韓國營養學會誌 27(9) : 940~948, 1994
Korean J Nutrition 27(9) : 940~948, 1994

Effect of Maternal Selenium Nutrition on Pulmonary Selenium, Glutathione Peroxidase, and Phospholipid Levels in Neonatal Rats

Kim, Hye Young

Department of Food and Nutrition, Yong In University, Kyunggi Do, Korea

ABSTRACT

The present study was designed to determine if prenatal and postnatal Se nutriture affects Se concentration, glutathione peroxidase(GSHPx) activity and phospholipid distribution of the neonatal rat lung. Female SD rats were bred and fed a semipurified Se-deficient(0.04ppm, Se−) or a Se-adequate(0.5ppm, Se+) diet through pregnancy and lactation. On d 2 of lactation, maternal dietary Se had no significant effect on pulmonary Se concentration of pups. On d 16 of lactation, mean milk Se concentration in Se− dams was significantly lower than that in Se+ dams. Milk Se concentration was reflected on lung Se concentration and GSHPx activity of d 16 pups, which were dramatically decreased in Se− pups. In addition, pulmonary disaturated phosphatidyl choline/total phosphatidyl choline ratio was also significantly decreased in Se− pups, implying impaired function of pulmonary surfactant. These data indicate that adequate Se nutrition is important in the maturation of neonatal rat lungs.

KEY WORDS : selenium · glutathione peroxidase · lung · surfactant.

Introduction

It is well known that adequate selenium(Se) nutrition is essential for proper reproduction. In several species Se supplementation during pregnancy and lactation is crucial for prevention of deficiency signs such as retained placenta in dairy cows, white muscle diseases in newborn lambs and pigs, and retarded growth and sparse coats in second generation rats[1-5]. Recently Se deficiency has been recognized as a common occurrence in premature infants. Premature infants have diminished hepatic Se stores compared to full-term infants and their plasma Se concentrations decrease dramatically in intensive care unit due to low supplies of Se in their diets[6][7]. However, the clinical significance of low Se status in premature infants has not been well investigated[8-10].

The lungs of many newborns, including men and rats, are not fully developed at birth. Approximately 80−90% of pulmonary alveolarization occurs during neonatal period[11]. Meanwhile, the increase in pulmonary surfactant phospholipid starts during late gestational period[12]. Therefore, Avery and Mead[13] have suggested a surfactant deficiency as one of the main reasons that respiratory distress syndrome(RDS) frequently develops in premature infants. The administration of

Accepted : October 21, 1994

exogenous surfactant to the infant is known to improve the course of the disease and decrease the oxygen requirement[14)15)].

Surfactant is produced by alveolar type II epithelial cell and increases the compliance of the lung by decreasing the surface tension of alveolar surface[16)]. About 80−90% of the surfactant is comprised of phospholipids, of which phosphatidyl choline(PC) is the primary component[17)]. Approximately 50−60% of the PC is fully saturated, mainly with palmitic acid[18)]. This disaturated phosphatidyl choline(DSPC) is the major surface-active component, and the decrease in DSPC/PC ratio is related with increase in surface tension of the alveoli[19)20)].

Adequate supply of dietary Se during pregnancy and lactation is critical in protecting neonatal rat pups from lung injuries when they are exposed to a high O_2(>95%) environment[21)22)]. The role of Se as an important antioxidant, glutathione peroxidase, has been proposed to benefit maintaining membrane integrity of the neonatal lung. However, it was also suspected that Se might have some other roles to benefit neonatal lungs. Because most studies about the effect of dietary Se on neonatal lungs were performed mainly under high O_2 environments, the role of Se under a normal environment is not known. Therefore, the present study was designed to determine the effect of Se nutrition on normal lung development. Specifically, Se concentration, GSHPx activity and DSPC/PC ratio of neonatal lungs were compared between Se-adequate and Se-deficient rats.

Materials and Methods

1. Animal care and diets

Nulliparous, female Sprague-Dawley rats(Harlan Industries, Indianapolis, IN), weighing 180-200g, were housed in individual, suspended, stainless steel wire-mesh cages in a room with controlled temperature(20−22℃) and lighting(12h light-dark cycle). Animals were fed a commercial ration(Purina Rodent Chow, Ralston Purina Co., St. Louis, MO) for a 2wk adaptation period. At 200−240g, rats were mated, and day 1 of pregnancy was determined by the presence of vaginal plugs and sperm. On day 1 of pregnancy, rats were randomly assigned to one of two experimental diets : Se-deficient or Se-adequate. Diets(Table 1) were formulated to contain all the nutrient in quantities adequate for reproduction except for

Table 1. Diet composition

Ingredient	Amount(g/kg)
Casein[1)]	200
Sucrose	300
Cornstarch	331
Corn oil	100
Cellulose[2)]	20
Vitamin mixture[3)]	10
Se-free AIN-76 mineral mixture[4)]	35
DL-methionine	2
Choline bitartrate	2

1) Vitamin-free test casein(Teklad, Madison, WI)
2) Alphacel, ICN Nutritional Biochemical Co., Cleveland, OH.
3) AIN-76 vitamin mixture.
Composition of vitamin mix(mg/kg diet) : thiamin HCl, 6.0 ; riboflavin, 6.0 ; pyridoxine HCl, 7.0 ; nicotinic acid, 30.0 ; calcium pantothenate, 16.0 ; folic acid, 2.0 ; biotin, 0.2 ; vitamin B_{12}, 0.01 ; vitamin K, 0.05 ; supplies in IU/kg diet : vitamin A, 4,000 ; vitamin D, 1,000 ; vitamin E, 50.0 ; sucrose, 9.9g.
4) AIN-76 mineral mixture prepared without Se.
Composition of mineral mix(g/kg mineral mix) : calcium phosphate, dibasic, 500.0 ; NaCl, 74.0 ; potassium citrate, monohydrate, 220.0 ; potassium sulfate, 52.0 ; magnesium oxide, 24.0 ; manganous carbonate, 3.5 ; ferric citrate, 6.0 ; zinc carbonate, 1.6 ; cupric carbonate, 0.3 ; potassium iodate, 0.01 ; chromium potassium sulfate, 0.55 ; sucrose, 118.05. Sodium selenite was added to provide 0.5ppm Se in formulation of the Se-adequate diet.

Se. Direct analysis of both diets by gas chromatography confirmed dietary Se contents to be 0.04 ppm(Se− diet) and 0.5 ppm(Se+ diet). Demineralized water(Nanopure, Barnstead, Boston MA) and experimental diets were fed ad libitum.

2. Experimental design

Female Sprague Dawley rats were bred and fed a semipurified Se-deficient(0.04 ppm, Se−) or a Se-adequate(0.5 ppm, Se+) diet through pregnancy and lactation. Two days before the expected date of delivery, dams were placed in solid-bottom maternity cages. The day after parturition(day 2), litters were weighed and culled so that there were 10 pups per dam. Extra 2-d-old pups were decapitated, and blood, lung and liver were separated and were used for biochemical analysis. Food dishes were positioned so that only dams had access to foods. Dams were permitted to nurse their young until day 16 of lactation. The numbers of dams used in the experiment were five per group.

On d 16 of lactation, dams and pups were separated for 2 h to allow for accumulation of milk. Dams were anesthetized with ketamine HCl(10 mg/100g BW) containing acepromazine(1mg/100 g BW) and injected intraperitoneally with oxytocin(0.25U/100g BW) to facilitate milking. Milk was collected with a suction apparatus in which milk flowed through polyethylene capillary tubing attached to a test tube kept in an ice bath. Both dams and 16-d-old pups were weighed, killed, and heparinized blood, lung and liver were collected. The lung was carefully perfused through the pulmonary artery with ice-cold isotonic buffer(0.1M potassium phosphate, 0.15M KCl, pH7.4) before removal.

3. Analytical procedures

Se concentrations in milk and in lung, plasma, and liver of dams and pups were determined according to the method of McCarthy, et al[23] using a gas chromatograph equipped with an electron capture detector(Hewlett-Packard 5710A, Avondale, PA) and a 0.53mm ID fused silica capillary column(Supelco, Bellefonte, PA). Activities of GSHPx were determined in blood and tissue homogenates by the modified, coupled assay of Paglia and Valentine[24]. Hydrogen peroxide was used as the substrate in the assay. Protein contents of tissues and blood were determined by a modified Lowry method[25].

Lungs from 16-d-old pups were used for phospholipid measurements. Lipids were extracted using chloroform : methanol(2 : 1, v/v)[26]. Individual phospholipids were separated by the thin-layer chromatopraphy method of Gilfillan, et al[27] using chloroform : methanol : petroleum ether : acetic acid : boric acid(40 : 20 : 30 : 10 : 1.8, v/v /v/v/w) as the developing solvent. Lipids were visualized on the plate by iodine vapor and identified by comparison with known phospholipids standards. After removal of the other phospholipids fractions from the chromatographic plate, the remaining phosphatidylcholine(PC) was treated directly with 5% osmium tetroxide to separate the disaturated phosphatidylcholine (DSPC) and unsaturated phosphatidylcholine (USPC) by using chloroform : methanol : ammonium hydroxide(80 : 28 : 6, v/v/v) as the developing solvent. Phospholipid concentration in the separated fractions was then determined by measuring phosphorus according to the method of Barlett[28].

4. Statistics

Data were evaluated using a student t test. A probability value of $\alpha=0.05$ was chosen as the level of statistical significance[29][30].

Results

1. Body weights and food intakes

Dietary Se concentration had no effects on food consumption or body weight of dams during reproduction and lactation. The mean body weights (g) and food intakes(g/d) of dams were 344.0± 6.3 and 20.8± 0.8, respectively, at the end of pregnancy, and 263.0± 3.3 and 34.0± 0.8 at d 16 of lactation. The mean body weight(g) of pups also was not affected by maternal dietary Se. The mean body weights of pups on d 2 and d 16 were 6.13± 0.14 and 33.1± 0.8, respectively.

2. Se concentrations and GSHPx activities of 2-d-old pups

Se concentrations and GSHPx activities in plasma, lung and liver of 2-d-old pups are listed in Table 2. No differences in pulmonary Se concentration and GSHPx activity were found between Se− and Se+ pups. Plasma and liver Se concentrations and GSHPx activities also were not much affected by maternal Se intake during pregnancy, and significant difference was only found in plasma GSHPx activity.

Table 2. Plasma, lung and liver selenium concentrations and glutathione peroxidase(GSHPx) activities of d2 pups

	Experimental group	
	Se−	Se+
Se concentrations(ng/g)		
Plasma	106.82 ± 15.60[1]	114.34 ± 16.28
Lung	138.08 ± 9.75	161.92 ± 21.65
Liver	210.34 ± 38.29	255.24 ± 18.49
GSHPx activities(U)[2]		
Plasma	22.14 ± 4.21	47.07 ± 15.73*
Lung	85.93 ± 0.35	86.07 ± 4.37
Liver	44.75 ± 2.58	66.28 ± 12.36

1) Mean ± SEM

2) U=μmol NADPH disappearance/min/g protein.

*Significantly different from Se− group by student t-test at $\alpha=0.05$ level

3. Se concentrations in milk, and dam and pup tissues on d 16 of lactation

Dietary Se concentration significantly affected lung, plasma and liver Se concentrations of dams

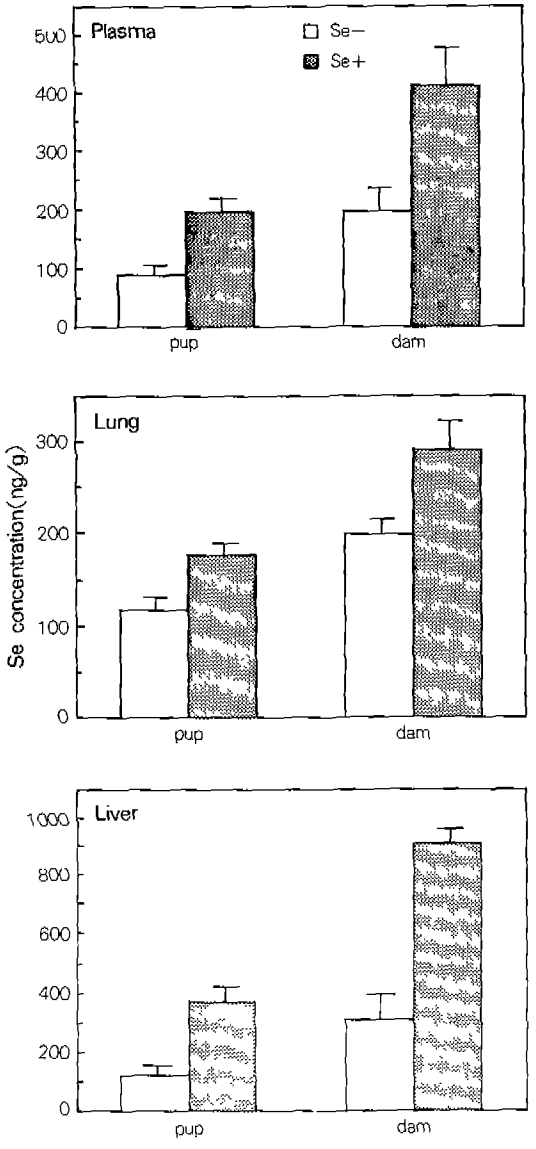

Fig. 1. Selenium concentrations in plasma, lung and liver of 16d old pups and their dams. Asterisk denotes significant difference from Se− group by student t-test at $\alpha=0.05$ level.

on d 16 of lactation(Fig. 1). The organ most significantly affected by dietary Se was the liver, followed by the plasma and the lung. The mean concentrations of Se in liver, plasma and lung from Se− dams were 34%, 47% and 68% of those in Se+ dams. Maternal dietary Se concentration also significantly affected milk Se concentration. The mean milk Se concentration(ng/ml) of Se- dams was 48.26± 8.34, as compared to 85.07± 11. 86 in Se+ dams($p<0.01$).

The significant difference in milk Se concentration was reflected on the accumulation of Se in pup organs during lactation. That is, on d 16, plasma, lung and liver Se concentrations of Se+ pups were significantly higher than those of Se− pups. The mean liver Se concentration of Se− pups was 67% lower than that of Se+ pups. Lung and plasma Se concentrations of Se− pups were 33% and 54% lower than those of Se+ pups, respectively.

4. GSHPx activities of dam and 16-d-old pup tissues

The activities of glutathione peroxidase in lung, plasma and liver of lactating dams and pups are shown in Fig. 2. The Se concentrations of dam and pup tissues were reflected on GSHPx activities of those tissues. Differences in tissue GSHPx activities were most dramatic in the liver, in which Se− dams and pups had 28% and 10% of the activities of Se+ dams and pups, respectively. Meanwhile, the lung and plasma GSHPx activities of Se− groups were 43−65% of those in Se+ groups.

5. Pulmonary phospholipid content of d 16 pups

Total phospholipid contents(μmol/g) in the lung were similar between Se− and Se+ pups on d 16, and were 21.92± 0.36 and 20.52± 1.07, respectively. Total phosphatidyl choline(PC) co-

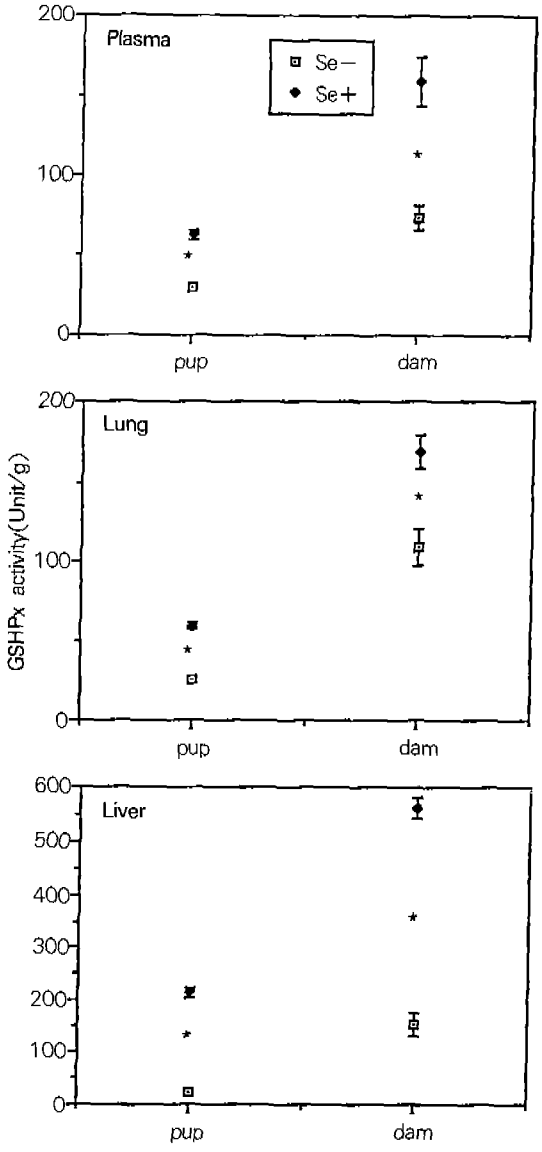

Fig. 2. Glutathione peroxidase activities in plasma, lung and liver of 16 day old pups and their dams. Asterisk denotes significant difference between Se− group and Se+ group by student t-test at $\alpha=0.05$ level.

ntents also were not different between Se− and Se+ groups and constituted 44.9% of total phospholipids in Se− rat lungs and 47.7% in Se+ rat lungs. However, the disaturated portion (DSPC) of the phosphatidyl choline, known as

Table 3. Percent distribution of phospholipids in lung tissue of 16 day old pups

Phospholipid(%)	Se−	Se+
Lysophosphatidylcholine	3.14 ± 1.43[1)]	1.53 ± 0.13
Sphingomyelin	8.54 ± 0.95	9.19 ± 0.93
Phosphatidylinositol	6.60 ± 0.53	6.89 ± 0.41
Phosphatidylserine	9.27 ± 2.08	7.23 ± 0.79
Phosphatidylethanolamine	19.24 ± 2.18	19.14 ± 1.16
Phosphatidylglycerol	6.74 ± 1.01	5.90 ± 1.61
Cardiolipin	1.61 ± 0.34	2.27 ± 0.64
Total phosphatidylcholine	44.87 ± 2.72	47.80 ± 2.39
Disaturated phosphatidylcholine	20.83 ± 1.25	25.70 ± 1.47*
Unsaturated phosphatidylcholine	24.04 ± 1.58	22.10 ± 1.51
DSPC/Total PC	46.5 ± 0.9	53.8 ± 1.8*

1) Mean ± SEM

*Significantly different from Se-deficient pups by student t-test at $\alpha=0.05$ level.

the major surface-active component of surfactant, was significantly lower in Se− pups compared to Se+ pups(Table 3).

Discussion

In this study, lung, plasma and liver Se concentrations of 2-d-old pups were not severely affected by maternal dietary Se. These findings suggest that transfer of Se to the fetus is well controlled during gestational period. Other investigators have also observed in the dog and cattle that Se readily crosses the placenta with low Se concentrations in the dams[31)32)]. In contrary, dietary Se significantly affected lung, liver and plasma Se concentrations of both dams and pups on day 16. These data suggest that the requirement of dietary Se significantly increases during lactation. Dietary Se directly affects milk Se concentration and the result is reflected on the Se contents and GSHPx activities of pup tissues. That is, even though Se nutritions during pregnancy and lactation both affect the accumulation of Se in pup tissues, the effect is more dramatic during lactational period[33)34)].

The effects of dietary Se on the accumulation of Se in pup tissues are different by the tissue[35)]. Liver and kidney Se concentrations have been reported to be severcly affected by dietary Se. However, heart has less labile pool of Se and is not affected easily by dietary Se deficiency[36)]. In this study, pulmonary Se concentration under normal environment has been measured for the first time and found to be affected much by dietary Se. Meanwhile it has been reported that pulmonary Se is well conserved under a high O_2 environment [21)22)]. That is probably due to an increase in the requirement of antioxidant, glutathione peroxidase, under a high oxygen stress.

The data from this study also suggest that marginal Se deficiency affects pulmonary phospholipid composition and decreases the ratio of DSPC/PC. This abnormal change in Se− group may predispose the animal to a severe pulmonary disease under a high stress condition. Because the production of pulmonary surfactant decreases [37)] and the requirement of glutathione peroxidase increases under a high oxygen environment[21)22)], Se-deficient rats may get lung lesions more easily compared to Se-adequate rats.

The mechanism by which Se deficiency decreases DSPC/PC ratio has not been known, but one

possibility may be an altered thyroid hormone metabolism. Thyroid hormones have positive effects on pulmonary surfactant synthesis and secretion[38]. T_3(3,3'-6-tri-iodothyronine) is more active for pulmonary phospholipid production than T_4(thyroxine). Recently it has been reported that type I iodothyronine 5'-deiodinase, the enzyme which converts thyroxine into T_3, is a selenium dependent enzyme. In addition, the activity of this enzyme in the rat liver significantly decreases during Se deficiency[39)40)]. Therefore, it is suggested that a significantly lower liver Se concentration of Se− pups compared to Se+ pups, shown in this study, might have led to a low production of iodothyronine deiodinase in Se− pup liver, and therefore to an altered pulmonary surfactant production.

Premature infants are frequently born with Se-deficiency. They easily get respiratory distress syndrome, because of the surfactant deficiency and less developed pulmonary systems[9)10)12)13)]. Because Se deficiency is related with impaired pulmonary surfactant function and low GSHPx activity, providing adequate Se to the premature infant may have beneficial effects. Recently Huston et al[3)] have reported that the supplementation of Se to the premature infant has shortened the treatment time of the infant in a high oxygen incubator. However, more studies are necessary to generalize the Se supplementation to the premature infant. Because dietary Se has a very narrow safety range, a special care is also necessary while it is supplemented.

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= 국문초록 =

임신·수유기의 셀레늄 영양이 어린쥐 폐의 셀레늄 농도, Glutathione Peroxidase 활성, 그리고 인지질 조성에 미치는 영향

김 혜 영
용인대학교 식품영양학과

본 연구는 임신·수유기동안의 셀레늄 영양이 어린쥐의 폐에 미치는 영향, 특히 폐의 셀레늄 농도와 glutathione peroxidase(GSHPx) 활성 정도 및 인지질의 조성에 미치는 영향을 살펴보고자 시행되었다. 어미 쥐의 식이 셀레늄은 새끼 쥐 폐의 셀레늄 축적에 큰 영향을 주는 것으로 나타났는 데, 이는 특히 임신기보다 수유기간 동안에 현저한 것으로 나타났다. 셀레늄이 결핍된 식이(0.04ppm)를 섭취한 군은 식이 셀레늄이 충분했던(0.5ppm) 군보다 수유 16일째의 모유의 셀레늄 농도가 훨씬 낮았으며, 이는 새끼 쥐 폐의 셀레늄 축적에 반영이 되어서 셀레늄이 부족한 모유로 자란 새끼 쥐들의 경우 셀레늄이 충분했던 쥐들보다 폐의 셀레늄 농도가 현저히 낮았다. 새끼 쥐 폐의 셀레늄 농도는 중요한 항산화제로 작용하는 GSHPx의 활성 정도에도 직접적인 영향을 미쳤으며, 폐포(alveoli)의 표면 장력을 낮추어서 원활한 기능을 하도록 돕는 인지질의 조성에도 영향을 주었다. 폐에서의 셀레늄의 이와 같은 역할들은 충분한 셀레늄 영양이 고산소 환경하에서의 폐의 손상을 막는 데에 도움이 되는 것과 밀접한 연관이 있는 것으로 사료된다.