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Dietary Intake and Urinary Excretion of Magnesium in Approximately 100 Preadolescent Black and White Girls

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To the Graduate Council:

I am submitting herewith a thesis written by Helen Graham Huber entitled "Dietary Intake and Urinary Excretion of Magnesium in Approximately 100 Preadolescent Black and White Girls." I have examined the final electronic copy of this thesis for form and content and recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Nutrition.

Gail W. Disney, Major Professor

We have read this thesis and recommend its acceptance:

Rossie L. Mason, Ada Marie Campbell

Accepted for the Council:
Carolyn R. Hodges

Vice Provost and Dean of the Graduate School

(Original signatures are on file with official student records.)

To the Graduate Council:

I am submitting herewith a thesis written by Helen Graham Huber entitled "Dietary Intake and Urinary Excretion of Magnesium in Approximately 100 Preadolescent Black and White Girls." I recommend that it be accepted in partial fulfillment of the requirements for the degree of Master of Science, with a major in Nutrition.

Gail W. Disney
Dr. Gail W. Disney
Major Professor

We have read this thesis and recommend its acceptance:

Bessie L. Mason

Ada Marie Campbell

Accepted for the Council:

L Evan Pond
Vice Chancellor
Graduate Studies and Research

DIETARY INTAKE AND URINARY EXCRETION OF MAGNESIUM IN APPROXIMATELY 100 PREADOLESCENT BLACK AND WHITE GIRLS

A Thesis

Presented for the

Master of Science

Degree

The University of Tennessee, Knoxville

Helen Graham Huber

June 1979

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ABSTRACT

This study was designed to accomplish two objectives. The first objective was to determine the relationship between the dietary intake of magnesium and urinary excretion of magnesium:creatinine in first void urine samples from preadolescent girls. The other objective was to evaluate the effects of age, race and income level on the dietary intake and urinary excretion of magnesium in a first void urine sample.

Dietary intakes and urinary magnesium:creatinine ratios were evaluated during a 2 year longitudinal period in approximately 100 preadolescent girls who were 9±0.5 years of age at the beginning of the study. These girls were participants in the S-87 Southern Regional Nutrition Project. The racial distribution for the girls was 50 percent white and 50 percent black. Fifty percent of the girls were from upper income families ($2,000 or more/person/year) and 50 percent were from lower income families ($1,000 or less/ person/year).

Magnesium in the first void urine sample was determined by atomic absorption spectrophotometry. Urinary creatinine was determined by Folin's Method. Mean dietary intakes and urinary magnesium:creatinine ratios were calculated. Due to the small size of the sampling distribution for each age

group, it was not possible to calculate the regression of dietary intakes of magnesium and urinary excretion of magnesium:creatinine for each race and income level on age. The Student's t-test was used for testing the differences between the 2 races and between the 2 income groups with regard to mean dietary magnesium intakes and the urinary excretion of magnesium, creatinine and magnesium:creatinine. Pearson correlation coefficients between dietary and urinary variables were determined.

The sample size for each age group was not large enough to make inferences to the population; therefore, the data for each age group were combined by race and income level. Whites had a significantly greater ($P < 0.05$) mean dietary magnesium intake than did blacks, but there was not a significant difference between income levels. The greatest percentage of subjects met 67 to 100 percent of the RDA for magnesium. Correlations among total dietary protein, calcium, phosphorus, and magnesium were highly significant ($P < 0.01$). Blacks had a significantly greater ($P < 0.01$) excretion of creatinine than did whites. There were no specific trends in excretory patterns of magnesium by race and income groups.

Because statistical analyses did not show a relationship between the dietary intake of magnesium and urinary excretion of magnesium:creatinine, it was not possible to develop a standard based on creatinine from a first void urine sample.

The development of such a standard would prove very useful in a large field study where 24-hour urine collections are difficult.

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CHAPTER I

INTRODUCTION

Because preadolescent females are in a critical stage in relation to nutritional health, there is a need for nutritional information pertaining to this age group. Very little information exists regarding the magnesium requirement of preadolescent girls. Owing to the lack of available data concerning magnesium metabolism of preadolescent girls, presently the RDA is based on estimations derived from studies with adults or younger children (1). Greger et al. (2) studied mineral metabolism in 14 girls, aged 12.5 to 14.5 years, consuming ordinary foods that were characteristic of their usual dietary habits. The authors suggested that since the girls were in negative magnesium balance on these dietary regimes, much more research is needed to understand the nutritional requirements of the preadolescent.

Minimal requirements have been most often determined by metabolic balance studies which by their precise nature limit the number of subjects which can be studied. Methods are needed for the study of mineral metabolism in field studies in which a large number of subjects can be studied. The availability of dietary information and first void urine samples from approximately 100 preadolescent girls who were participating in the S-87 Southern Regional Nutrition Project

provided an opportunity to study the relationship between magnesium excretion in a first void urine sample and the dietary intake of magnesium.

The purpose of the study reported herein was to evaluate the relationship between the dietary intake of magnesium and the urinary excretion of magnesium:creatinine in first void urine samples of preadolescent black and white girls. The effects of race, income level and age on dietary magnesium intakes and the urinary excretion of magnesium:creatinine were also determined.

CHAPTER II

REVIEW OF LITERATURE

I. INDISPENSABILITY AND DISTRIBUTION

That magnesium is essential for life has been recognized since 1932 when Kruse et al. (3) identified an acute magnesium deficiency syndrome in rats. Magnesium ranks fourth among the principle cations in the extracellular fluid and it is the second most abundant cation within the cellular fluid (4,5, 6,7). The skeleton contains approximately one-half of the body's magnesium, while the remainder is distributed equally between muscle and nonmuscular soft tissue (8). The cerebrospinal fluid contains 2.5 mEq of magnesium/liter, while the erythrocyte and serum content ranges from 4.3 to 6.2 mEq/ liter and 1.5 to 2.1 mEq/liter, respectively (9). The plasma level is kept within a narrow range of 1.5 to 1.8 mg/100 ml (5).

II. BIOLOGICAL ROLE

Magnesium is necessary for energy-requiring biological functions which modulate neural excitability and muscular contraction (6,10,11). Magnesium exists in a single divalent state and serves as a prosthetic ion in enzymatic reactions critical to cellular metabolism (4,8,12). Reactions

encompassing a very wide spectrum of synthetic processes necessary for life show an absolute requirement for magnesium (13). Enzymes activated by the magnesium cation include all those utilizing ATP or catalyzing the transfer of phosphate groups. Within the cell, the active form of ATP exists with magnesium bound to the phosphate moiety (4).

Both magnesium and ATP are involved in nucleic acid synthesis (8,12). In the nucleus, sections of chromosomes are held together by calcium and magnesium; therefore, it appears possible that changes in magnesium concentrations in vivo exert a control on DNA synthesis and may influence the degree of chromosomal aberration (12). Ribosomes also require an optimal intracellular magnesium concentration in order to maintain their physical stability; otherwise they will dissociate into smaller particles (14). By inference, the activating effect of magnesium extends to protein, fat, and carbohydrate synthesis (4,8,10,14).

Very little is known concerning changes in intermediary metabolism during magnesium deficiency. Ghazi and Heaton (15) investigated the effect of magnesium deficiency on energy metabolism and protein synthesis by liver in the rat. They hypothesized that during magnesium deficiency, the primary disturbance occurred in energy metabolism and inhibition of protein synthesis was secondary to it. Mitochondria from magnesium-deficient rats and controls had similar rates of

oxygen uptake indicating that the electron transport chain was unaffected, but partial uncoupling of oxidative phosphorylation was indicated by lower P:O ratios. Liver slices from magnesium-deficient rats had a 35 percent decrease in rates of oxygen uptake when related to the protein content of the tissue as an index of muscle mass. There was a 29 percent reduction in amino acid incorporation into tissue protein. Despite the fact that magnesium is required for protein synthesis where it is needed for enzyme activation and maintaining structural conformation of nucleic acids and ribosomes (8,12,14), there is no existing evidence that magnesium deficiency in vivo directly inhibits the process (15).

III. RECOMMENDED DIETARY ALLOWANCE FOR MAGNESIUM

The RDA is 250 mg of magnesium per day for children aged 7 to 10 years weighing 30 kg and 300 mg for 11 to 14 year old children weighing 44 kg (16). Not only is the RDA currently expressed without specific reference to dietary factors most likely to influence magnesium utilization (17), but also, it is based on estimations derived from studies with adults or younger children (1). Greger et al. (2) studied mineral metabolism in 14 girls, aged 12.5 to 14.5 years, who were in negative magnesium balance on their customary dietary regimes. Although a dietary intake of 6 mg/kg body weight or above will

produce a positive magnesium balance, it is questionable whether this amount can be relied upon to maintain equilibrium constantly (18). It appears that a magnesium intake of 7 to 10 mg/kg body weight will compensate for a decrease in magnesium absorption and utilization caused by dietary and physiological factors (18).

IV. FOOD SOURCES

Cereals and vegetables are the main sources of magnesium in diets of all socioeconomic classes (19). Even though whole grains and brown rice contain more magnesium than refined grain products, their addition to a diet has been demonstrated to cause a negative balance due to the interference of phytic acid with magnesium absorption (18). As expenditure on food increases, cereals as a magnesium source become progressively displaced by poorer sources such as milk and dairy products. The magnesium supply from fruit increases disproportionately with an increase in food expenditure. Meat and fish contribute a relatively constant amount of approximately one-ninth of the total (19).

Greger et al. (20) determined the magnesium content of a variety of processed and convenience foods typically consumed by adolescents. In general, bread, cereal products, chocolate and licorice candies had 2 to 3 times as much magnesium per weight as milk, vegetables, and meat and meat alternatives.

When the ratios of μg of magnesium per kcal were calculated, vegetables had 4 times as much magnesium as other foods such as meat, milk, and cereal products. Snack foods such as candies and chocolate baked desserts had a particularly high magnesium content. Therefore, these foods may be a major source of dietary magnesium as they are very popular among this age group (20).

V. MAGNESIUM DEFICIENCY

The failure to detect magnesium deficiency in human subjects is understandable since the tissues affected include the cardiovascular, renal and neuromuscular systems (12,13, 21). Knowledge of the specific symptomatology and biochemical abnormalities is obviously important to its recognition (21). Magnesium deficiency symptoms have not been completely defined because of the failure to induce experimental symptomatic human deficiencies and accounts of clinical magnesium depletion have always occurred in a setting of predisposing and complicating disease states (12,13).

The effect of maturity on the susceptibility to magnesium deficiency and on the availability of magnesium was investigated in the rat by Smith and Field (22). Feeding rats a magnesium-free diet resulted in changes in plasma magnesium that were independent of age. Even though the minimal values reached after one week on the diet were similar

in both young and old rats, the clinical signs of magnesium deficiency were more severe in the young rat. Convulsions were noted only in young animals. Reddening of the ear and hyperirritability took longer to develop in the adult (11 to 14 days) than in the young animal (8 to 11 days). Only bone showed a reduction in its concentration of magnesium indicating that the reserves of magnesium in the body were confined mostly to the skeleton. After 18 days, the decrease in the magnesium concentration of the femur was 9.4 percent in the adult, and 28.2 percent in the young (22).

VI. MAGNESIUM HOMEOSTASIS AND TRANSPORT PROCESSES

In general, magnesium homeostasis is mediated through the relationship between absorbed dietary magnesium and magnesium excretion (12,13,18,23). Graham et al. (23) suggested that the absorption of magnesium was influenced by the amount presented to the intestinal mucosa. On an average diet containing 20 mEq of magnesium/day, 44.3 percent of the ingested ^{28}Mg radioisotope was absorbed. Absorption on a low diet (1.9 mEq/day) and a high diet (47 mEq/day) was 75.8 percent and 23.7 percent, respectively. Uniform absorption throughout the small intestine began within 1 hour and continued steadily for 2 to 8 hours. Little absorption occurred in the large bowel (12,13,18,23).

All evidence suggests that magnesium is not absorbed by an active transport system (24,25,26,27). Magnesium was not transported against a concentration gradient from mucosal to serosal solutions in rat everted gut sac preparations which suggested that the absorption of magnesium was by simple ionic diffusion (24,25). Increasing the mucosal magnesium concentration increased the absorption of magnesium (26). It is possible that magnesium is absorbed by a facilitated diffusion process involving a limited carrier system because calcium and magnesium interfered with the absorption of one another (27).

VII. INFLUENCE OF DIETARY CONSTITUENTS

Protein Interrelationships

Hunt and Schofield (28) suggested that the utilization of magnesium was altered by various levels of dietary protein. Schwartz et al. (29) observed a direct relationship between magnesium requirements and protein intake due to the critical involvement of magnesium in protein synthesis. When growing rats were fed inadequate amounts of magnesium, the effects of depletion were less severe with low protein diets than high protein diets. Higher levels of protein with marginal levels of dietary magnesium resulted in reduced weight gain and accelerated magnesium depletion of the total body. However, this depletion appeared to be offset by a tendency

toward greater total magnesium retention with high protein intakes while food intake was adequate. These data confirm the increase in magnesium requirements with an increased protein intake for maximal growth and nitrogen retention. The authors speculated that magnesium may be preferentially retained in tissues at cellular sites of protein synthesis possibly at the expense of other metabolic processes (29).

McCance et al. (30) demonstrated that with adults consuming an adequate magnesium intake (5.8 to 6.5 mg/day), retention was slightly better on a high than on a low protein intake. The average daily magnesium balance, expressed as a percentage of intake, on a low protein basal diet (45 to 70 g/day) was +12, and when that diet was supplemented with 100 to 130 g of protein per day, the average daily magnesium balance was +30. No significant effect on renal or intestinal excretions of magnesium was detected when the protein content of diets adequate in magnesium was increased.

For the purposes of comparison, these results cannot be applied to children. Schofield and Morrell (31) investigated the influence of protein intake on the magnesium balance of 35 preadolescent girls. Their data indicated that despite the improved magnesium intake in the 2 to 2.5 g N/kg body weight periods, magnesium retention, expressed in terms of percentage of intake, decreased with an increased protein intake. The mean daily magnesium balance, expressed as a percentage of intake, decreased from +16 to +11 (31).

Calcium, Magnesium and Phosphate Interrelationships

The effect of dietary $Ca:PO_4$ ratios on skeletal and magnesium metabolism in the rat has been investigated by Clark (32) who demonstrated that an increase in dietary $Ca:PO_4$ ratios increased the serum calcium level but decreased those of phosphate and magnesium. Inorganic and organic phosphate ingestion raised urinary calcium excretion, but the change in magnesium metabolism was of a lesser magnitude than that of calcium. This amount of magnesium was larger than could be accounted for from bone alone; therefore, magnesium as an intracellular cation could be used to neutralize excess hydrogen ions in the urine. In a similar study, magnesium exchange was not greatly affected by the ingestion of large amounts of phosphate and storage of phosphorus was not necessarily accompanied by a retention of magnesium or calcium (13).

Laboratory evidence indicated that high calcium intakes precipitated magnesium deficiency in rats (33,34,35) and guinea pigs (36) by increasing their dietary requirement for magnesium. Metabolic studies in rats have demonstrated that increased calcium intakes resulted in increased urinary magnesium losses. Alcock and MacIntyre (37) postulated that this close correlation between calcium and magnesium urinary excretion suggested a common tubular reabsorptive pathway. They reported that an increase in calcium absorption from the

gut and an increase in plasma calcium levels were associated with a magnesium deficiency in rats.

Clark (38) noted the antagonistic action of calcium and magnesium in rats. He discovered that the introduction of small amounts of magnesium to an adequate diet resulted in an initial fall in calcium retention. As magnesium intakes increased, fecal magnesium losses were depressed and magnesium retention was elevated. In another experiment, he determined that magnesium absorption could be increased when dietary levels of calcium were low (39). Therefore, the available data indicated that calcium interfered with the absorption and enhanced the renal excretion of magnesium. It is probable that calcium and magnesium share a common transport mechanism across the gut epithelium (23,39).

Magnesium Excretion

Magnesium excretion is by glomerular filtration and is almost completely reabsorbed in the tubules (5,12,13,18). A reproducible diurnal rhythm in calcium and magnesium excretion was demonstrated in a study conducted by Briscoe and Ragan (40). The amount excreted in the urine was less at night (41). The rhythmic variation was demonstrable in many individuals who were consuming diets of different composition and whose physical activity varied (42).

Excretory patterns on different magnesium intakes were fairly constant when the excretion data were expressed as

percentages of intakes (18). Approximately 60 to 80 percent of the magnesium intake is excreted in the feces and the remainder, other than that retained in new tissue or lost in sweat or desquamated skin, is excreted in the urine (13).

Researchers observed the metabolic behavior for each of 36 healthy preadolescent girls consuming foods typical of their dietary regimes for a total of 2,200 subject days (43). They reported values of 50.8 to 114.5 ug of magnesium/ml of urine. Other researchers determined that the magnesium content of normal urine ranged from 0.05 to 0.30 g/24 hours (13,44,45). Urinary creatinine excretion has been used extensively to indicate the completeness of 24-hour urine collections. It is assumed that the amount of creatinine excreted is fairly constant in an individual and that the influence of diet or urine volume is negligible (46). Graystone (47) reported an increase in creatinine excretion with age. Values for creatinine fall within 1.0 to 1.5 g/24 hours (44,45,46,47). According to these data, normal magnesium:creatinine ratios would fall within the range of 0.05 to 0.20.

CHAPTER III

EXPERIMENTAL PROCEDURE

I. GENERAL PLAN

The S-87 Regional Nutrition Project was a 2 year longitudinal study which supplied approximately 100 preadolescent girls between the ages of 9±0.5 and 11±0.5 years for the present study. Fifty percent of the girls were from upper income families ($2,000 or more/person/year) and 50 percent were from lower income families ($1,000 or less/person/year). The racial distribution for the sample was 50 percent white and 50 percent black. The subjects were obtained through the cooperation of the city and county school systems of Knox County, Tennessee. Girls with no known metabolic disorders were selected. An informed written consent was signed by the parents or guardians. Dietary intakes and urinary excretion of magnesium in a first void urine sample were investigated.

II. COLLECTION METHODS

Dietaries

Dietary information was obtained from each subject twice a year using 24-hour recalls of food intake. The first 24-hour recall was completed approximately 2 weeks prior to

the day of urine collection and the second one was taken the same day the urine was collected. The 24-hour dietary recalls were obtained by trained interviewers using food models. Average nutrient compositions of summarized dietaries were calculated using the Extended Table of Nutrient Values from the Department of Experimental Statistics, Louisiana State University, Baton Rouge, Louisiana.

Urine

Following an overnight fast, the first voided urine upon rising in the morning was collected in a screw cap polyethylene quart bottle. Each subject received written instructions concerning the collection and care of her urine sample. The bottles used for the urine collection were labeled as to the subject and the date. The samples were picked up approximately 1 hour after collection, placed in ice, and transported to the laboratory. Each urine sample was stored at -30° for future analyses.

III. ANALYSES OF URINE

Magnesium

Magnesium utilization was investigated by using first void urine samples of preadolescent girls consuming self-selected diets. All glassware was acid-rinsed in 0.1N HCl. Only redistilled water was used. Precaution was taken at all phases of analyses in order to minimize contamination.

Method. The analytical stock magnesium sulfate solution contained 1,000 ppm of magnesium in aqueous solution. Five ml of the analytical stock solution were diluted with redistilled water to 100 ml in a volumetric flask. Working standards, prepared from the stock solution, contained 0.1, 0.2, and 0.5, 1.0, 1.5, and 2.0 ppm of magnesium. Two ml of 0.5 percent lanthanum chloride were added to 8 ml of each working standard. The concentration of lanthanum chloride protected the magnesium determination from interference from as much as 200 ppm of phosphorus (48,49). The standard operating conditions for the determination of magnesium with the Perkin-Elmer 303 Atomic Absorption Spectrophotometer (50) were the following: wavelength—285Å; range—UV; slit—5; source—hollow cathode; fuel—acetylene; flow—9; and oxidizer—air. Once the absorbance (A) of the magnesium standards was determined, a standard curve was drawn which was linear within the range of 0.1 to 2.0 ppm.

One ml of urine was diluted with redistilled water to 25 ml in a volumetric flask. Each volumetric flask was inverted several times for mixing. Then 1 ml of diluted urine was added to 1 ml of $LaCl_3$ and 8 ml of redistilled water. A dilution factor of 250 was derived from the 1:25 and 1:10 dilutions. All aliquots were prepared in duplicate and thoroughly mixed for 30 seconds by an automatic mixer.[1]

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[1]Vortex-Genie, Scientific Industries, Springfield, MA.

The absorbance (A) of the urinary magnesium samples was then determined.

Calculations.

$$\frac{\mu g \text{ of } Mg^{+2}}{ml \text{ of urine}} = \frac{A \text{ of sample}}{A \text{ of std}} \times \text{conc of std} \times \text{dilution factor}$$

Creatinine

Creatinine in first void urine samples was allowed to react with alkaline solutions of picrate at room temperature according to Folin's Method (51). The creatinine-picrate derivative was a color complex that could be quantitated at 520 nm on the Beckman Model 24 Spectrophotometer.[2]

Reagents.

1. Picric acid, 0.04M: 10.17 g reagent grade (containing approximately 10 percent H_2O) picric acid was diluted to 1 liter in a volumetric flask.

2. Sodium hydroxide, 0.75N: 30.0 g NaOH was dissolved in 1 liter of water.

3. Alkaline-Picrate solution: equal volumes of picric acid reagent and 0.75N NaOH were combined and mixed thoroughly.

4. Creatinine stock standard, 1.5 mg/ml: 1.50 g reagent grade creatinine was dissolved in 1 liter of 0.1N HCl.

5. Working standard of creatinine, 15.0 µg/ml: 1 ml of stock standard was diluted to 100 ml with water.

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[2]Beckman Instruments, Inc., Fullerton, CA.

Method. One ml aliquots of urine were transferred to 100 ml volumetric flasks and diluted to volume with water. Duplicate 3 ml aliquots of the diluted urine samples were pipetted into test tubes. To the duplicates of urine, 2 ml of alkaline-picrate reagent were added and mixed thoroughly with an automatic mixer.[3] At the end of 20 minutes, the absorbance (A) of the samples was recorded at 520 nm. Each series of samples included standards of creatinine containing 0, 7.5, 15, 30, and 45 ug/tube.

Calculations.

$$\frac{\text{ug of creatinine}}{\text{ml of urine}} = \frac{\text{A of sample}}{\text{A of std}} \times \text{conc of std} \times \text{dilution factor}$$

IV. STATISTICAL ANALYSES

Various forms of statistical analyses were implemented by the use of the Statistical Analysis System at the University of Tennessee (52). Magnesium:creatinine ratios were calculated for each subject. Means and standard errors of the means were determined for dietary intakes and urinary excretion values by age, race, and income level. Multiple regression analysis was chosen as a means of interpreting the regression of one variable on several independent variables (53). The Student's t-test was used for testing

[3]Vortex-Genie, Scientific Industries, Springfield, MA.

the differences between the 2 races and between the 2 income groups with regard to magnesium intakes and the urinary excretion of magnesium, creatinine and magnesium:creatinine.

CHAPTER IV

RESULTS

Mean dietary intakes of magnesium by race, income level and age are shown in Table 1. For all 3 age groups, high income whites had the highest mean dietary intake of magnesium. High income black girls, at 9±0.5 and 10±0.5 years of age, had the lowest mean dietary intake of magnesium.

The regression of age on dietary intakes of magnesium and urinary magnesium:creatinine ratios for each race and income level could not be determined due to the small size of the sampling distribution. Therefore, the data for all groups were combined by race and income level for further analyses. The influence of race and income level on dietary intakes of magnesium is presented in Table 2. Whites had a significantly greater ($P < 0.05$) mean dietary magnesium intake than did blacks when income levels were controlled. There was no significant difference ($P < 0.05$) in mean dietary intakes between income levels when races were controlled.

Comparisons of magnesium intakes with the RDA by race, income level and age are shown in Table 3. During the study, only 3 low income white girls, a 9±0.5 year old and 2 11±0.5 year olds, were below 33 percent of the RDA.

More 9±0.5 year old low income whites met 33 to 67 percent of the RDA than did 10±0.5 or 11±0.5 year old low

TABLE 1

DIETARY INTAKES OF MAGNESIUM BY RACE, INCOME LEVEL AND AGE

	White Income		Black Income	
	Low	High	Low	High
	----------mg---------		----------mg---------	
9±0.5	219±20[1] (n = 30)	247±20 (n = 11)	234±10 (n = 28)	214±10 (n = 22)
10±0.5	241±20 (n = 21)	263±20 (n = 19)	228±20 (n = 6)	210±20 (n = 15)
11±0.5	213±10 (n = 30)	261±10 (n = 25)	225±10 (n = 30)	221±10 (n = 28)

[1]Values are means ± SEM.

TABLE 2

EFFECTS OF RACE AND INCOME LEVEL ON DIETARY INTAKES OF MAGNESIUM FOR PREADOLESCENT GIRLS AGED 9±0.5 to 11±0.5 YEARS

	Race		Income	
	White (n = 136)	Black (n = 129)	Low (n = 145)	High (n = 120)
	mg		mg	
Dietary Magnesium	240±6.88[1,2]	222±5.89[2]	226±6.43	236±6.40

[1]Values are means ± SEM.

[2]Means are significantly different ($P < 0.05$) as indicated by the t-test.

TABLE 3

COMPARISONS OF MAGNESIUM INTAKE WITH THE RDA BY RACE, INCOME LEVEL AND AGE

		Percent of Subjects											
		Below 33% RDA			33-67% RDA			67-100% RDA			Above 100% RDA		
		9±0.5	10±0.5	11±0.5	9±0.5	10±0.5	11±0.5	9±0.5	10±0.5	11±0.5	9±0.5	10±0.5	11±0.5
White	Low Income	1.1	0	2.6	9.9	5.0	7.1	13.2	14.7	13.3	8.8	14.7	3.5
	High Income	0	0	0	1.1	0	4.4	6.6	16.4	10.6	4.4	14.7	7.1
Black	Low Income	0	0	0	1.1	1.6	8.8	19.9	3.3	14.2	9.9	5.0	3.5
	High Income	0	0	0	6.6	6.5	12.4	12.1	13.1	8.8	5.5	5.0	3.5
TOTAL		1.1	0	2.6	18.7	13.1	32.7	51.8	47.5	46.9	28.6	39.4	17.6

income whites. The percentages of high income whites, and low and high income blacks meeting 33 to 67 percent of the RDA were higher for girls aged 11±0.5 years than for girls aged 9±0.5 years. The high income blacks composed the greatest percentage of this category during the study.

The greatest percentage of subjects met 67 to 100 percent of the RDA. More low income blacks (19.9 percent) met 67 to 100 percent of the RDA than low income whites (13.2 percent), high income whites (6.6 percent), or high income blacks (12.1 percent) at age 9±0.5 years. More 10±0.5 year old whites (31.1 percent) fell into this category than blacks (16.4 percent) of the same age, regardless of income level. More low income subjects (27.5 percent) met 67 to 100 percent of the RDA than high income subjects (19.4 percent) at 11±0.5 years of age. The low income whites composed the greatest percentage of this category during the study.

In the greater than 100 percent of the RDA category, there was a trend toward a peak at 10±0.5 years of age for whites. The percentage of blacks falling into this category gradually decreased with age. Low income groups and whites composed the greatest percentage of this category during the study.

The means for the urinary excretion of magnesium, creatinine and magnesium:creatinine by race and income level for 9±0.5, 10±0.5 and 11±0.5 year old girls are shown in

Tables 4, 5, and 6, respectively. There were no significant differences ($P < 0.01$) in urinary magnesium or magnesium: creatinine ratios for race and income levels when the 3 age groups were considered separately (Tables 4-6) or combined (Table 7). Creatinine excretion was significantly greater ($P < 0.01$) for blacks (Table 7). The results for creatinine excretion indicated an increased urinary excretion with age.

Quartiles were established for the urinary excretion ratios. The percentages of subjects from each race, income level and age group in each quartile and the range of each quartile are illustrated by bar graphs in Figures 1, 2, and 3. There were no specific trends in excretion ratios by race or income level during the study. For 9±0.5 year olds, whites and low income groups had greater urinary magnesium: creatinine ratios than did blacks and high income groups (Figure 1). This situation completely reversed itself for 10±0.5 year olds. At this age, a greater percentage of blacks and high income groups had excretion ratios in the upper quartiles than did white and low income groups (Figure 2). For 11±0.5 year olds, more whites and high income groups had excretion ratios in the 0.10 to 2.00 range than did blacks or low income groups (Figure 3). There were more whites than blacks in the 4th quartile at every age (Figures 1-3).

One objective of the study was to investigate the relationships among dietary magnesium intakes and urinary

TABLE 4

URINARY VALUES FOR MAGNESIUM, CREATININE AND MAGNESIUM:CREATININE BY RACE AND INCOME LEVEL FOR 9±0.5 YEAR OLD GIRLS

	White Income		Black Income	
	Low (n = 30)	High (n = 11)	Low (n = 28)	High (n = 22)
	ug/ml			
Urinary Magnesium	205.26±27.70[1]	123.52±30.31	200.92±18.06	121.62±18.00
Creatinine	1354.53±253.81	829.54±69.43	1279.10±139.62	1241.57±117.03
Magnesium: Creatinine	0.233±0.04	0.150±0.04	0.169±0.02	0.126±0.04

[1]Values are means ± SEM.

TABLE 5

URINARY VALUES FOR MAGNESIUM, CREATININE AND MAGNESIUM:CREATININE BY RACE AND INCOME LEVEL FOR 10±0.5 YEAR OLD GIRLS

	White Income		Black Income	
	Low (n = 21)	High (n = 19)	Low (n = 6)	High (n = 15)
	ug/ml			
Urinary Magnesium	120.56±23.76[1]	133.61±19.97	173.56±57.24	222.43±33.31
Creatinine	1103.43±107.40	1023.37±93.99	1310.71±206.45	1565.71±162.31
Magnesium: Creatinine	0.153±0.02	0.143±0.02	0.148±0.05	0.206±0.05

[1]Values are means ± SEM.

TABLE 6

URINARY VALUES FOR MAGNESIUM, CREATININE AND MAGNESIUM:CREATININE BY RACE AND INCOME LEVEL FOR 11±0.5 YEAR OLD GIRLS

	White Income		Black Income	
	Low (n = 30)	High (n = 25)	Low (n = 30)	High (n = 28)
	ug/ml			
Urinary Magnesium	180.64±21.56[1]	133.35±20.35	143.67±19.62	153.84±17.69
Creatinine	1650.86±160.62	1047.70±149.51	1553.04±205.80	2068.42±220.37
Magnesium: Creatinine	0.167±0.06	0.146±0.02	0.136±0.04	0.092±0.01

[1]Values are means ± SEM.

TABLE 7

EFFECT OF RACE AND INCOME LEVEL ON URINARY MAGNESIUM, CREATININE AND MAGNESIUM: CREATININE FOR PREADOLESCENT GIRLS AGES 9±0.5 to 11±0.5 YEARS

	Race		Income	
	White (n = 136)	Black (n = 129)	Low (n = 145)	High (n = 120)
	ug/ml			
Urinary Magnesium	164.89±10.39[1]	165.64±10.18	180.38±10.52	146.97±9.58
Creatinine	1244.34±78.17[2]	1558.87±81.93[2]	1398.14±81.18	1396.62±82.26
Magnesium: Creatinine	0.171±0.018	0.141±0.014	0.172±0.019	0.137±0.062

[1]Values are means ± SEM.

[2]Means are significantly different ($P < 0.01$) as indicated by the t-test.

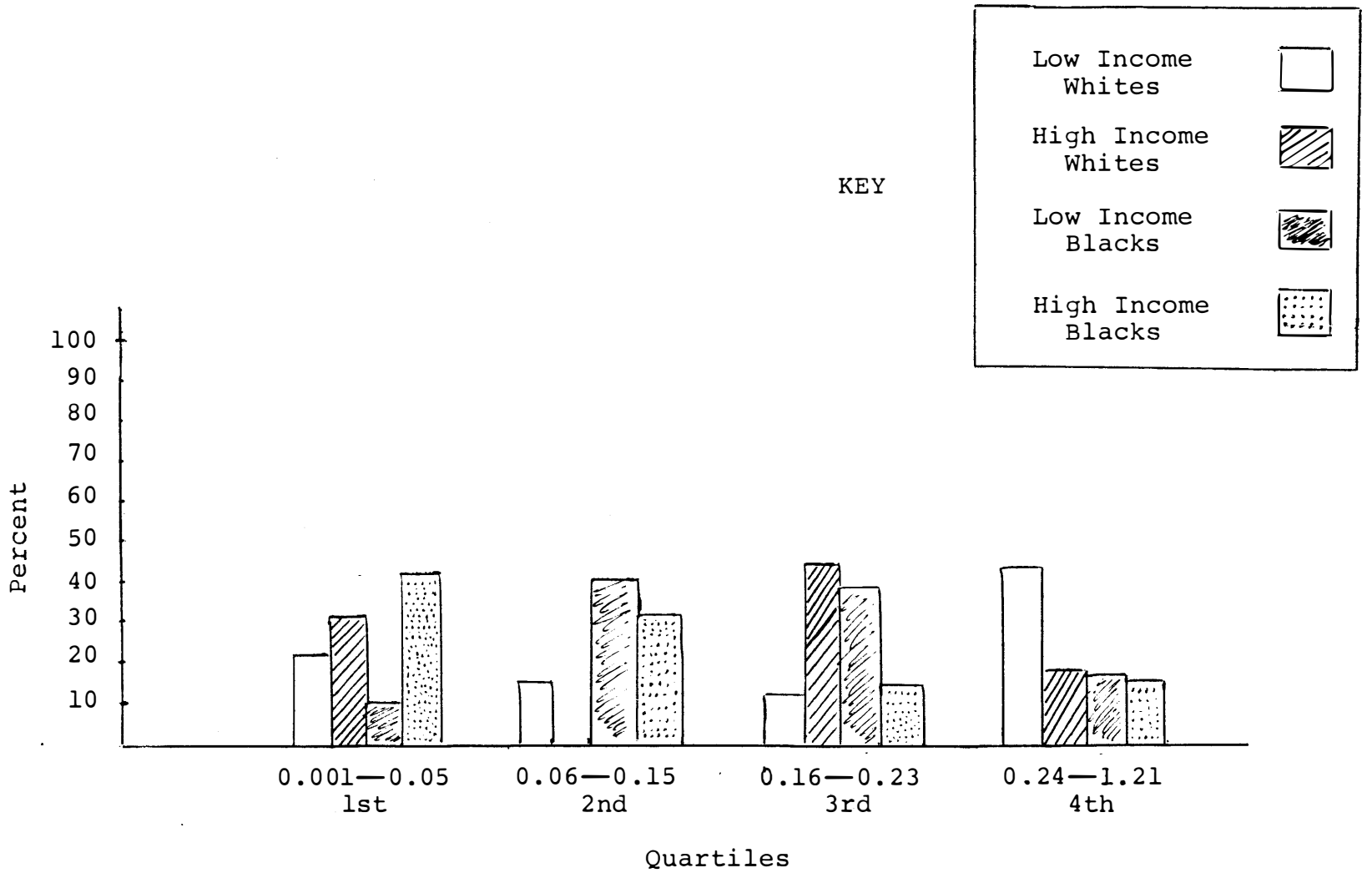

Figure 1. Urinary Excretion of Magnesium:Creatinine for Girls 9±0.5 Years Old.

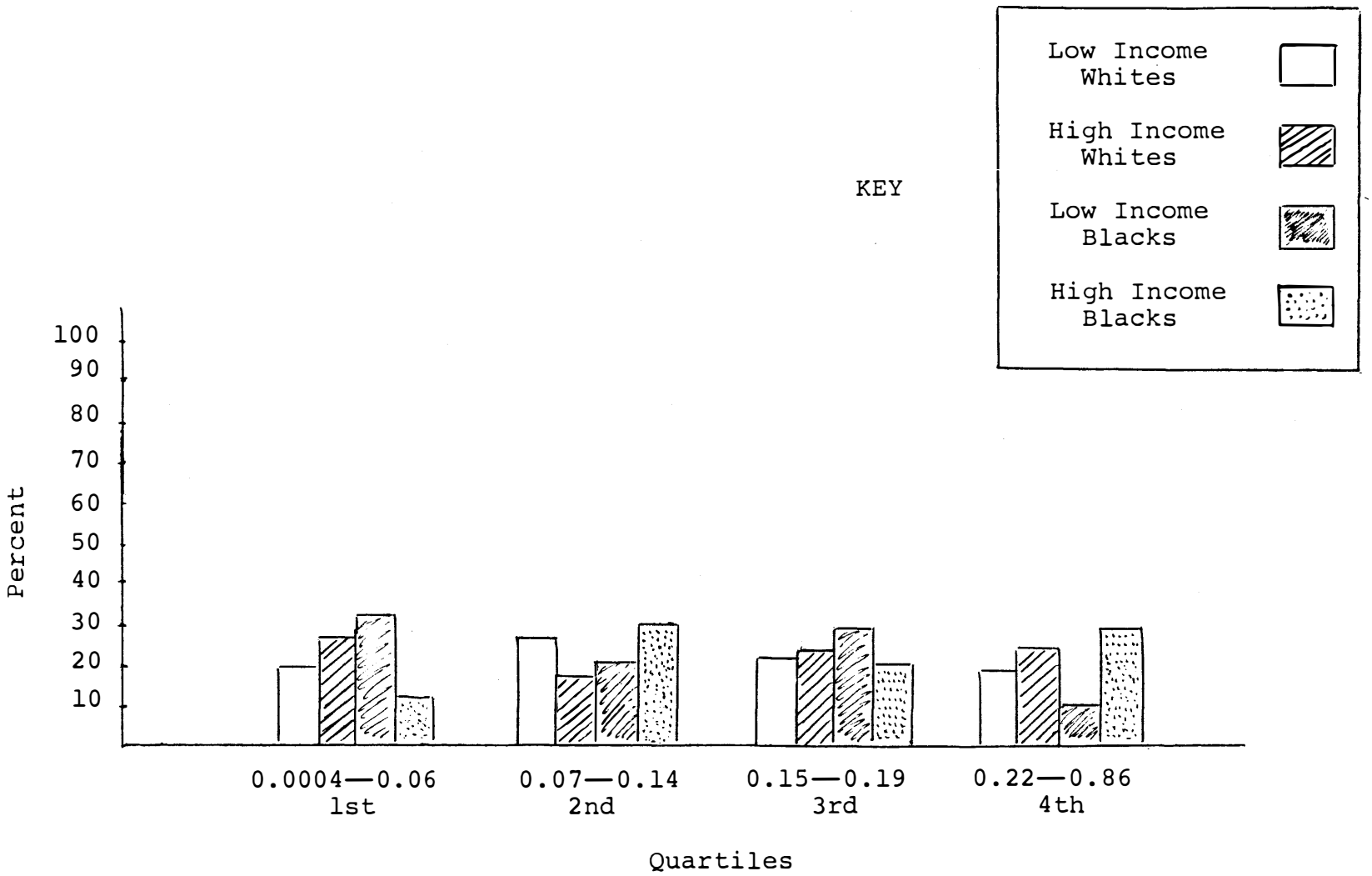

Figure 2. Urinary Excretion of Magnesium:Creatinine for Girls 10±0.5 Years Old.

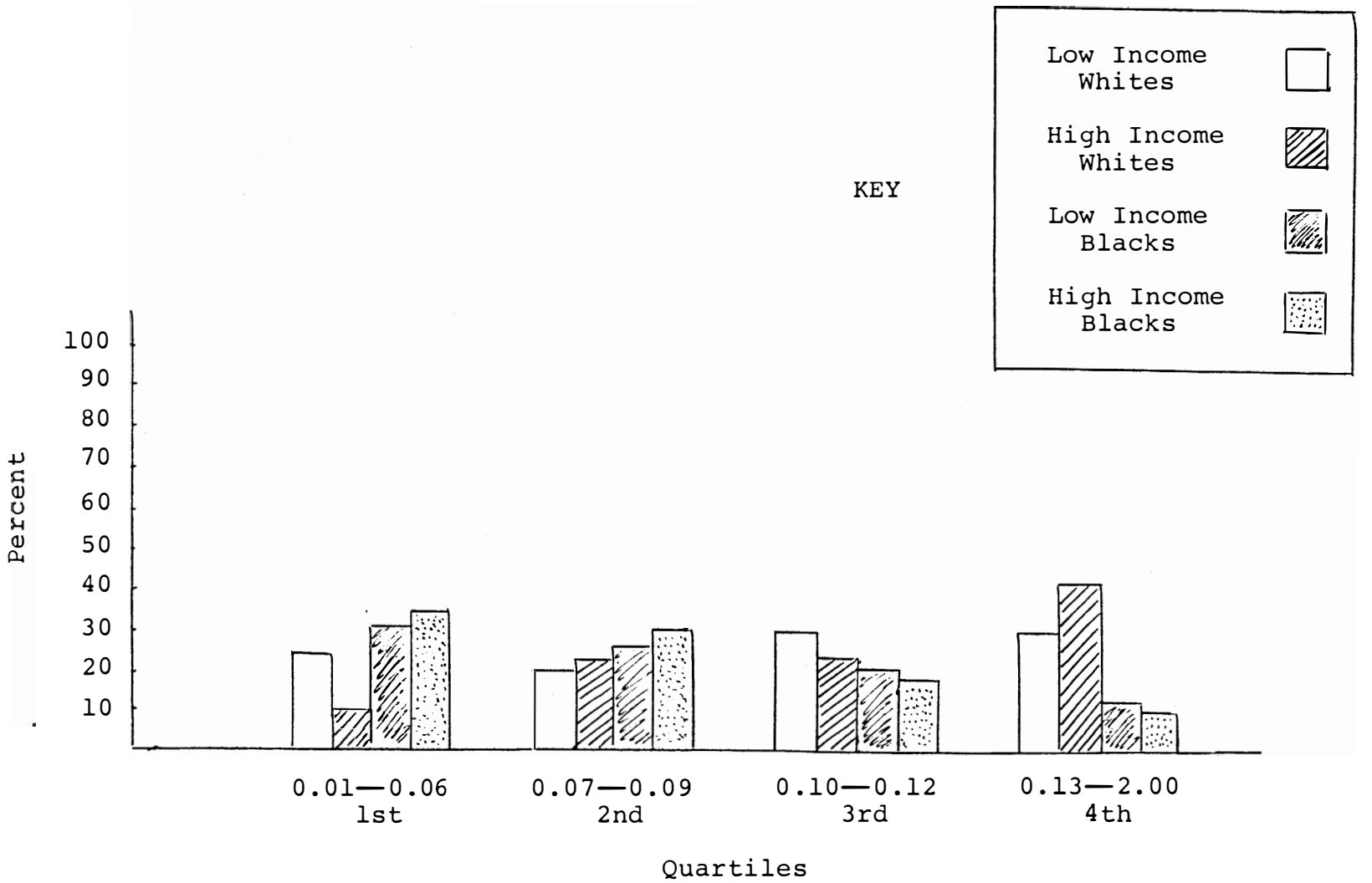

Figure 3. Urinary Excretion of Magnesium:Creatinine for Girls 11±0.5 Years Old.

magnesium:creatinine ratios in preadolescent black and white girls. Pearson correlation coefficients were calculated for dietary and urinary variables and the values are presented in Table 8. Correlations among total protein, calcium, phosphorus and dietary magnesium were highly significant ($P < 0.01$). There were no significant correlations between dietary and urinary magnesium variables.

TABLE 8

PEARSON CORRELATION COEFFICIENTS BETWEEN DIETARY AND URINARY VARIABLES FOR PREADOLESCENT GIRLS AGED 9±0.5 TO 11±0.5 YEARS

	Total Protein	Calcium	Phosphorus	Dietary Magnesium	Urinary Magnesium
Calcium	0.77[a]				
Phosphorus	0.92[a]	0.93[a]			
Dietary Magnesium	0.87[a]	0.81[a]	0.91[a]		
Urinary Magnesium	-0.13	-0.06	-0.11	-0.09	
Magnesium: Creatinine	0.05	0.06	0.03	0.05	0.01

[a] $P < 0.01$.

CHAPTER V

DISCUSSION

Mean dietary intakes of magnesium for the girls were lower than the RDA for their respective age groups except for 10±0.5 year old high income whites (Table 1, p. 21). Although the greatest percentage of the girls did meet 67 to 100 percent of the RDA during the 2 year longitudinal study, it is questionable whether this amount could be relied upon to maintain equilibrium constantly during periods of rapid growth (16,18). Based on data from a metabolic study, it appeared that a magnesium intake of 7 to 10 mg/kg body weight, would compensate for dietary and physiological factors which might interfere with magnesium utilization (18). Calculations for mg of magnesium/kg body weight, based on the minimum figure, revealed that 9±0.5 year old low income whites, 10±0.5 year old blacks, and all 11±0.5 year olds were below this recommendation. The total percentages of girls who met above 100 percent of the RDA were 28.6 percent, 39.4 percent, and 17.6 percent for 9±0.5, 10±0.5, and 11±0.5 year olds, respectively (Table 3, p. 23). More low income whites than blacks were below 33 percent of the RDA and more high income blacks than whites met 33 to 67 percent of the RDA.

The influence of income level on dietary magnesium intakes found in this study was not in agreement with the

findings of Duckworth and Warnock (19). Their research indicated that the income level and dietary intake of magnesium were inversely related. The results of the present study showed that the difference in mean dietary magnesium intakes between high and low income groups was not significant ($P < 0.05$). The influence of race was significant (Table 2, p. 22).

Positive correlations among dietary variables were highly significant ($P < 0.01$) (Table 8). The positive correlation seen between total dietary protein and dietary magnesium was not surprising because total protein included vegetable as well as animal protein. This finding was consistent with that reported by other researchers (19,20).

The findings of the present study involving human subjects were not statistically significant with regard to urinary magnesium and dietary variables. Clark (38) reported that magnesium intake and urinary magnesium excretion were inversely related in the rat. The results of Schwartz et al. (29) indicated that total magnesium retention in the rat was greater with high protein intakes while food intake was adequate; therefore, the urinary excretion of magnesium would be less. The group mean intakes for protein were greater than the RDA for the preadolescent girls at each age.[4] Alcock and

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[4] Wakefield, T. (1979) The Relationship of Biochemical and Dietary Parameters to Growth in Preadolescent Black and White Girls. Unpublished doctoral dissertation.

MacIntyre (37) demonstrated that increased calcium intakes resulted in increased urinary magnesium losses in the rat.

In previous studies, the urinary excretion of magnesium has been analyzed in 24-hour urine samples (13,44,45,46,47). Mean urinary magnesium values by race and income level reported in this study, ranging from 120 to 222 ug/ml of urine (Tables 4-6, pp. 26-28) are slightly high compared to the values of 50.8 to 114 ug/ml reported in a metabolic balance study of preadolescent girls (43). The mean urinary magnesium value for all subjects was 165.25 ug/ml. Mean creatinine values by race, income level and age were in agreement with values reported in other studies (44,45,46,47). It seemed logical that blacks had a significantly greater ($P < 0.01$) creatinine excretion than did whites (Table 7, p. 29) because blacks were taller and heavier. The increase in urinary excretion of creatinine with age was consistent with the findings of Graystone (47).

There are no established standards on normal ratios of magnesium:creatinine. Calculations of magnesium:creatinine ratios from existing data resulted in a range of 0.05 to 0.20 (13,44,45). Mean magnesium:creatinine ratios by race and income level, ranging from 0.137 to 0.172, compared favorably with ratios calculated from literature values. The mean ratio for all subjects was 0.16.

Failure to find correlations between the dietary intake and urinary excretion of magnesium or to identify specific

trends in the urinary excretion of magnesium was disappointing. Such information would have proven quite useful in the development of a magnesium:creatinine standard from a first void urine sample to be used in large field studies where 24-hour collections are difficult.

CHAPTER VI

SUMMARY

One of the purposes of this study was to determine the relationship between the dietary intake of magnesium and urinary excretion of magnesium:creatinine from a first void urine sample of preadolescent girls. The other purpose was to evaluate the effects of race and income levels on the dietary intake and urinary excretion of magnesium in a first void urine sample.

Whites had a significantly greater ($P < 0.05$) mean dietary magnesium intake than did blacks, but there was not a significant difference between income levels when the data were combined for all 3 age groups. The greatest percentage of subjects met 67 to 100 percent of the RDA. Correlations among total dietary protein, calcium, phosphorus and magnesium were highly significant ($P < 0.01$).

For girls aged 9 ± 0.5 to 11 ± 0.5 years, there were no specific trends in excretory patterns for magnesium by race and income groups. Blacks had a significantly greater ($P < 0.01$) creatinine excretion than did whites. There were no significant differences ($P > 0.05$) for urinary magnesium or magnesium:creatinine ratios by race or income groups.

There were no significant correlations between dietary magnesium intakes and the urinary excretion of magnesium or

magnesium:creatinine ratios. Therefore, it was not possible to develop a magnesium standard based on creatinine from a first void urine sample.

REFERENCES

REFERENCES

1. Wang, C. C., Kaucher, M. & Wing, M. (1936) Metabolism of adolescent girls. IV. Mineral metabolism. Am. J. Dis. Child. 52, 41-50.

2. Greger, J. L., Baligar, P., Abernathy, R. P., Bennett, O. A. & Peterson, T. (1978) Calcium, magnesium, phosphorus, copper, and manganese balance in adolescent females. Am. J. Clin. Nutr. 31, 117-121.

3. Kruse, H. D., Orent, E. R. & McCollum, E. V. (1932) Studies on magnesium deficiency in animals. J. Biol. Chem. 96, 519-539.

4. Bhagavan, N. V. (1974) Biochemistry—A Comprehensive Review, pp. 123, 699, J. B. Lippincott Co., Philadelphia.

5. Vaughn, J. (1975) Magnesium. In: The Physiology of Bone, pp. 129-133, Clarendon Press, England.

6. Tibbetts, D. M. & Aub, J. C. (1957) Magnesium metabolism in health and disease. J. Clin. Invest. 16, 491-501.

7. Barker, E. S. (1960) Physiologic and clinical aspects of magnesium metabolism. J. Chron. Dis. 11, 278-291.

8. Wacker, W. E. (1968) Magnesium metabolism. New Eng. J. M. 278, 658-663.

9. Shils, M. E. (1975) Magnesium. In: Present Knowledge in Nutrition, 4th ed., pp. 247-255, Nutrition Foundation Pub., Wash., D.C.

10. Robinson, C. H. (1972) Normal and Therapeutic Nutrition, pp. 110-111, Macmillan Co., New York.

11. Lilienthal, J. L. (1952) Interrelations of magnesium, potassium, phosphorus, and creatine in skeletal muscle of man. J. Clin. Invest. 31, 850-858.

12. Prasad, A. S. & Oberleas, N. (1976) Trace Elements in Human Health and Disease, pp. 1-78, Academic Press, New York.

13. Nordin. B. E. (1976) Calcium, Phosphate and Magnesium Metabolism, pp. 36-186, Churchill Livingstone, England.

14. Lehninger, A. L. (1950) Role of metal ions in enzyme systems. Physiol. Rev. 30, 393-429.

15. Ghazi, G. A. & Heaton, F. W. (1978) Effect of magnesium deficiency on energy metabolism and protein synthesis by liver. Intern. J. Biochem. 9, 421-426.

16. National Research Council. (1974) Recommended Dietary Allowances, 8th ed. National Academy of Sciences—National Research Council, Wash., D.C.

17. Schmidt, C. L. & Greenberg, D. M. (1935) Occurrence, transport, and regulation of calcium, magnesium, and phosphorus in the animal organism. Physiol. Rev. 15, 297-434.

18. Seelig, M. S. (1964) The requirement of magnesium by the normal adult. Summary and analysis of published data. Am. J. Clin. Nutr. 14, 342-389.

19. Duckworth, J. & Warnock, G. M. (1942) Magnesium requirements in relation to calcium requirements, with observation on adequacy of diets in common use. Nutr. Abs. & Rev. 12, 167-183.

20. Greger, J. L., Marhefka, S. & Geissler, A. H. (1978) Magnesium content of selected foods. J. Food Sci. 43, 1610-1613.

21. Shils, M. E. (1975) Magnesium. In: Modern Nutrition in Health and Disease (Goodhart, R. S. & Shils, M. E., ed.), pp. 287-297, Lea & Febiger, Inc., Philadelphia.

22. Smith, B. S. & Field, A. C. (1963) Effect of age on magnesium deficiency in rats. Brit. J. Nutr. 17, 591-599.

23. Graham, L. A., Caesar, J. J. & Burgen, A. S. (1960) Gastrointestinal absorption and excretion of 28Mg in man. Metab. Clin. Exp. 9, 646-659.

24. Hendrix, J. Z., Alcock, N. W. & Archibald, R. M. (1963) Competition between calcium, strontium and magnesium for absorption in isolated rat intestine. Clin. Chem. 9, 734-744.

25. Aldor, T. A. & Moore, E. W. (1970) Magnesium absorption by everted sacs of rat intestine and colon. Gastr. 59, 745-755.

26. Ross, D. B. (1962) In vitro studies on the transport of magnesium across the intestinal wall of the rat. J. Physiol. 160, 417-428.

27. O'Donnell, J. M. & Smith, M. W. (1973) Uptake of calcium and magnesium by rat duodenal mucosa analysed by means of competing metals. J. Physiol. 299, 733-749.

28. Hunt, S. & Schofield, F. A. (1969) Magnesium balance and protein intake level in adult human females. Am. J. Clin. Nutr. 22, 367-373.

29. Schwartz, R., Walker, G., Linz, M. D. & MacKellar, I. (1973) Metabolic responses of adolescent boys to two levels of dietary magnesium and protein. Am. J. Clin. Nutr. 6, 510-518.

30. McCance, R. A., Widdowson, E. M. & Lehmann, H. (1942) The effect of protein intake on the absorption of calcium and magnesium. Biochem. J. 36, 686-691.

31. Schofield, F. A. & Morrell, E. (1960) Calcium, phosphorus and magnesium. Fed. Proc. 19, 1014-1016.

32. Clark, I. (1969) Importance of dietary calcium: phosphate ratios on skeletal calcium, magnesium, and phosphate metabolism. Am. J. Physiol. 217, 865-870.

33. Tufts, E. V. & Greenberg, D. M. (1938) The biochemistry of magnesium deficiency. I. Chemical changes resulting from magnesium deprivation. J. Biol. Chem. 122, 693-726.

34. Watchorn, E. & McCance R. A. (1937) Subacute magnesium deficiency in rats. Biochem. J. 31, 1379-1390.

35. Colby, R. W. & Frye, C. M. (1951) Effect of feeding various levels of calcium, potassium and magnesium to rats. Am. J. Physiol. 166, 408-412.

36. O'Dell, G. L. (1960) Magnesium requirement and its relation to other dietary constituents. Fed. Proc. 19, 648.

37. Alcock, N. & MacIntyre, I. (1962) Interrelation of calcium and magnesium absorption. Clin. Sci. 22, 185-191.

38. Clark, I. (1965) Relation of magnesium ions to calcium and phosphate absorption. Nature 207, 982-989.

39. Clark, I. (1969) Metabolic interrelations of calcium magnesium, and phosphate. Am. J. Physiol. 217, 871-878.

40. Briscoe, A. M. & Ragan, C. (1966) Dirunal variations in calcium and magnesium excretion in man. Metab. Clin. Exp. 15, 1002-1010.

41. Mills, J. N. (1968) Human circadian rhythms. Physiol. Rev. 46, 128-171.

42. Manchester, R. C. (1933) The diurnal rhythm in water and mineral exchange. J. Clin. Invest. 12, 995-1008.

43. United States Department of Agriculture. (1967) Basic Data on Metabolic Patterns in 7- to 10-Year-Old Girls in Selected Southern States, pp. 1-3, 111-125, Washington, D.C.

44. Lagura, R. T., Claudio, V. W. & Thiele, V. F. (1974) Nutrition and Diet Therapy, p. 323, C. V. Mosby Co., St. Louis.

45. Harper, H. A., Rodwell, V. W. & Mayes, P. A. (1977) Review of Physiological Chemistry, 16th ed., p. 625, Lange Medical Publications, CA.

46. Edwards, O. M., Bayliss, R. I. S. & Millen, S. (1969) Urinary creatinine excretion as an index of the completeness of 24-hour urine collection. The Lancet II, pp. 1165-1166.

47. Graystone, J. E. (1968) Creatinine Excretion During Growth. In: Human Growth Body Composition, Cell Growth, Energy, and Intelligence (Cheek, D. B. ed.), pp. 182-197, Lea & Febiger, Philadelphia.

48. Willis, J. D. (1961) The determination of calcium and magnesium in urine by atomic absorption spectroscopy. Anal. Chem. 33, 556-559.

49. Allan, J. E. (1958) Atomic absorption spectrophotometry with special reference to the determination of magnesium. Analyst 83, 466-471.

50. Perkin-Elmer Corporation. (1966) Analytical methods for atomic absorption spectrophotometry. Perkin-Elmer Corporation, Norwalk, CT.

51. Folin, O. (1914) On the determination of creatinine and creatine in urine. J. Biol. Chem. 17, 469-474. Modified by Hawk, P. B., Oser, B. L. & Summerson, W. H. (1954) Creatinine. In: Practical Physiological Chemistry. 13th ed., pp. 899-901, The Blakiston Co., Inc., New York.

52. Barr, A. J. & Goodnight, J. H. (1972) Statistical Analysis System. Department of Statistics, North Carolina State University, Raleigh, North Carolina.

53. Kelly, F. J., Beggs, D. L., McNeil, K. A., Eichelberger, T. & Lyon, J. (1969) Multiple Regression Approach, pp. 11-17, 45-61, Southern Illinois University Press, Carbondale & Edwardsville.

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