Effect of Dietary Selenium Level on Feed Intake and Weight Gain of Rats

R. J. Vetter, W. V. Kessler, M. P. Plumlee, and R. B. Harrington
Departments of Bionucleonics and Animal Sciences
Purdue University, West Lafayette, Indiana 47907

Introduction

The first evidence that selenium was required in the diet was reported by Schwartz and Folz (11), who demonstrated that selenium was a component of Factor 3, a nutritional substance which prevented necrotic liver degeneration in rats (10). Since then, selenium has been shown to be essential for several species (6,8,14). The National Academy of Sciences Subcommittee on Selenium (15) emhasized the importance of alleviating selenium deficiency in domestic animals. Feedstuffs made from grains grown in geographical areas low in soil selenium must be supplemented with selenium to prevent severe deficiencies (12,13). But the amount of selenium added to the diet must be carefully controlled to prevent detection by the animal (2,3) and development of toxicity symptoms. This study was conducted to provide additional data on the effect of various dietary levels of sodium selenite on palatability, growth rate, and tissue selenium levels of weanling rats.

Materials and Methods

Animals and Diets

Male rats which were Sprague Dawley descendants[2] weighing about 100 g were used in this investigation which was completed in a series of three experimental trials. Each trial contained a 3-day pre-experimental adjustment period in which the rats were housed individually in metal cages with raised wire floors. The rats were handled daily for gentling during this adjustment period and were maintained on a diet consisting of a commercial ration and distilled water supplied *ad libitum*. After this adjustment period each rat was weighed to the nearest gram, replaced in the individual cages, and randomly assigned a treatment number.

Diets for Trials I and II were prepared by dissolving sodium selenite in distilled water containing ^{75}Se as sodium selenite and mixing this solution with a commercial ration[3]. The final mixture contained the desired level of selenium and 0.36 μCi of ^{75}Se per 100 g of diet. Each diet was determined to be mixed homogeneously by analyzing several aliquots for ^{75}Se and comparing the results expressed as counts per minute per gram of feed. The diets for Trial III were prepared as above except that no ^{75}Se was added. The commercial diet was analyzed colorimetrically (9) and was found to contain 0.120 ± 0.005 ppm of selenium.

[2]Laboratory Supply Co., Inc. Indianapolis, Indiana.
[3]Allied Mills, Inc., Chicago, Illinois.

Radioactivity Analysis

A physical half-life determination and a differential gamma ray spectrum were obtained by counting an aliquot of the stock ^{75}Se solution. The results indicated no radionuclide impurities when compared to a reference spectrum and physical half-life (4). Ascending paper chromatography employing two different solvent systems (1,5,7) showed that the chemical purity of the selenite used in this investigation was greater than 99% and that the radiochemical purity of the ^{75}Se selenite was greater than 95%. Experimental animals and tissue samples were analyzed for ^{75}Se with a scintillation detection system containing a large thallium activated sodium iodide well crystal located in a low background shield. The crystal used was 25.4 cm in diameter and 28 cm long with a well 10.2 cm in diameter and 20.3 cm deep. The detection system was calibrated to differentially count the ^{75}Se gammas in an energy range from 265 to 402 KeV.

Trial I

The purpose of this trial was to determine if rats could detect high levels of selenium in their diet and to determine the tissue levels of selenium. Sixty rats were randomly divided into six groups with 0, 4, 8, 12, 16, or 20 ppm selenium added to the diet. Small aluminum feeders designed to minimize spillage were tared and weighed after being filled with the prepared diet. Feeders were weighed every 2 days, refilled, and weighed again. Rats were weighed every 2 days and on the sacrifice day. Distilled water was supplied *ad libitum*.

At 4-day intervals two rats were taken from each group, counted for whole body ^{75}Se and sacrificed by decapitation and exsanguination. Liver, kidneys, blood, and gut were removed from each rat and analyzed for ^{75}Se.

Trial II

This trial was designed to more accurately determine the highest level of selenium which would not affect feed intake or weight gain. The experimental design was identical to that in Trial I except that the levels used were 0, 1, 2, 3, 4, or 5 ppm selenium added to the diet and rats were sacrificed at 7-day intervals.

Trial III

The third trial was designed to allow the rat to choose between four levels of dietary selenium. Latin square design was used to rotate the feeders in such a way as to prevent the animal from guessing which feeder contained his favorite level and to eliminate bias due to location preference. Selenium was added to the diet at levels of 0, 2, 4, or 6 ppm. The trial contained 32 rats randomly assigned to cages and each cage contained all four levels. Distilled water was allowed *ad libitum*. The four feeders were filled and weighed every fourth day for 16 days.

Calculation of Data

All parameters indicated in the figures are expressed as arithmetic means. Analysis of the data in Trials I and II consisted of analysis of variance with the sum of squares broken into orthogonal polynomial components for the main effects and all their first order interactions. Correlation coefficients were computed between all variables. In Trial III, all data were analyzed by analysis of variance, and correlation coefficients were computed between periodic weight gain and total selenium intake, periodic weight gain and total amount of feed

consumed, cumulative weight gain and total selenium intake, and cumulative weight gain and total amount of feed consumed.

Results and Discussion

Trial I

The results presented in Figure 1 indicate that when the diet was supplemented with 0 or 4 ppm of selenium the rats ate nearly the same amount of feed and gained nearly the same weight. But when the diet was supplemented with a level of 8 ppm of selenium or greater, feed intake and weight gain decreased considerably. Tissue level of selenium increased with dietary level as shown in Figure 2, and there was a significant ($P < .05$) positive correlation. Tissue level of selenium was highest in the kidney, then liver, blood, and whole body. As dietary level increased, kidney level increased at a rate greater than the rates in other tissues. There was a significant ($P < .05$) negative correlation between feed intake and tissue level of selenium and between weight gain and tissue level of selenium. This resulted from a decrease in tissue level of selenium with time which may have been due to the increased ability of the animal to metabolize selenium as the animal matured. This increased ability may have been due to an enzyme induction process. There was a significant positive correlation ($P < .05$) between whole body level and blood level, liver level, kidney level, or gut content selenium. Therefore, in future studies of this type involving large animals, whole body level or blood level of ^{75}Se might be helpful in determining if selenium affected the parameter of interest.

A statistical analysis indicated a significant difference ($P < .01$) between levels and times for all variables except liver level of selenium which showed a significant difference in levels only, and there was a significant interaction.

Trial II

This trial was conducted with lower levels and over a longer period of time in an attempt to better define the highest level of selenium which could be added to the diet without affecting feed intake or weight gain. Figure 1 shows that after 4 weeks rats fed diets supplemented with 4 or 5 ppm of selenium were eating less feed and gaining less weight than those fed diets supplemented with 3 ppm of selenium or less. Rats fed diets supplemented with 0, 1, 2, or 3 ppm selenium ate similar amounts of feed and gained nearly the same weight. The control rats sacrificed on day 35 appear to have converted feed to body weight at an efficiency somewhat less than those sacrificed earlier. This may be due to consistent spillage of small amounts of feed by one of the rats. Tissue distribution of selenium was similar to that in Trial I, but the levels were much lower as shown in Figure 2. As in Trial I, there was a significant ($P < .05$) positive correlation between dietary level and tissue level of selenium and a significant ($P < .05$) negative correlation between feed intake and tissue level and between weight gain and tissue level.

Trial III

The design of this trial allowed the rats equal access to four diets, but eliminated bias due to location preference and previous location of the control

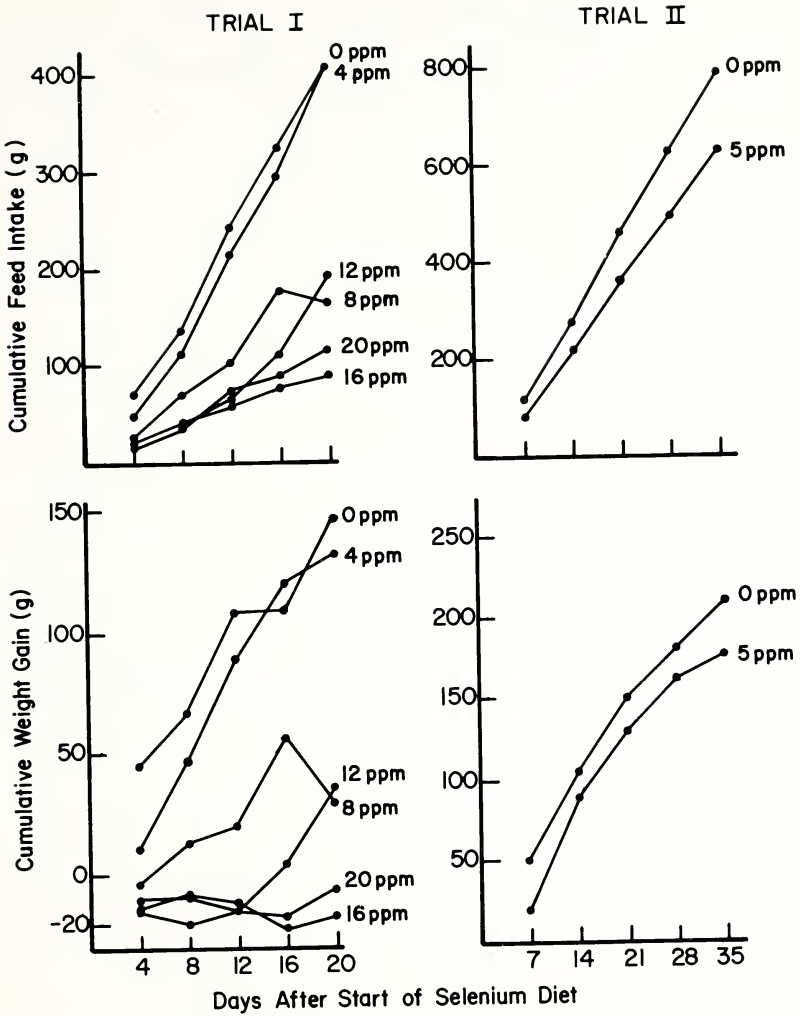

FIGURE 1. *Cumulative feed intake and weight gain of rats fed various levels of selenium. (In Trial II, 1-4 ppm are omitted. If plotted, they would lie between 0 and 5 ppm.)*

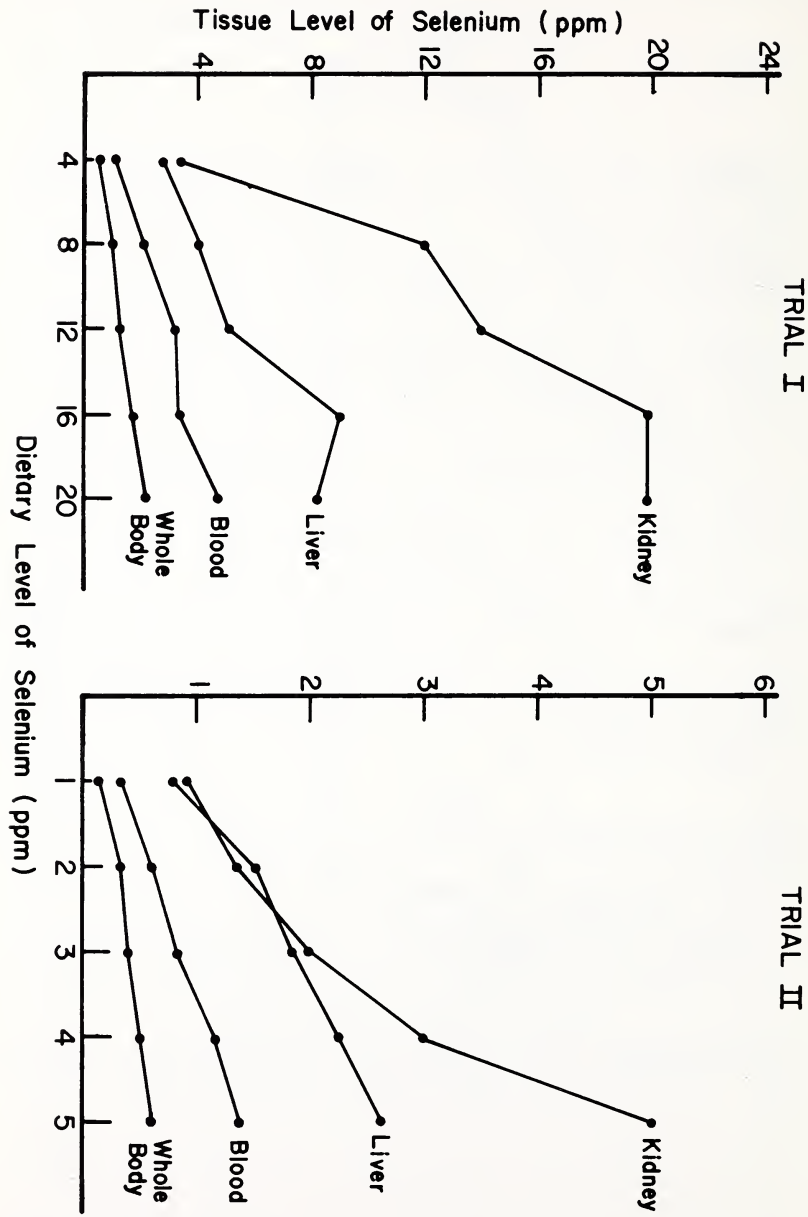

FIGURE 2. *Tissue level of selenium as influenced by dietary level. (Each point represents average tissue level of selenium for all rats on the dietary level of selenium indicated).*

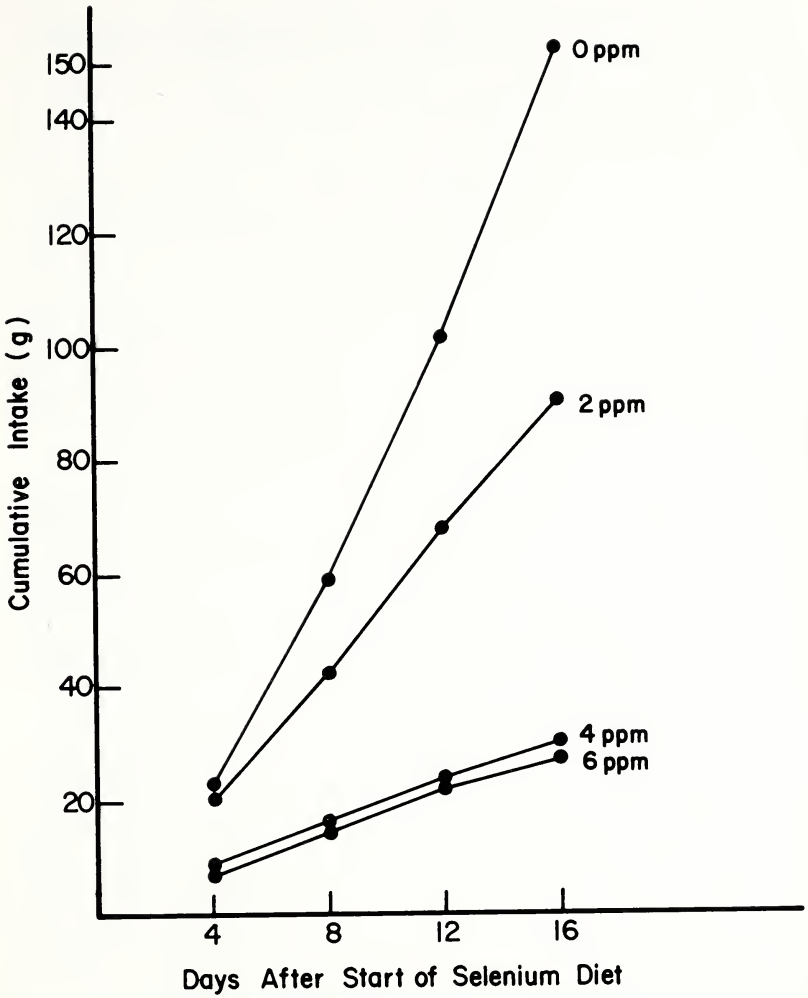

FIGURE 3. *Feed intake by rats offered a choice between diets containing 0, 2, 4 and 6 ppm of selenium.*

diet. Figure 3 shows that more of the control diet was eaten than any diet supplemented with selenium. Data on individual rats showed that some preferred the diet supplemented with 2 ppm of selenium but most preferred the control diet. A few rats ate approximately the same amount of all the diets. It is clearly evident, however, that most of the rats chose either the control diet or the diet supplemented with 2 ppm selenium. This suggests that rats can detect selenium in the diet at levels of 2 ppm or greater. Since a small amount of all levels was eaten by each rat, it appears that the rats were tasting the food to determine which one they liked the best. Correlation coefficients showed no relationship between feed intake or weight gain and the amount of selenium consumed by the rat.

The results of these experiments support the conclusions of Franke and Potter (3). The rat can sense large amounts (2 ppm or greater) of selenium in its diet and would either starve or choose a lower selenium diet if faced with a choice. Dietary selenium additions of 3 ppm or less to a commerical rat chow did not affect feed intake or growth rate in rats.

Literature Cited

1. BURSTALL, F. H., G. R. DAVIES, R. P. LINSTEAD, and R. A. WELLS. 1950. Inorganic chromatography on cellulose. II. The separation and detection of metals and acid radicals on strips of absorbent paper. J. Chem. Soc. **1950**:516-528.
2. FRANKE, K. W., and E. P. PAINTER. 1938. A study of the toxicity and selenium content of seleniferous diets: with statistical consideration. Cereal Chem. **15**:1-24.
3. FRANKE, K. W., and V. R. POTTER. 1936. A new toxicant occurring naturally in certain samples of plant foodstuffs. XIII. The ability of rats to discriminate between diets of varying degrees of toxicity. Science **88**:330-332.
4. HEATH, R. L. 1964. Scintillation Spectrometry Gamma-Ray Spectrum Catalogue, 2nd ed., U.S. Atomic Energy Commission, Idaho Operations Office, IDO-16880-2.
5. LEDERER, M. 1955. Paper chromatography of inorganic ions. X. A study of rhenium, technetium and some other non-metals. Anal. Chim. Acta. **12**:146-150.
6. MUTH, O. H., J. E. OLDFIELD, L. F. REMMERT and J. R. SCHUBERT. 1958. Effects of selenium and vitamin E on white muscle disease. Science **128**:1090.
7. PLUCHET, E. and M. LEDERER. 1960. Adsorption paper chromatography of inorganic anions in acetate buffers. J. Chrom. **3**:290-296.
8. RAHMAN, M. M., C. W. DEYOE, R. E. DAVIES and J. R. COUCH. 1960. Selenium and exudative diathesis in chicks and poults. J. Nutr. **72**:71-76.
9. ROSENFIELD, I. and O. A. BEATH. 1964. Selenium Geobotany, Biochemistry, Toxicity, and Nutrition. Academic Press, New York. 411 p.
10. SCHWARZ, K. 1951. Production of dietary necrotic liver degeneration using American Torula yeast. Proc. Soc. Exp. Biol. Med. **77**:818-823.
11. SCHWARZ, K. and C. M. FOLTZ. 1957. Selenium as an integral part of Factor 3 against dietary necrotic liver degeneration. J. Am. Chem. Soc. **79**:3292-3293.
12. SCOTT, M. L. 1973. The selenium dilemma. J. Nutr. **103**:803-810.
13. SCOTT, M. L., G. OLSON, L. KROOK, and W. R. BROWN. 1967. Selenium-responsive myopathies of myocardium and of smooth muscle in the young poult. J. Nutr. **91**:573-583.
14. SHARMAN, G. A. M., K. L. BLAXTER and R. S. WILSON. 1959. Prevention of enzootic muscular dystrophy by selenium administration. Vet. Record **71**:536.
15. SUBCOMMITTEE ON SELENIUM. 1971. Selenium in Nutrition. National Academy of Sciences. 79 p.