

S(1) = 45.2
S(2) = 48.4

D1L1 = 48.2
D1L2 = 47.8
D1L3 = 47.5
D2L1 = 44.9
D2L2 = 45.8
D2L3 = 46.7

C1E1S1L1 = 44.7
C1E1S1L2 = 45.2
C1E1S1L3 = 44.0
C1E1S2L1 = 48.2
C1E1S2L2 = 48.1
C1E1S2L3 = 49.8
C1E2S1L1 = 44.2
C1E2S1L2 = 45.8
C1E2S1L3 = 46.6
C1E2S2L1 = 48.0
C1E2S2L2 = 48.8
C1E2S2L3 = 47.9
C2E1S1L1 = 45.6
C2E1S1L2 = 44.6
C2E1S1L3 = 45.4
C2E1S2L1 = 47.5
C2E1S2L2 = 49.1
C2E1S2L3 = 49.4
C2E2S1L1 = 45.4
C2E2S1L2 = 45.2
C2E2S1L3 = 45.8
C2E2S2L1 = 49.0
C2E2S2L2 = 47.8
C2E2S2L3 = 48.0

P(2)	Diet	1/3	797.89	155.35**
	Strain	1/3	504.40	47.37**
	Diet X Level	2/6	14.66	20.68**

Table 35. Significant Main Effects and Interactions of Egg Weight (Experiment Three). (Con't.)

Means (gm)

D(1) = 51.1		D1L1 = 48.2	
D(2) = 47.1		D1L2 = 47.8	
		D1L3 = 47.5	
S(1) = 47.5		D2L1 = 44.9	
S(2) = 50.7		D2L2 = 45.8	
		D2L3 = 46.7	
P(3)	Diet	1/3	1380.31	338.31**
	Strain	1/3	669.01	50.31**
	Diet X Level	2/6	18.69	19.88**

Means (gm)

D(1) = 54.3		D1L1 = 54.6	
D(2) = 48.9		D1L2 = 54.7	
		D1L3 = 53.6	
S(1) = 49.7		D2L1 = 48.6	
S(2) = 53.4		D2L2 = 48.8	
		D2L3 = 49.5	
P(4)	Diet	1/3	1154.44	120.26**
	Strain	1/3	841.68	25.46*
	D X E X L	2/6	19.94	17.56**

Means (gm)

D(1) = 55.6	D1E1L1 = 55.9	D2E1L1 = 49.8
D(2) = 50.7	D1E1L2 = 56.0	D2E1L2 = 50.6
	D1E1L3 = 54.2	D2E1L3 = 51.6
S(1) = 51.0	D1E2L1 = 55.7	D2E2L1 = 51.0
S(2) = 55.2	D1E2L2 = 55.8	D2E2L2 = 50.6
	D1E2L3 = 56.0	D2E2L3 = 50.6

Table 35. Significant Main Effects and Interactions of Egg Weight (Experiment Three). (Con't.)

P(5)	Diet	1/3	1189.04	141.67**
	Strain	1/3	575.85	122.38**

Means (gm)

D(1) = 57.0	S(1) = 52.8
D(2) = 52.0	S(2) = 56.3

P(6)	Diet	1/3	1217.19	103.10*
	Strain	1/3	384.51	16.43**
	D X L	2/6	46.08	6.20*

Means (gm)

D(1) = 58.6	D1L1 = 58.8
D(2) = 53.5	D1L2 = 59.6
	D1L3 = 57.5
S(1) = 54.6	D2L1 = 52.7
S(2) = 57.5	D2L2 = 53.4
	D2L3 = 54.3

P(7)	Diet	1/3	713.65	159.30**
	Strain	1/3	420.08	44.38**
	D X L	2/6	44.08	16.12**
	E X L	2/6	17.89	5.29*

Means (gm)

D(1) = 59.2	D1L1 = 60.0	E1L1 = 57.4
D(2) = 55.3	D1L2 = 59.4	E1L2 = 56.7
	D1L3 = 58.1	E1L3 = 57.8
S(1) = 55.7	D2L1 = 54.9	E2L1 = 57.5
S(2) = 58.7	D2L2 = 54.9	E2L2 = 57.5
	D2L3 = 56.2	E2L3 = 56.5

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

Table 36. Main Effect Means of Haugh Units by Four Week Intervals (Experiment Three).

Main Effects	Age (Weeks)						
	28	32	36	40	44	48	52
C(1)	89.2	87.3	86.2	84.1	82.0	81.3	80.8
C(2)	88.4	87.6	86.2	84.0	82.2	81.1	79.9
E(1)	88.8	87.6	86.5	84.3	82.3	81.4	80.7
E(2)	88.7	87.3	85.8	83.8	82.0	81.0	80.0
D(1)	88.2	86.5[1]	83.0[2]	79.9[2]	77.9[2]	75.3[2]	77.4[2]
D(2)	89.4	88.4	89.3	88.2	86.4	87.0	83.3
S(1)	88.6	87.1[1]	85.6[1]	82.9[2]	80.8[2]	79.9[2]	79.3[1]
S(2)	89.0	87.8	86.7	85.2	83.5	82.4	81.4
L(1)	89.2	87.6	85.9	84.2	82.4	81.3	80.2
L(2)	88.7	87.3	86.1	83.9	81.5	80.9	80.8
L(3)	88.5	87.5	86.4	84.1	82.4	81.3	80.0
Aver.	88.8	87.4	86.2	84.1	82.1	81.2	80.4

[1]Significantly different at ($P < 0.05$).

[2]Significantly different at ($P < 0.01$).

Type of diet also significantly ($P < 0.01$) influenced hatchability of fertile eggs. The average for D(1) was 92.3 percent and for D(2) 58.1 percent. Although level of selenium addition did not significantly influence hatchability, it appeared that 0.1 ppm was the most desirable level to use. In all cases, when selenium was increased to 1.0 ppm there was a tendency to have slightly lower hatchability. Vitamin E level significantly affected egg hatchability. Fertile eggs

Table 37. Significant Main Effects and Interactions of Haugh Units (Experiment Three).

	Source	df	MS	F
P(1)	E X D	1/3	20.74	17.18*
	C X E X D	1/3	21.53	14.87*
	E X S	1/3	10.31	40.95**
	C X E X D X L	2/6	10.18	6.49*

Means (H.U.)

E1D1 = 88.6	C1E1D1L1 = 89.8	C2E2D1L1 = 87.8
E1D2 = 87.9	C1E1D1L2 = 88.8	C2E2D1L2 = 89.2
E2D1 = 89.1	C1E1D1L3 = 88.4	C2E2D1L3 = 87.6
E2D2 = 89.7	C1E1D2L1 = 91.6	C2F2D2L1 = 89.6
	C1E1D2L2 = 88.6	C2E2D2L2 = 88.4
E1S1 = 88.9	C1E1D2L3 = 88.4	C2E2D2L3 = 88.2
E1S2 = 88.9	C1E2D1L1 = 87.0	
E2S1 = 88.3	C1E2D1L2 = 87.8	
E2S2 = 89.2	C1E2D1L3 = 87.8	
	C1E2D2L1 = 91.6	
C1E1D1 = 89.0	C1E2D2L2 = 89.4	
C1E1D2 = 87.6	C1E2D2L3 = 91.2	
C1E2D1 = 89.5	C2E1D1L1 = 89.4	
C1E2D2 = 90.7	C2E1D1L2 = 88.0	
C2E1D1 = 88.2	C2E1D1L3 = 87.2	
C2E1D2 = 88.2	C2E1D2L1 = 87.5	
C2E2D1 = 88.8	C2E1D2L2 = 89.3	
C2E2D2 = 88.7	C2E1D2L3 = 89.4	

P(2)	Diet	1/3	164.83	20.96*
	E X D	1/3	12.86	12.78*
	C X E X D	1/3	11.75	10.32*
	Strain	1/3	21.27	10.51*
	E X S X L	2/6	24.84	7.13*

Table 37. Significant Main Effects and Interactions of Haugh Units (Experiment Three). (Con't.)

Means (H.U.)

D(1) = 86.5	C1E1D1 = 86.8	E1S1L1 = 87.1
D(2) = 88.4	C1E1D2 = 88.0	E1S1L2 = 86.8
	C1E2D1 = 85.7	E1S1L3 = 87.6
S(1) = 87.1	C1E2D2 = 88.9	E1S2L1 = 89.1
S(2) = 87.8	C2E1D1 = 87.1	E1S2L2 = 88.4
	C2E1D2 = 88.6	E1S2L3 = 86.7
E1D1 = 87.0	C2E2D1 = 86.7	E2S1L1 = 87.4
E1D2 = 88.3	C2E2D2 = 88.2	E2S1L2 = 87.0
E2D1 = 86.2		E2S1L3 = 87.1
E2D2 = 88.5		E2S2L1 = 87.2
		E2S2L2 = 87.0
		E2S2L3 = 88.6

P(3)	Diet	1/3	1951.38	179.77**
	Strain	1/3	63.37	15.11*
	E X S X L	2/6	35.70	10.54*

Means (H.U.)

D(1) = 83.0	E1S1L1 = 84.7	E2S1L1 = 85.7
D(2) = 89.3	E1S1L2 = 86.2	E2S1L2 = 85.3
	E1S1L3 = 86.8	E2S1L3 = 85.1
S(1) = 85.6	E1S2L1 = 87.7	E2S2L1 = 85.9
S(2) = 86.7	E1S2L2 = 87.4	E2S2L2 = 85.6
	E1S2L3 = 86.4	E2S2L3 = 87.7

P(4)	Diet	1/3	3232.43	2506.11**
	E X D	1/3	64.40	10.21*
	Strain	1/3	245.70	48.56**
	C X D X S X L	2/6	7.32	7.01*

Table 37. Significant Main Effects and Interactions of Haugh Units (Experiment Three). (Con't.)

Means (H.U.)

D(1) = 79.9	C1D1S1L1 = 80.6	C2D1S1L1 = 82.6
D(2) = 88.2	C1D1S1L2 = 78.2	C2D1S1L2 = 82.8
	C1D1S1L3 = 77.2	C2D1S1L3 = 84.0
S(1) = 82.9	C1D1S2L1 = 80.7	C2D1S2L1 = 84.6
S(2) = 85.2	C1D1S2L2 = 79.4	C2D1S2L2 = 83.2
	C1D1S2L3 = 80.6	C2D1S2L3 = 83.6
E1D1 = 80.7	C1D2S1L1 = 87.4	C2D2S1L1 = 85.5
E1D2 = 88.0	C1D2S1L2 = 87.3	C2D2S1L2 = 85.6
E2D1 = 79.2	C1D2S1L3 = 88.2	C2D2S1L3 = 86.4
E2D2 = 88.4	C1D2S2L1 = 90.5	C2D2S2L1 = 88.4
	C1D2S2L2 = 88.6	C2D2S2L2 = 90.6
	C1D2S2L3 = 90.8	C2D2S2L3 = 89.2

P(5)	Diet	1/3	3409.27	2130.34**
	Strain	1/3	336.41	121.56**
	D X E	1/3	32.75	13.06*

Means (H.U.)

D(1) = 77.9	D1E1 = 78.4
D(2) = 86.4	D1E2 = 77.3
	D2E1 = 86.1
S(1) = 80.8	D2E2 = 86.6
S(2) = 83.5	

P(6)	Diet	1/3	6348.96	10145.37**
	Strain	1/3	285.04	208.53**
	E X S X L	2/6	68.49	18.06**
	D X E	1/3	69.00	10.33*

Table 37. Significant Main Effects and Interactions of Haugh Units (Experiment Three). (Con't.)

			Means (H.U.)	
	D(1) = 75.3		E1S1L1 = 80.4	E2S1L1 = 79.7
	D(2) = 87.0		E1S1L2 = 78.4	E2S1L2 = 80.4
			E1S1L3 = 81.3	E2S1L3 = 79.3
	S(1) = 79.9		E1S2L1 = 84.1	E2S2L1 = 81.1
	S(2) = 82.4		E1S2L2 = 83.0	E2S2L2 = 81.9
			E1S2L3 = 81.0	E2S2L3 = 83.6
	D1E1 = 76.1			
	D1E2 = 74.5			
	D2E1 = 86.6			
	D2E2 = 87.4			
P(7)	Diet	1/3	1532.78	314.80**
	Strain	1/3	186.77	33.91*
	E X S X L	2/6	77.98	8.87*
			Means (H.U.)	
	D(1) = 77.4		E1S1L1 = 79.8	E2S1L1 = 77.9
	D(2) = 83.3		E1S1L2 = 78.9	E2S1L2 = 80.8
			E1S1L3 = 80.4	E2S1L3 = 78.1
	S(1) = 79.3		E1S2L1 = 82.8	E2S2L1 = 80.3
	S(2) = 81.4		E1S2L2 = 82.8	E2S2L2 = 80.9
			E1S2L3 = 79.5	E2S2L3 = 82.2

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

Table 38. Percent Fertility and Percent Hatchability of Egg From Hens Fed Various Levels of Selenium and Vitamin E (Experiment Three).

Corn-Soy						Glucose-Isolated Soy-Torula Yeast					
0 ppm Vitamin E ppm Se			10 ppm Vitamin E ppm Se			2 ppm Vitamin E ppm Se			10 ppm Vitamin E ppm Se		
0	0.1	1.0	0	0.1	1.0	0	0.1	1.0	0	0.1	1.0
					Fertility						
85.0[1]	84.9	83.6	85.4	89.2	89.1	44.1	46.3	34.6	28.6	39.0	42.0
89.5[2]	85.6	79.2	84.7	85.9	86.0	35.8	33.5	45.7	31.0	42.1	29.6
87.3[3]	85.3	81.6	85.0	87.6	87.6	38.8	37.3	41.7	30.1	40.8	33.7
					Hatchability of Fertile Eggs						
94.6[1]	95.2	91.4	90.3	96.3	91.3	75.6	65.8	72.2	36.7	68.8	45.9
90.7[2]	91.1	89.0	95.8	91.0	91.3	57.6	69.7	58.8	46.6	52.8	47.2
92.7[3]	93.1	90.4	93.0	93.8	91.3	65.0	68.3	62.8	43.2	59.2	46.7

[1]Values in this row represent fertility or hatchability of hatch one with 150-200 eggs per treatment.

[2]Values in this row represent fertility or hatchability of hatch two with 150-200 eggs per treatment.

[3]Values in this row represent average fertility or hatchability based on totals of the treatments for two hatches.

Table 39. Body Weight Gain of Three-Week-Old Progeny (Experiment Three).[1]

	Corn-Soy					
	0 ppm Vitamin E ppm Se			10 ppm Vitamin E ppm Se		
	0	0.1	1.0	0	0.1	1.0
Starting wt. (gm)	36	37	36	36	36	36
3-week gain (gm)[2]	113	91	109	109	105	109
3-week gain (gm)[3]	111	109	111	106	127	120
	Glucose-Isolated Soy-Torula Yeast					
	2 ppm Vitamin E ppm Se			10 ppm Vitamin E ppm Se		
	0	0.1	1.0	0	0.1	1.0
Starting wt. (gm)	33	33	34	33	33	32
3-week gain (gm)[2]	102	102	110	113	91	108
3-week gain (gm)[3]	117	115	110	108	100	110

[1]Chicks were fed the starter listed in Table 4. Heading on this table refer to dams diet treatment.

[2]Chick starter (Table 4) which contains 0.05 ppm Se.

[3]Chick starter (Table 4) plus 0.05 ppm Se for a 0.10 ppm total.

from hens fed the lower level of vitamin E averaged 79.3 percent and those from the higher vitamin E level 71.1 percent hatchability. There is no obvious reason for slightly increased levels of dietary vitamin E to reduce hatchability. The effect seemed particularly evident for eggs from hens on the purified diet treatments.

Progeny from one of the hatches were used in a three-week growth study (Table 39). To determine the possible influence of selenium

carry-over from the dam was an objective of this short term study. The treatments shown in Table 39 indicate the dam's dietary treatment. The chicks were fed a basal starter (Table 4) containing 0.05 ppm Se or the same diet with an additional 0.05 ppm Se added. Chicks hatched from dams fed the purified diets were slightly smaller at hatching. There were no significant effects of these diet variables on chick weight gain.

A slight trend for the additional selenium to increase weight gain of chicks from hens receiving the purified diet with the 2 ppm vitamin E was noted. It appeared that in order to demonstrate a growth response from selenium, a diet much lower in available selenium would be needed.

Table 40 lists egg selenium values obtained from analysis of eggs saved from hens at 32 weeks of age. At this time hens had received their dietary treatments for 12 weeks. Table 41 shows the significant main effects and interactions. Diets differed significantly ($P < 0.01$) in their effect on egg selenium. This was due to difference in natural selenium content (0.48 vs 0.05 ppm).

Strains differed as evidenced by the E X S interaction; however, the DeKalb 131 hens deposited slightly less selenium in their eggs than the Regional Controls fed either level of vitamin E. Level of selenium fed exerted significantly ($P < 0.01$) different effects on the amounts of selenium deposited in the egg. The increase in dietary selenium did not cause the same increase in egg selenium for both diets. Hens fed the corn-soy diets containing a liberal amount of natural selenium deposited only slightly more selenium in their eggs as levels were

Table 40. Effect of Dietary Selenium Additions on Egg Selenium (Experiment Three).[1]

Diet	Se Added (ppm)	Vitamin E Added (ppm)	Se Content of Eggs (ppm)	Standard Deviation
Corn-Soy[2]	---	---	0.42[4]	0.02
	---	10	0.43	0.01
	0.1	---	0.45	0.02
	0.1	10	0.45	0.03
	1.0	---	0.51	0.03
	1.0	10	0.54	0.03
Glucose-Isolated Soy-Torula Yeast[3]	---	2	0.11	0.01
	---	10	0.11	0.02
	0.1	2	0.19	0.03
	0.1	10	0.17	0.02
	1.0	2	0.41	0.03
	1.0	10	0.37	0.03

[1]Hens received diets for 12 weeks before eggs were sampled.

[2]Diet contains 0.48 ppm selenium.

[3]Diet contains 0.05 ppm selenium.

[4]Each value represents the average of eight experimental units (3 hens each). Pooled samples of five eggs were used for each analysis.

increased. However, those hens fed the purified diets containing little natural selenium deposited a greater portion of the dietary addition in their eggs. Hens fed the highest level (1.0 ppm Se) in purified diets did not deposit as much selenium in their eggs as was found in eggs from the unsupplemented corn-soy diets. This suggests that inorganic selenium additions to a diet containing fairly high levels of natural dietary selenium will not cause excessive levels of selenium do be

Table 41: Significant Main Effects and Interaction of Egg Selenium Content at 32 Weeks (Experiment Three).

Source	df	MS	F
Diet	1/3	1293.63	4937.53**
E X S	1/3	1.33	10.49*
Level (Se)	2/6	347.81	723.05**
D X L	2/6	87.53	196.97**

Means (ppm)

D(1) = 0.466
D(2) = 0.233

E1S1 = 0.357
E1S2 = 0.338
E2S1 = 0.354
E2S2 = 0.349

L(1) = 0.267
L(2) = 0.315
L(3) = 0.467

D1L1 = 0.423
D1L2 = 0.451
D1L3 = 0.524
D2L1 = 0.111
D2L2 = 0.180
D2L3 = 0.410

*Significantly different at ($P < 0.05$).

**Significantly different at ($P < 0.01$).

deposited in eggs. From a practical view, if inorganic Se additions were made to a low Se diet it would increase egg selenium. If a similar addition was made to a diet already containing sufficient Se, little increase should be expected in the selenium content of the eggs.

GENERAL DISCUSSION

Three experiments were conducted to study effects of various dietary additions of selenium and arsenic on chickens. Additions were made to corn-soy and purified diets. When a single protein source (Torula yeast or isolated soy protein) was fed, growth was slower resulting in mature body weights which were less than those obtained with the corn-soy diet. Improved production was obtained when both Torula yeast and isolated soy protein were used together in a diet having slightly higher energy and protein. This indicates that perhaps the proper amino balance was not available using the single protein source. Hens should have adjusted their feed intake so that lack of energy should not have caused the poor production.

Adequate vitamin E, antioxidants and sulfur amino acids as well as all other known nutrients necessary for poultry nutrition were included in all diets in order to be able to attribute any response to the selenium and arsenic additions. When several diets were prepared they were blended before the selenium additions were made in order to alleviate differences due to variation in the selenium content of ingredients.

In one experiment the use of isolated soybean protein resulted in significantly ($P < 0.01$) thinner egg shell than was obtained with hens fed the corn-soy diet. This type of protein requires a more critical balance among calcium, phosphorus and vitamin D_3, and perhaps nutrients in this diet provided here were not adequate to meet the exact requirement.

Further study with this type of diet would allow one to determine the optimum calcium and phosphorus levels to use; although performance with this type of diet has not been satisfactory in work reported by others (Thapar et al., 1969).

Excessively high mortality occurred with purified diets. This was most evident when a glucose-isolated soy diet was fed. Thrush (Candidiasis) was the cause of nearly all of these deaths. A mold inhibiting drug, Nystatin, and improved management practices in feeding helped alleviate this problem. If feed remaining in the troughs became moist, an ideal environment for mold growth developed. However, if this feed was periodically removed, and hens fed more nearly what they would consume daily, the problem became less severe.

Two ppm represents a level about 10 times that suggested to be the selenium requirement (Thompson and Scott, 1969). Eight ppm Se is an upper marginal level becuase it borders on toxicity. The one experiment where lower levels (0.1 and 1.0 ppm) of selenium were added there was no effect on egg size or feed consumption, as was observed in all cases where 2 ppm was used in that size and intake were reduced. Lower feed consumption was also noted by others (Poley et al., 1941; Thapar, 1964) when 2 ppm Se was included in laying hen diets.

Selenium additions of 2 ppm or less produced varied responses. Mortality was lowest in groups fed 2 ppm Se in both the growing and laying periods. This was true in all cases except one in which 8 ppm Se resulted in slightly lower mortality during the laying period when the

Torula yeast diet was fed. When the lower additions (0.1 and 1.0 ppm) were used, there was no significant effect on mortality due to level of selenium. In general, 8 ppm resulted in the highest mortality, but this toxic effect was overcome by arsenic. Effects of selenium and arsenic in mortality agree well with results of Thapar et al. (1969), however on the present study the beneficial effect of 2 ppm was even more obvious.

Chick growth was improved slightly at younger ages (8 weeks or less) when 2 ppm Se was included in the diets. There was no advantage for growth from selenium added to feed for pullets as they matured. Mature hens fed additional selenium weighed less throughout the laying period. This may have been due to higher production or to the reduced feed intake which was noted when either 2 ppm or 8 ppm selenium were added. The body weight depression due to 8 ppm Se was completely overcome by including 15 ppm arsenic in the corn-soy diet and 8 ppm arsenic in the purified diets. Previous work at this laboratory had shown arsenic to be most effective in practical and purified diets at 15 and 8 ppm, respectively.

The addition of 2 ppm selenium to a corn-soy diet containing 0.45 ppm Se resulted in higher hen-day egg production. The effect was significant during the first production cycle but of less magnitude during the second. Perhaps younger animals respond to selenium supplements, but older ones who have sufficient Se within their bodies do not respond as readily or require additional supplements or those that required selenium had already succumbed. Egg production was slightly improved by adding

2 ppm Se to the glucose-isolated soy diet. Work by Thapar et al. (1969) did not show any beneficial effect from a 2 ppm Se addition to a similar diet. In the present study, no improvement in production was noted when similar additions were made to the glucose-Torula yeast diet. Performance was quite poor with these purified diets so perhaps information obtained would have been more meaningful had more normal production been attained. The purified diets were rather low in selenium so responses should have been detected if performance had been more acceptable.

Lower selenium additions (0.1, 0.2, and 1.0 ppm) did not significantly affect hen-day egg production. In several cases it appeared that the basal corn-soy diet gave superior performance, indicating there was no advantage to add selenium to a diet containing 0.5 ppm. Although no significant differences were detected, low level selenium additions to a purified diet may have been helpful, but the results were not consistant.

The high level of 8 ppm Se lowered hen-day egg production and reduced egg size in all cases. The reduction was not significant except with the glucose-Torula yeast diet. Arsenic included with the 8 ppm Se increased egg production in the corn-soy diets but had little effect in this regard in purified diets. Results of the present study agree in general with those of Thapar et al. (1969) where 8 ppm Se significantly lowered egg production and arsenic effectively overcame this toxicity.

Addition of 2 ppm Se to a corn-soy diet resulted in production of smaller eggs in all cases. In one of two experiments this was a

significant reduction. Adding 1 ppm of selenium to corn-soy diets resulted in a slightly smaller egg being produced. It seems that for optimum egg size the total selenium content of the diet should not exceed 1-2 ppm. No reduction in egg size was observed when 2 ppm Se was added to purified diets which contained only about 0.07 ppm Se. Eight ppm added selenium reduced egg size to even a greater extent than did 2 ppm in most cases. Thapar (1964) reported a significant decrease in egg weight with 8 ppm Se but no effect on egg size when 2 ppm was added to the corn-soy diet.

The mechanism involved with the egg size depression is unknown. Perhaps selenium at excessive levels acts as a competive inhibitor in certain enzyme systems. Selenium is known to become concentrated in liver and other organs associated with protein synthesis (McFarland et al., 1970). Perhaps excess selenium interfers with egg formation, but one should study total protein output to measure this. Egg size is more often associated with lipids, that is, inadequate linoleic does not allow for optimum egg size. Perhaps excessive selenium interfers with the proper metabolism involving the formation of lipid materials in this egg.

When 2 ppm Se was added to the corn-soy diet slightly thinner egg shells resulted. The decrease was not significant, but was observed in both studies where measured. Eight ppm did not cause a further decrease in shell thickness nor were any adverse effects noted with selenium additions to purified diets. Perhaps the thinner shell produced by hens

fed the corn-soy diets with 2 ppm added selenium was a result of more eggs being produced by those groups and not due to any toxicity of selenium.

Interior egg quality measured in Haugh Units was unaffected by adding 2 ppm selenium to any of the diets tested. In some instances higher selenium additions resulted in higher interior quality but probably these differences were due to fewer eggs being produced in these treatments. Treatments containing the selenium-arsenic combination frequently had eggs with the highest Haugh Unit values. There is no apparent explanation for this. Hens fed purified diets produced eggs having significantly higher Haugh Unit values than from those fed the corn-soy diets. This was probably because of the reduced numbers produced. The DeKalb 131 strain consistently produced eggs with higher interior quality.

Fertility of eggs was not influenced by dietary selenium additions of 2 ppm or less. When 8 ppm selenium was added fertility appeared to be lower but the differences were not significant. Thapar (1964) found that hens receiving dietary additions of 8 ppm had higher egg fertility than observed with eggs from hens fed unsupplemented diets. Poley et al. (1937) found fertility unaffected even when 15 ppm Se was provided in hen diets by seleniferous grains. There is no apparent explanation for these conflicting results. One should be cautious when considering egg fertility data because hens which are laying poorly do not become fertile as readily as good layers. More problems are encountered in the

techniques of artificial insemination with poor layers such as would be found among hens on the 8 ppm Se treatments. Unless unhatched eggs are examined carefully, errors can also occur in distinquishing fertile from non-fertile eggs.

Hatchability can readily be observed because either chicks hatch or they do not, therefore few errors can be made when determining this criteria. In all cases when 8 ppm Se was added, hatchability of fertile eggs was significantly lowered. This was true with both the corn-soy and purified diets. The toxic effect of 8 ppm Se was most noticeable when the glucose-isolated soy diet was used. Thapar et al. (1969) did not observe such a toxic effect on hatchability when a similar isolated soy diet was fed. Without question hatchability of fertile eggs was the most sensitive measure of selenium toxicity. Arsenic partially protected against this toxicity as evidenced by the higher hatchability in treatments containing arsenic along with the high level of selenium. Lower levels of selenium had no significant effect on hatchability. No reason can be given for the lower hatchability observed with the glucose-isolated soy-Torula yeast diet compared to that when only one protein was used in the purified diets.

Values observed for liver selenium content were quite similar to those of Thapar et al. (1969). Arsenic reduced liver selenium in the corn-soy diets but not in the purified type diets. There is no apparent explanation for this difference. Various tissues sampled for Se analysis from 64-week-old hens showed increasing amounts of Se as dietary levels were increased although the efficiency of selenium deposition was much

reduced with the higher Se levels. Thigh and breast muscle did not contain as much Se as was found in the kidney and liver and furthermore these edible tissues were less influenced by dietary Se. Feathers contained high levels of Se in comparison to the other tissues. This might be due to the replacement of sulfur with Se in the sulfur amino acids or more likely the selenium is stored in feathers in some other manner. The effects of arsenic were not consistent among the tissues studied.

Similarly, dietary additions of selenium increased egg selenium, however, only a small portion of dietary Se was deposited in the eggs. Assuming a hen eats 100 gm of feed per day containing 2 ppm Se, she consumes 0.2 mg of selenium daily. If 40 gm of egg is produced per hen per day, in which there is 0.56 ppm Se, there would be only 0.02 mg of Se deposited daily or about 1/10 of the amount ingested. Similar calculations for a hen consuming feed containing 8 ppm Se indicate that about 6 percent of the amount ingested was deposited in the egg. Calculations made for egg selenium from the basal diets compared to that for the supplemented diets indicate there was much less selenium deposited in eggs due to the dietary additions. This may be because the natural selenium is in the organic form and highly available, whereas the added inorganic form is less potent.

In nearly all cases, arsenic lowered the amount of selenium in the eggs. Vitamin E had no effect. There was no increase in egg Se with length of time on treatments. The results indicated that when Se was

added to a diet containing fairly adequate levels of selenium there was only a slight increase in egg selenium, whereas when selenium was added to a low selenium diet, the small increase in egg selenium was more readily detected. Eggs from hens fed purified diets with 0.2 or 2 ppm added Se contained amounts of selenium similar to those from the corn-soy unsupplemented diet. This further shows that Se added as sodium selenite is not deposited in edible products as readily as that which occurs in the natural form in feeds.

SUMMARY

Dietary selenium additions from 0.1 ppm to 8.0 ppm were made to several types of diets for layer-type hens in three experiments. In one experiment a significant ($P < 0.01$) increase in hen-day egg production from 2 ppm Se was observed from hens fed a corn-soy diet. In all studies the lower levels (0.1, 0.2, 2.0 ppm Se) were not detrimental to egg production with any of the various types of diets used. Eight ppm Se reduced egg production in two experiments and the toxic effect was counteracted by arsenic additions.

The purified diets fed to laying hens consistently resulted in significantly lower ($P < 0.01$) hen-day egg production than that obtained with the corn-soy diet. Cage size or vitamin E level did not affect hen-day egg production. Poor performance was obtained when a glucose-isolated soy or glucose-Torula yeast diet was fed. However, when a purified diet composed of glucose, isolated soy protein and Torula yeast was fed, greatly improved performance resulted.

Either 2 ppm or 8 ppm Se significantly decreased egg size when added to the corn-soy diet. When the glucose-isolated soy diet was fed neither 2 ppm nor 8 ppm selenium resulted in a significant decrease in egg size. Addition of 8 ppm Se or the 8 ppm Se plus 8 ppm As combination resulted in significantly smaller ($P < 0.01$) egg size with the glucose-Torula yeast diet. The lower selenium additions (0.1 and 1.0 ppm) used in Experiment Three did not decrease egg size significantly.

However, in most cases eggs from hens fed the 1 ppm Se were slightly smaller. Eggs produced by hens fed the purified diets were significantly smaller in all experiments. DeKalb 131 hens produced significantly larger eggs than did the Regional Control strain.

Selenium supplementation had no significant effect on body weight gain. When 2 ppm Se was fed chicks gained slightly faster initially but were no heavier than those fed basal diets by 20 weeks of age. Chicks hatched from hens fed purified diets showed no significant response to selenium supplements to the diet of the dams; although, they grew faster with selenium additions to their diet. Hens fed 2 ppm Se were somewhat lighter than those fed the basal diets throughout both Experiments One and Two.

Mortality was lowered slightly by feeding 2 ppm Se during the growing period as well as the laying period. Lower mortality was observed in laying periods of both Experiment One and Two when 2 ppm Se was added. Lower selenium levels used in Experiment Three had no significant effect on mortality. Throughout all experiments more hens died when the purified diets were fed. Slightly more death loss occurred among hens on the 8 ppm Se and the Se-As combination treatments. When one considers the laying periods of both Experiment One and Two (the total period from 24-104 weeks), mortality of hens fed the 2 ppm Se was half that of the basal treatments. There were no significant mortality differences due to level of selenium or vitamin E in Experiment Three.

Fertility was reduced when 8 ppm Se were added to the corn-soy and glucose-Torula yeast diet of Experiment One. Arsenic completely restored percent fertility to that of the basal treatments. A reduction of fertility occurred with either 2 ppm or 8 ppm Se in the corn-soy diets fed in Experiment Two. Arsenic only partially alleviated this depression. No effect on fertility occurred with the lower selenium supplements.

No adverse effect on hatchability of fertile eggs was noted with selenium additions of 2 ppm or lower. Eight ppm Se produced a dramatic toxic effect on embryos evidenced by their death during late stages of development. Arsenic partially overcame this effect.

As dietary additions of selenium were increased the levels of selenium in tissues and eggs increased. However, at higher intake levels there was a lower proportion of the dietary intake deposited in the eggs and tissues. Liver, kidney and especially feathers were highest in selenium. Thigh and breast muscle contained relatively low levels. In most instances, the arsenic lowered tissue deposition of selenium.

When purified diets were supplemented with selenium at 0.1, 0.2, 1.0 or even 2.0 ppm the selenium found in eggs was not greater than that observed in eggs laid by hens fed the unsupplemented corn-soy diets.

Selenium at a nutritional level (0.1 ppm) added to a layer diet containing fair amounts of natural selenium should have little effect on the amount deposited in the eggs.

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APPENDIX

Appendix Table 1. Interrelationships of Selenium, Vitamin E, Antioxidants, Sulfur Amino Acids and Polyunsaturated Fatty Acids.[4]

Disease	Animal	Tissue	Influence by PUFA	Prevented by Vit. E	Prevented by Se	Prevented by Anti-Oxid.	Prevented by Sulfur A.A.
I. Reproductive failure							
Embryonic degeneration							
Type A	Female rat, hen turkey	Vascular system of embryo	X	X		X	
Type B	Cow, ewe			1	X[2]		
Sterility	Male rat, guinea pig, hamster, dog, cock	Male gonads		X			
II. Liver, blood, brain, capillaries, pancreas							
Liver necrosis	Rat, pig	Liver		X	X		
Fibrosis	Chick, mouse	Pancreas			X		
Erythrocyte hemolysis	Rat, chick, man (premature infant)	Erythrocytes	X	X		X	
Plasma protein loss	Chick, turkey	Serum albumen		X	X		
Anemia	Monkey	Bone marrow		X		X	
Encephalo-malacia	Chick	Cerebellum	X	X		X	
Exudative diathesis	Chick, turkey	Vascular system		X	X		
Kidney degeneration	Rat, mouse monkey, mink	Kidney tubular epithelium	X	X	X		
Steatitis (ceriod)	Mink, pig, chick	Adipose tissue	X	X		X	
Depigmentation	Rat	Incisors	X	X		X	

Appendix Table 1. Interrelationships of Selenium, Vitamin E, Antioxidants, Sulfur Amino Acids and Polyunsaturated Fatty Acids.[4]

Disease	Animal	Tissue	Influence by PUFA	Prevented by			
				Vit. E	Se	Anti-Oxid.	Sulfur A.A.
III. Nutritional myopathies							
Type A (Nutritional muscular dystrophy)	Rabbit, guinea pig, monkey, duck, mouse, mink	Skeletal muscle		X		?	
Type B (White muscle disease)	Lamb, calf, kid	Skeletal and heart muscles		1	X[2]		
Type C	Turkey	Gizzard, heart		1	X		
Type D	Chicken	Skeletal muscle[3]		X			X

[1]Not effective in diets severely deficient in selenium.

[2]When added to diets containing low levels of vitamin E.

[3]Low level (0.5%) of linoleic acid necessary to produce dystrophy; higher levels did not increase vitamin E required for prevention.

[4]From Scott, (1969) and Feed stuffs of May 9, 1970.

Appendix Table 2. Calculated Nutrients of Practical Type Diets Used.

Nutrient	Practical Diets		
	Starter	Grower	Layer
Protein, %	21.6	14.0	16.0
M. E., kcal/kg	2876.0	2372.0	3045.0
C. Fat, %	2.95	4.31	5.84
C. Fiber, %	3.85	9.86	3.0
Salt, %		0.65	0.55
Ca, %	1.05	1.65	2.5
P(Total), %	0.89	1.03	0.67
P(Avail.), %		0.82	0.48
Na, %		0.26	0.31
K, %		0.53	0.63
Mg, %		0.19	0.14
Mn, ppm		62.3	36.5
Zn, ppm		53.9	43.4
Fe, ppm		128.8	71.0
Cu, ppm		11.61	8.64
Co, ppm		0.96	0.84
I, ppm		0.76	0.62
Arg, %		0.87	1.09
Lys, %		0.62	0.81
Met, %	0.38	0.21	0.29
Met + Cys, %	0.69	0.44	0.54
Trp, %		0.17	0.21
Gly, %		0.66	0.73
His, %		0.28	0.38
Leu, %		0.99	1.52
Ile, %		0.60	0.77
Phe, %		0.63	0.81
Phe + Tyr, %		1.20	1.42
Thr, %		0.49	0.66
Val, %		0.74	0.82

Appendix Table 3. Calculated Nutrients of Glucose-Isolated Soy Protein Diets Used in Experiment One.

Nutrient	Glucose-Isolated Soy[1]		
	Starter	Grower	Layer
Protein, %	21.6	14.0	16.0
M. E., kcal/kg	3075.0	3060.0	2955.0
C. Fat, %			2.05
C. Fiber, %			5.04
Ca, %			2.59
P(Total), %			0.28
P(Avail.), %			0.19
Arg, %			1.12
Lys, %			0.94
Met, %	0.56	0.48	0.39
Met + Cys, %	0.70	0.58	0.62
Tvp, %			.12
Gly, %			0.66
His, %			0.39
Leu, %			1.32
Ile, %			0.75
Phe, %			0.87
Phe + Tyr, %			1.49
Thr, %			0.55
Val, %			0.76

[1]Nutrients provided by vitamin and salt mixes are not included in this table.

Appendix Table 4. Calculated Nutrients of Glucose-Torula Yeast Diets Used in Experiment One.

Nutrient	Glucose-Torula Yeast[1]		
	Starter	Grower	Layer
Protein, %	21.6	14.0	16.0
M. E., kcal/kg	2570.0	2735.0	2580.0
C. Fat, %			2.0
C. Fiber, %			5.12
Ca, %		0.94	2.63
P(Total), %		0.37	0.73
P(Avail.), %			0.18
Arg, %			0.85
Lys, %			1.33
Met, %	0.44	0.46	0.42
Met + Cys, %	0.69	0.57	0.57
Trp, %			0.22
Gly, %			0.69
His, %			0.34
Leu, %			1.26
Ile, %			1.00
Phe, %			0.80
Phe + Tyr, %			1.48
Thr, %			0.80
Val, %			0.88

[1]Nutrients provided by vitamin and salt mixes are not included in this table.

Appendix Table 5. Calculated Nutrient Content of Glucose-Isolated Soy-Torula Yeast Diets.

Nutrient	Glucose-Isolated Soy-Torula Yeast[1]	
	Starter	Layer
Protein, %	22.0	17.2
M. E., kcal/kg	2770.0	2800.0
C. Fat, %	2.04	3.02
C. Fiber, %	5.11	5.09
Ca, %	0.97	2.72
P(Total), %	0.83	0.73
P(Avail.), %	0.37	0.37
Arg, %	1.40	1.08
Lys, %	1.62	1.26
Met, %	0.50	0.43
Met + Cys, %	0.77	0.64
Trp, %	0.24	0.19
Gly, %	1.14	0.73
His, %	0.52	0.41
Leu, %	1.82	1.42
Ile, %	1.24	0.97
Phe, %	1.19	0.92
Phe + Tyr, %	2.10	1.64
Thr, %	0.96	0.75
Val, %	1.16	0.91

[1]Nutrients provided by vitamin and salt mixes are not included in this table.

significant reduction. Adding 1 ppm of selenium to corn-soy diets resulted in a slightly smaller egg being produced. It seems that for optimum egg size the total selenium content of the diet should not exceed 1-2 ppm. No reduction in egg size was observed when 2 ppm Se was added to purified diets which contained only about 0.07 ppm Se. Eight ppm added selenium reduced egg size to even a greater extent than did 2 ppm in most cases. Thapar (1964) reported a significant decrease in egg weight with 8 ppm Se but no effect on egg size when 2 ppm was added to the corn-soy diet.

The mechanism involved with the egg size depression is unknown. Perhaps selenium at excessive levels acts as a competive inhibitor in certain enzyme systems. Selenium is known to become concentrated in liver and other organs associated with protein synthesis (McFarland et al., 1970). Perhaps excess selenium interfers with egg formation, but one should study total protein output to measure this. Egg size is more often associated with lipids, that is, inadequate linoleic does not allow for optimum egg size. Perhaps excessive selenium interfers with the proper metabolism involving the formation of lipid materials in this egg.

When 2 ppm Se was added to the corn-soy diet slightly thinner egg shells resulted. The decrease was not significant, but was observed in both studies where measured. Eight ppm did not cause a further decrease in shell thickness nor were any adverse effects noted with selenium additions to purified diets. Perhaps the thinner shell produced by hens