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Blood Selenium and Glutathione Peroxidase Levels and Dietary Selenium of Free-Living and Institutionalized Elderly Subjects[1] (41614)

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Abstract. The purpose of this study was to evaluate the selenium status of healthy free-living and institutionalized elderly people. For the 36 free-living elderly dietary selenium intake averaged 94 ± 44 μg Se/day and a positive correlation coefficient was found between dietary selenium and dietary calories ($r = 0.46$; $P < 0.05$), dietary protein ($r = 0.60$; $P < 0.01$), and dietary fat ($r = 0.43$; $P < 0.05$). Diet histories from the institutionalized subjects revealed a strong correlation coefficient between selenium and carbohydrate ($r = 0.51$; $P < 0.005$) and selenium and calories ($r = 0.44$; $P < 0.05$). Mean erythrocyte and plasma selenium levels for the free-living subjects were 0.20 ± 0.06 μg/ml and 0.10 ± 0.03 μg/ml, respectively, while mean erythrocyte glutathione peroxidase (GSH-Px) activity was 27.5 ± 5.0 units/g protein. For the free-living subjects positive correlation was found between dietary selenium and erythrocyte selenium levels ($r = 0.38$; $P < 0.05$) but no correlation existed between dietary selenium and plasma selenium ($r = 0.13$; $P > 0.05$) and RBC GSH-Px ($r = -0.15$; $P > 0.05$). The dietary selenium levels and blood selenium and GSH-Px levels were above the levels found in populations proposed to be at risk for selenium deficiency. Thus, these elderly appear to have adequate selenium status.

Nutritional status of the elderly has received wide attention, but studies of selenium needs of this population have not been extensively investigated (1). If the elderly have reduced caloric consumption in compliance with the RDA recommendation (2), an inadequate intake of selenium may result, especially if nutrient density is low. A positive correlation between the calories consumed and the selenium intake for twenty-two subjects, 14 to 64 years, has been demonstrated (3). Thus the elderly, who have caloric intakes below the levels consumed by younger populations may be consuming less selenium than the adequate and safe dietary level of 50–200 μg Se/day recommended by the National Research Council (2).

The only known mammalian function for selenium is as an essential component for glutathione peroxidase (GSH-Px); however, other selenoproteins may exist in rat liver and sheep and rat semen (4, 5). A positive correlation between blood GSH-Px activities and blood selenium levels was found in healthy New Zealand subjects (6) and U. S. university students and staff (7). The New Zealand population consumed between 30 and 50 μg Se/day while the U.S. population had Se dietary levels ranging from 50 to 200 μg Se/day (6, 7). The only documented naturally occurring human selenium deficiency disease is a cardiomyopathy called Keshan disease found in Chinese children who consume less than 30 μg Se/day (8). Keshan disease was prevented with dietary selenium supplementation (8). In addition, it has been reported that one patient receiving intravenous hyperalimentation (IVH) had low blood Se levels and a congestive cardiomyopathy (9) and one New Zealand IVH patient had muscle pain which was relieved with selenium supplementation (10, 11). Selenium has also been shown to decrease the incidence of various forms of cancer in rodents treated with carcinogens (12). Although selenium toxicity is not a major problem in humans, animals from seleniferous areas exhibited the well documented selenium toxicity symptoms of alkali disease (13).

The present study was undertaken to eval-

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uate the selenium nutritional status in a free-living elderly population by measurement of their dietary selenium intakes, erythrocyte and plasma selenium levels, and erythrocyte GSH-Px activity. Diets of the elderly were also evaluated for nutritional adequacy by estimating the calories from protein, fat, and carbohydrate and the level of the micronutrients vitamins C and A. In order to evaluate the significance of protein and energy consumption on dietary selenium levels, correlations between dietary selenium and protein and energy levels were calculated. Erythrocyte and plasma selenium levels and erythrocyte GSH-Px activities, all suggested measures of selenium status, were compared to dietary selenium intake to determine if there was a relationship between dietary selenium and the biochemical measures of selenium status. Because the diets of institutionalized elderly can be measured more accurately and are standardized, the selenium levels of the diets of 8 institutionalized healthy elderly females were compared to the 36 free-living elderly.

Materials and Methods. *Subject description and dietary records. Subjects: free living.* Thirty-six subjects (31% male, 69% female ranging in age from 58–80 years (Table I) were participants in a Title III C congregate feeding site or were members of a retired workers organization. The subjects signed an informed consent approved by the University of Texas Health Sciences Committee for the protection of human subjects. Medical histories were completed and evaluated by a physician in order to determine whether the subjects were healthy. The mean age was 70.6 years, and the mean hematocrit, hemoglobin, and blood pressure (Table I) values were within normal ranges (14). Fifty-four percent of the subjects were overweight for their heights (2), while 24% of the subjects smoked more than one cigarette per day and 40% consumed one or more alcoholic drinks per day.

A dietitian interviewed each subject to obtain a 24-hr diet history of a typical day which was usually the previous 24 hr. For the 24-hr diet history the subjects were asked to report everything they ate or drank within the time period. A food frequency tally was used to validate the 24-hr diet history especially for frequently forgotten food items such as condiments, drinks, and bread.

Subjects: institutionalized. Eight subjects (100% female), ages 65 to 92 (Table I) who were residents at one privately owned nursing home for 5–17 months, were selected for this diet analysis because they were consuming a regular diet and had no known nutritional problems. On four consecutive Fridays, the total day's food intake for each subject was weighed. The institution was on a cycle menu so each Friday was a different menu. Plate waste was weighed and was subtracted from the quantity of food offered. Hematocrits and hemoglobins were determined on four of the eight subjects (Table I).

Calculations of nutrient content of daily diets. One-day recall records for the free-living population and the 4-day records of food consumption for the institutionalized subjects were evaluated for calories, protein, fat, vitamins A and C (15), and selenium. The dietary selenium was estimated from published values (16–19) or by analysis. Selenium concentrations have been estimated in many foods (16–19), but there appears to be variability in selenium concentrations of foods due to the location where the food was grown or raised and due to processing. Furthermore, there were several foods consumed by our subjects, that had not been analyzed for selenium. Thus, we determined the selenium levels of 62 foods (50% of all foods listed in diet histories) that had no reported value or foods whose selenium levels may vary because of processing or geographical area. The foods analyzed from this study were compared to levels reported by others in order to document some of the variability in food selenium levels (20).

Our dietary selenium levels were calculated using literature values (16–19) and values of locally purchased foods analyzed in our laboratory. Welsh and co-workers (3) found a good correlation ($r = 0.88$) between diets analyzed for selenium and calculated for selenium with the analyzed diets having slightly lower levels of selenium than the calculated diets. Therefore, calculating dietary selenium should provide a reasonable estimate of actual selenium intake (3).

Blood preparation and analysis. A vena puncture was performed on all free-living elderly subjects with a syringe and needle. Immediately, hematocrits and hemoglobins were determined on a portion of the whole blood.

TABLE I. DESCRIPTIONS OF THE FREE-LIVING AND INSTITUTIONALIZED SUBJECTS

Descriptors	Free-living[a]			Institutionalized[b,c,d]		
	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range
Age (years)	70.6	5.26	58–80	76	9.0	64–92
Hematocrit (%)	45	6.0	31–62	37	6.9	28–42
Hemoglobin (g/dl)	14.3	1.8	9.5–19.2	12.1	2.0	9.5–13.5
Blood pressure						
Diastolic	78.1	14.55	50–125	—	—	—
Systolic	135.9	24.40	102–180	—	—	—

[a] Thirty-six subjects.
[b] Eight subjects.
[c] Hematocrit and hemoglobin measured on four subjects only.
[d] Blood pressure data not available.

The remaining blood was maintained at 4°C, treated with heparin, centrifuged, and the plasma removed and frozen. The cellular fraction was rinsed twice with saline to remove the buffy coat (21) and the cells were hemolized with cold deionized water.

Selenium levels were determined in the following manner. The hemolized cells were digested with concentrated nitric, perchloric, and hydrochloric acids (22, 23). After completion of digestion, 2 ml of 0.04 *M* EDTA was added to the digestate. The pH of the solution was adjusted to 1.0 with 7 *M* NH_4OH and made up to a constant volume (50 ml) with 0.1 *M* HCl. Each solution was incubated at 60°C for 20 min with 0.1% 2,3-diaminonaphthalene (prepared daily), cooled, and extracted with 10 ml of reagent-grade cyclohexane. The fluorescence of each extract was measured at 366 nm excitation and 606 nm emission with a spectrofluorometer[3] (experimentally determined wavelengths for maximum excitation and emission intensity). The instrument was calibrated before each run by preparing a standard curve using extracts from solutions containing known amounts of selenium. This method of calibration was validated periodically by the method of standard addition (24) and all chemicals, containers, and water were routinely checked for selenium contamination. All reported selenium values in this study were duplicated with a standard deviation of 0.0026. Results from NBS liver standard (1.1 ppm ± 0.05) were 1.0 ± 0.02 ppm.

A fraction of the hemolized red blood cells was removed and diluted with 0.02 *M* potassium phosphate buffer, pH 7.0, for determination of glutathione peroxidase activity. The glutathione peroxidase activity was determined by measuring the oxidation of NADPH by cumene peroxide via glutathione reductase (25). The reaction mixture had the following composition: dialyzed glutathione reductase in 0.005 *M* phosphate buffer, 0.1 ml; 0.003 *M* EDTA, 0.02 *M* phosphate buffer, pH 7.00, 0.5 ml; 0.01 m*M* $NADPH_2$, 0.1 ml; 40 m*M* glutathione, pH 5.0, 0.05 ml; 0.10 m*M* sodium azide, 0.10 ml; water plus diluted buffered hemolized blood, 0.1 ml. After a 5-min preincubation at room temperature, 10 m*M* peroxide, 0.01 ml, was added. The change in absorbance with time was measured spectrophotometrically at 340 nm. Blanks (solution containing all reagents but the sample) were measured before each set of unknowns. A matched set of optical glass cuvettes were used. The protein in each sample was determined by the method developed by Lowry (26). Enzyme activity was expressed as micromoles NADPH oxidized per minute per gram of protein. Statistics: Pearson's correlations and the Student *t* test were used.

Results. The daily intake of energy, protein, fat, carbohydrates, and selected micronutrients from the free-living and institutionalized subjects' diets are listed in Table II. No statistical difference between the two groups was found in any of the measured dietary parameters. The percentage of calories derived from fat ranged from 17 to 51%, whereas the percentage of calories from protein ranged from 6 to 59%. When protein was calculated

[3] Varian SF-330.

TABLE II. THE LEVEL OF SELECTED NUTRIENTS IN SUBJECT'S DIETS

Nutrients	Free living[a]			Institutionalized[b]		
	$\bar{X}$	SD	Range	$\bar{X}$	SD	Range
Calories (kcal)	1721	555	872–3455	1507	444	1024–2461
Protein (g)	72.9	30.6	29.8–193.2	78.0	34.0	49–161.0
Protein/body weight (g/kg)	1.07	0.50	0.35–3.09	1.40	0.48	0.88–2.44
Fat (g)	67.2	33.1	16.6–273.1	67.0	31.0	39.4–123.1
Carbohydrates (g)	191.7	78.1	27.7–459.8	146.9	16.0	94.7–172.3
% kcal protein	18.9	8.4	6.0–59.0	21.0	3.0	18–26
% kcal fat	35.7	11.2	16.5–51.4	40.0	4.0	43–45
% kcal carbohydrates	45.4	11.2	8.1–67.3	39.0	6.0	28–50
Vitamin A (IU)	8647	10,538	928–61,439	4436	2556	1460–9523
Vitamin C (mg)	183	258	12–1389	93	83	37–288
Selenium (μg)	94.2	43.9	35.9–187.2	107.7	41.7	84.7–200.6

[a] Thirty-six subjects.
[b] Eight subjects.

in terms of grams protein per kilograms body weight for each subject, the mean levels for free-living and institutionalized subjects were 1.07 and 1.40, respectively.

TABLE III. CORRELATIONS BETWEEN DIETARY SELENIUM AND BLOOD SELENIUM MEASURES AND GSH-PX ACTIVITY

Measures	Correlation	
	r	P
Free-living[a]		
Diet		
Calories (kcal/day)/Se (μg/day)	0.46	< 0.05
Protein (g/day)/Se (μg/day)	0.60	< 0.01
Carbohydrate (g/day)/Se (μg/day)	−0.19	> 0.05
Fat (g/day)/Se (μg/day)	0.43	< 0.01
Plasma		
Dietary Se (μg/day)/plasma Se (μg/g)	0.13	< 0.05
Erythrocyte		
Dietary Se (μg/day)/erythrocyte Se (μg/ml)	0.38	> 0.05
Dietary Se (μg/day)/erythrocyte GSH-Px (units[b])	−0.15	< 0.05
Institutionalized[c]		
Diet		
Calories (kcal/day)/Se (μg/day)	0.44	< 0.05
Protein (g/day)/Se (μg/day)	0.36	< 0.05
Carbohydrate (g/day)/Se (μg/day)	0.51	< 0.005
Fat (g/day)/Se (μg/day)	0.42	< 0.005

[a] Thirty-six subjects.
[b] Units expressed as μmole NADPH oxidized/min/g protein.
[c] Eight subjects.

The selenium intake for all subjects ranged from 36 to 201 μg/day. The mean values of dietary selenium for free-living and institutionalized subjects were 94 ± 44 and 108 ± 42 μg Se/day, respectively. Although not significantly different, the free-living females tended to consume higher levels of selenium (96 μg Se/day) than the free-living males (90.3 μg Se/day). For the free-living subjects, 50% of the dietary selenium was contributed by animal and animal products, but when evaluated for each subject, no correlation existed between the percentage of protein provided by animal sources and dietary selenium. Seven of the free-living and none of the institutionalized subjects had selenium values below 50 μg/day which is the level postulated as a minimum adequate daily intake (2). For the free-living subjects, the correlation coefficient (Table III) between daily caloric and selenium intake was 0.46 ($P < 0.05$) (Fig. 1), and the correlation coefficient for daily protein and selenium intake was 0.60 ($P < 0.01$) (Fig. 2). Also, the selenium–fat correlation coefficient was 0.43 ($P < 0.01$) but the correlation between selenium and carbohydrate was −0.19 ($P > 0.05$). For the free-living subjects the following correlation coefficients were found: calories and protein, $r = 0.69$, $P < 0.01$; calories and fat, $r = 0.53$, $P < 0.01$; calories and carbohydrate, $r = 0.78$, $P < 0.01$; calories and vitamin A, $r = -0.02$, $P > 0.05$; and calories and vitamin C, $r = 0.10$, $P > 0.05$. The correlation coefficient between the institutionalized subjects daily caloric intake and selenium intake was

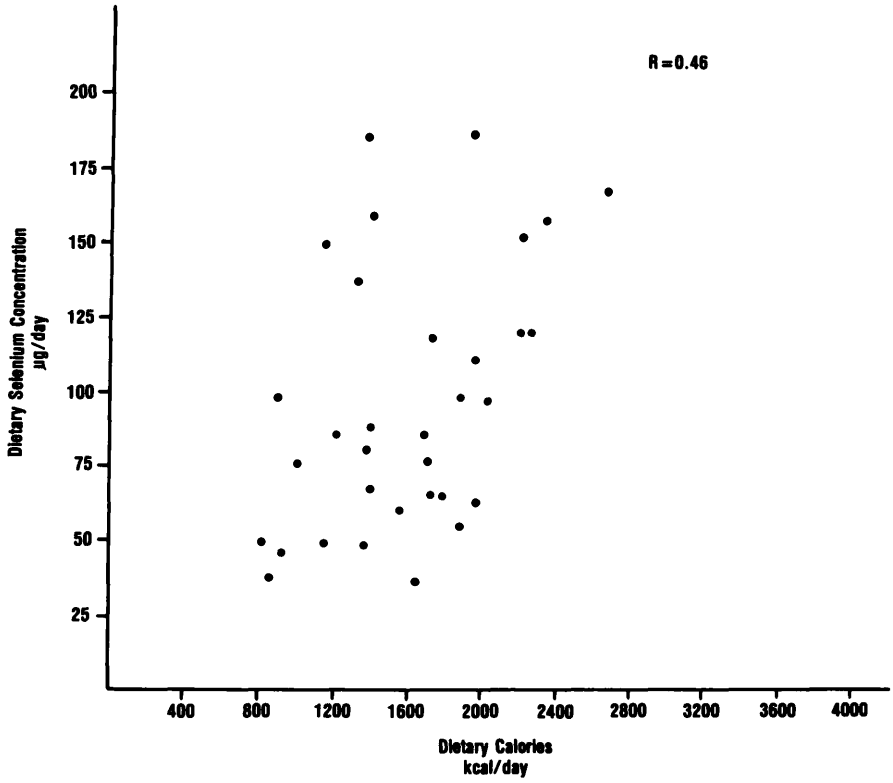

FIG. 1. Correlation between dietary selenium levels and dietary calories in the free-living elderly subjects.

0.44 ($P < 0.05$) and the following correlation coefficients were found: protein and selenium, $r = 0.36$, $P < 0.05$; carbohydrate and selenium, $r = 0.51$, $P < 0.005$; fat and selenium, $r = 0.42$, $P < 0.005$.

The selenium levels in erythrocytes and plasma and GSH-Px activities in erythrocytes of the free-living elderly are shown in Table IV. The mean erythrocyte selenium concentration was 0.20 µg Se/ml, while the plasma selenium concentration was 0.10 µg/g and the erythrocyte GSH-Px activity was 28 units/g of protein. The correlation coefficient (Table III) between dietary intake of selenium and the erythrocyte selenium concentration was 0.38 ($P < 0.05$) (Fig. 3), but the correlation between dietary selenium intake and plasma selenium concentration was 0.13 ($P > 0.05$) (Fig. 4). A negative low correlation (−0.15) was found between dietary selenium and erythrocyte GSH-Px activity. The mean values of erythrocyte selenium concentration and GSH-Px activity (Table IV) were higher for females than males ($P < 0.001$) but sex had no effect on plasma selenium levels. No correlation between erythrocyte selenium level and erythrocyte GSH-Px activity was found ($r = -0.13$, $P > 0.05$).

Discussion. *Dietary measures.* The average intake of the free-living subjects met all the recommended dietary allowances evaluated (2) for the nutrients listed in Table II, but several of the individual free-living subjects did have intakes of vitamin C and protein below 1/3 of the RDA value. The mean caloric levels for both the free-living and institutionalized people were below the suggested RDA values and these results agree with those of others (27, 28). Despite errors in estimating food consumption, O'Hanlon and Kohrs (27) suggest that many older people may consume inadequate levels of calories and this was reflected in the present study. The institutionalized subjects had minimal to generous intakes of the energy contributing nutrients and vitamins C and A. No statistically significant differences existed between the measured nutrient intakes of the free-living and institu-

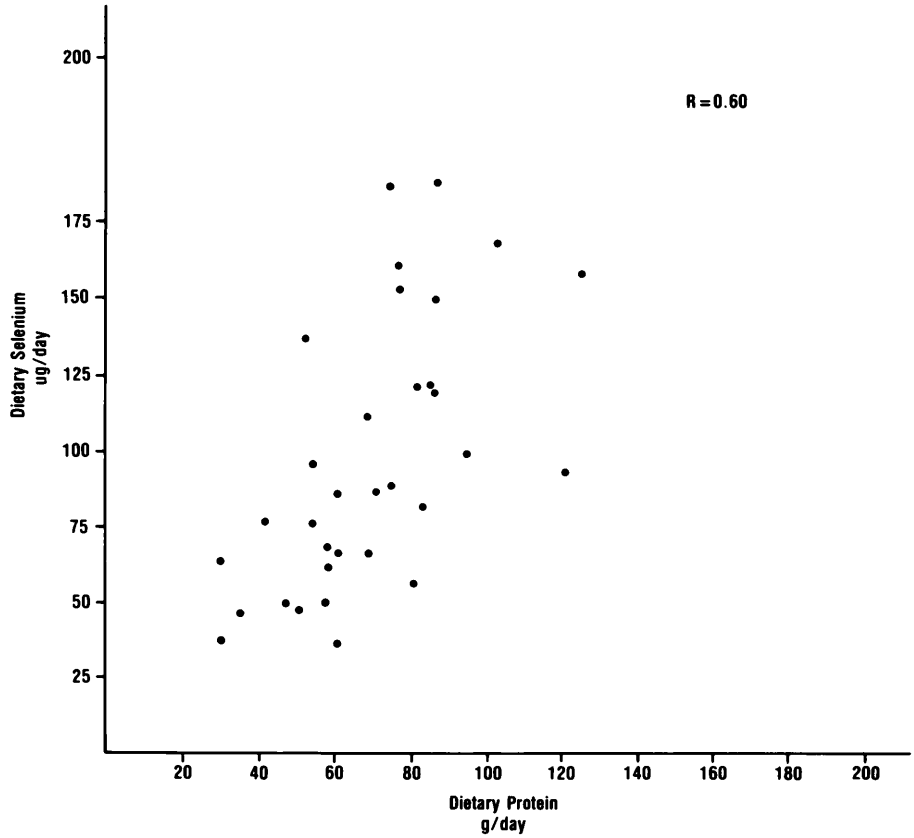

FIG. 2. Correlation between dietary selenium levels and dietary protein in the free-living elderly subjects.

tionalized subjects, but the free-living subjects had a greater range of values for each nutrient than the institutionalized subjects. This is due to both the sample size (36 versus 8 subjects) and the greater variety of foods consumed by the free-living subjects.

TABLE IV. SELENIUM LEVELS IN ERYTHROCYTES AND PLASMA AND GSH-PX ACTIVITIES IN ERYTHROCYTES OF FREE-LIVING ELDERLY[a]

Measures	$\bar{X}$	SD	Range
Erythrocytes Se (μg/ml)	0.20	0.06	0.09–0.32
Males	0.17[b]	0.04	
Females	0.20	0.06	
Plasma Se (μg/ml)	0.10	0.03	0.06–0.13
Males	0.09	0.02	
Females	0.10	0.02	
Erythrocyte GSH-Px activity (units[c]/g of protein)	27.5	5.0	19.8–35.1
Males	25.9[b]	4.0	
Females	29.0	3.6	

[a] Thirty-six subjects.
[b] $P < 0.001$ as measured by Student's t test.
[c] Units are expressed as NADPH oxidized/min.

In general, the elderly consumed adequate amounts of selenium. All the institutionalized and 80% of the free-living subjects met the recommended selenium intake of 50–200 μg Se/day, but 19% (seven) of the free-living subjects consumed between 30 and 50 μg Se/day. These dietary selenium levels were similar to those of the New Zealand population (6, 29–31) yet greater than those levels reported with the only known selenium responsive disease, Keshan disease (8).

A positive correlation was found between dietary selenium, caloric, carbohydrate, and protein intake for both the free-living and institutionalized subjects (Table III, Figs. 1 and 2). Welsh and co-workers (3) found that selenium–calorie correlation was stronger than any other correlations between dietary selenium and any other dietary nutrients. However, for our free-living subjects, there was a stronger correlation between dietary selenium

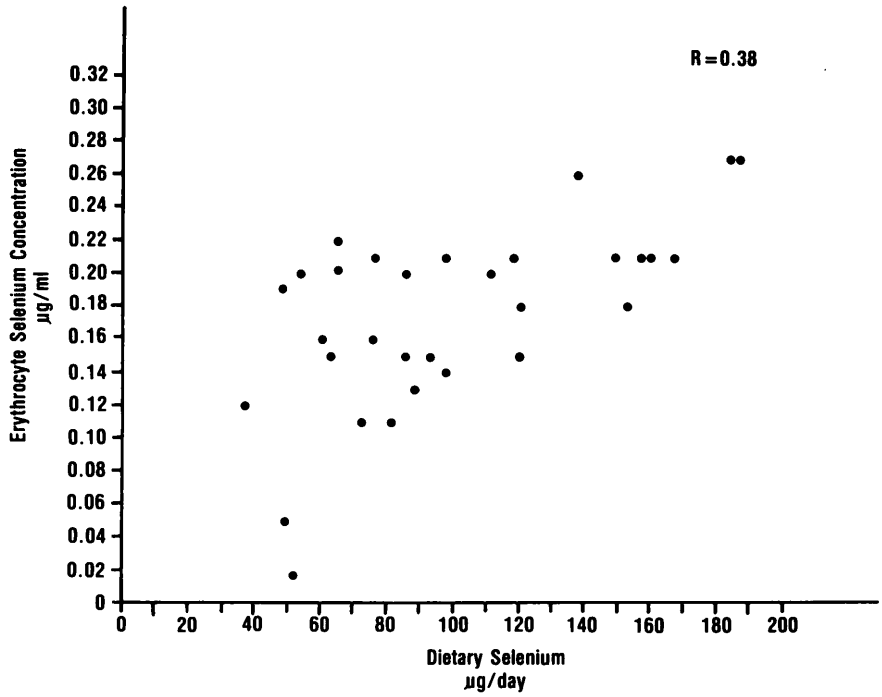

FIG. 3. Correlation between erythrocyte selenium levels and dietary selenium in the free-living elderly subjects.

and protein than between dietary selenium and calories whereas for the institutionalized subjects the strongest correlation was between selenium and carbohydrate intake.

Fifty percent of the protein consumed by the free-living subjects was from animal sources; but poor correlations existed between the subject's source of protein and their dietary selenium level. A more indepth study tracing selenium intake to origin of protein is

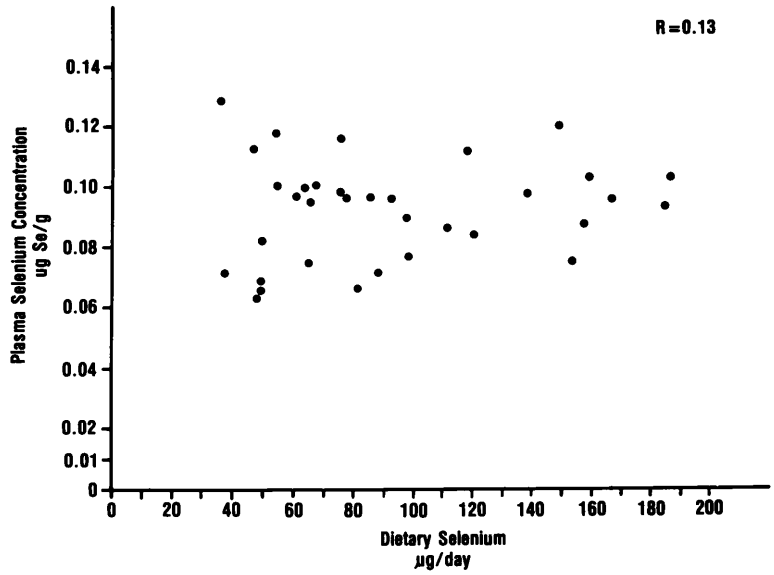

FIG. 4. Correlation between plasma selenium levels and dietary selenium in the free-living elderly subjects.

necessary in order to further evaluate the relationship of protein and selenium intake.

The combination of the results from these two elderly populations indicate that dietary selenium increases as total calories as well as protein and carbohydrate increased. The conflicting correlations for selenium and fat suggest that dietary sources of fat will affect selenium levels. For example, the fat accompanying poultry and poultry products would contribute selenium whereas the fat accompanying high-fat meats such as sausage would not contribute selenium (20). These strong correlations are probably due to the fact that many foods contribute to the dietary selenium levels. Furthermore, if a U.S. diet contains a variety of foods and meets the caloric and protein needs, it probably will meet the selenium needs.

Biochemical measures. Our elderly subjects appear to have adequate selenium nutritional status. The selenium levels in plasma and erythrocytes and GSH-Px activity in erythrocytes (Table IV) are similar to levels found by others (7, 32–34) and were twice those found in the New Zealand population (29–31).

Dietary selenium levels correlated with erythrocyte selenium levels. It has been postulated that erythrocyte selenium levels may be a predictor of long term selenium status (35) as evidenced by studies with patients receiving a selenium-deficient intravenous hyperalimentation (IVH). Although the dietary selenium was calculated from a 24-hr diet history, food frequency was also considered in order to obtain a representative diet. These data suggest that the dietary selenium levels found in this elderly population predicted their usual selenium intake.

In this elderly population, the females consumed slightly more selenium than the males and this was reflected in a significantly higher erythrocyte selenium level and GSH-Px activity (Table IV). There was no difference in hematocrits or hemoglobin levels between the sexes. There was no statistical effect of sex on plasma selenium levels and this was consistent with previous studies from our laboratory (7) and others (29) with younger subjects. At present, there is no other explanation for the sex difference in erythrocyte selenium levels and GSH-Px activity than that the females consumed more selenium than the males. Since the male group was small, care should be made concerning any conclusions about these reported differences.

Plasma selenium levels did not correlate with dietary selenium for these subjects. This may be explained by possible daily fluctuations of plasma selenium and shifts in metabolism. There is at present a lack of understanding of the effect of recent selenium consumption on plasma selenium levels in individuals with adequate selenium intake. These questions warrant further study.

With these elderly subjects there was no increase in erythrocyte GSH-Px activity with increasing levels of dietary selenium or erythrocyte selenium concentrations and this parallels the results from the New Zealand subjects (30, 31). Thus, the erythrocyte GSH-Px activity was an insensitive indicator of dietary selenium when dietary selenium was between 50 and 200 μg Se/day. Hence, two postulates can be made. First, if GSH-Px is the only function for selenium in humans, then dietary selenium above the suggested range (50–200 μg Se/day) is not needed because higher levels would not cause any biochemical changes. However, as suggested by the fact that other selenoproteins with unknown functions have been found in other species, selenium may be part of other biochemical processes. So the measurement of GSH-Px activity may not indicate the upper requirement for selenium. Indeed, research with rodents has demonstrated that higher levels of dietary selenium than required for maximum GSH-Px activity reduces the incidence of colon (36) and mammary (37) cancer. Thus, investigation of other selenoproteins is necessary for a more definitive functional measure of selenium status.

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