# 子牛白筋症におけるトコフェロール,セレニウム値および血液グルタチオンペルオキシダーゼ活性値について



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# Studies on Serum Tocopherol, Selenium Levels and Blood Glutathione Peroxidase Activities in Calves with White Muscle Disease

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ABSTRACT. For the purpose of clarifying the cause of white muscle disease (WMD) in calves, tocopherol and selenium levels and blood glutathione peroxidase (GSH-Px) activity were measured on 10 calves with WMD and nine of their dams. The main clinical symptoms of the 10 calves with WMD were motor disturbances including recumbency and stiffness. Serum enzyme activities (GOT, GPT, CPK, LDH) in calves with WMD increased markedly, and this increase was also observed in some of their dams. Serum tocopherol levels of calves with WMD were low, 70% of which showing deficient levels of less than 70 μg/100m*l*. Serum selenium levels of all the calves were lower than 35 ppb, indicating a deficiency, and were accompanied by low blood GSH-Px activity. α-Tocopherol and selenium concentrations in organs were very low. Dams of calves with WMD showed low serum tocopherol levels, 22% of which indicating deficient levels below 150 μg/100m*l*. Serum selenium levels in dams showed a marked decrease to under 20 ppb, and also low blood GSH-Px activity. Feedstuffs supplied in the farms to affected calves indicated very low α-tocopherol contents (below 3 mg/100g DM) and low selenium concentrations below 50 ppb in DM. It was concluded that WMD in calves was attributable to nutritional muscular dystrophy caused by deficiencies in tocopherol and selenium in feedstuffs supplied to their dams.—KEY WORDS: calf, selenium, tocopherol, white muscle disease.

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There have been numerous reports describing that white muscle disease (WMD) in calves is a nutritional degeneration of skeletal and cardiac muscle caused by deficiencies in tocopherol and selenium [7, 14, 18, 20, 21, 26, 27]. Since Ichijo *et al.* in 1981 reported the first case of WMD of calves in Japan, suggesting that WMD was due to deficiencies in tocopherol and selenium [11], other workers have reported the disease [30, 31]. In Hokkaido, the low selenium content of the soil [6] and the decrease in tocopherol content by oxidation of feedstuffs in winter [10, 11, 32] can be considered to account for the outbreak of WMD in calves.

In this study, for the purpose of clarifying the cause of WMD in calves in Japan, the tocopherol and selenium concentrations in affected beef calves and their dams were determined.

## MATERIALS AND METHODS

Ten calves of the Japanese Black Cattle with WMD, 38 to 130 days of age, and 9 of their dams (excepting the dam of case No. 2) from the Tokachi and Kitami Districts in Hokkaido were used for this study. The study was conducted during the housing period from winter to spring of January 1984 to June 1985 (Table 1). Five Holstein cows from the Tokachi District and 15 Holstein cows from Gunma Prefecture were prepared as controls for the examinatioin of serum selenium levels. Three Holstein calves from the Tokachi District were also examined for the determination of α-tocopherol and selenium levels in their organs. In order to study the relationship between serum selenium levels and blood glutathione peroxidase (GSH-Px) activity,

Table 1. Calves with white muscle disease and their dams examined

| Case No. | Farm | District | Date | Breed | Calves with WMD[a)] Age (days) | Sex | Dams |
|---|---|---|---|---|---|---|---|
| 1 | A | Tokachi | 1984.1 | Japanese black | 42 | Female | ○ |
| 2 | B | Kitami | 1984.2 | Japanese black | 40 | Female | NT[b)] |
| 3 | C | Tokachi | 1984.3 | Japanese black | 47 | Female | ○ |
| 4 | D | Tokachi | 1984.4 | Japanese black | 38 | Female | ○ |
| 5 | E | Tokachi | 1984.5 | Japanese black | 41 | Female | ○ |
| 6 | F | Tokachi | 1984.6 | Japanese black | 50 | Male | ○ |
| 7 | G | Tokachi | 1984.6 | Japanese black | 60 | Female | ○ |
| 8 | H | Tokachi | 1984.8 | Japanese black | 90 | Male | ○ |
| 9 | I | Tokachi | 1985.2 | Japanese black | 130 | Female | ○ |
| 10 | J | Tokachi | 1985.6 | Japanese black | 60 | Female | ○ |

a) White muscle disease.
b) Not tested.

Table 2. Cattle examined for control

| District | Breed | Number of cattle examined Cow | Calf | Heifer | Items examined |
|---|---|---|---|---|---|
| Tokachi | Holstein | 5 | | | Serum selenium levels |
| Gunma prefecture | Holstein | 15 | | | |
| Tokachi | Holstein | | 3 | | α-Tocopherol and selenium levels in organs |
| Tokachi | Japanese black | 18 | 27 | | Serum selenium levels and blood GSH-Px activities |
| Tokachi | Aberdeen Angus | | 25 | | |
| Tokachi | Holstein | | 8 | 15 | |

93 cattle (18 Japanese Black cows, 27 Japanese Black calves, 25 Aberdeen Angus calves, 8 Holstein calves and 15 Holstein heifers), which had been kept under various feed systems, were also examined (Table 2).

Clinical findings including serum enzyme activities (GOT, GPT, CPK, LDH) in calves with WMD and their dams were examined. Serum tocopherol and selenium levels were measured by fluorometric analysis [1] and Olson's fluorometric method [22]. Blood GSH-Px activity was determined by the method of Paglia & Valentine [23]. Contents of α-tocopherol and selenium in feedstuffs and organs were measured by high performance liquid chromatography [13] and Olson's method [22], respectively.

For pathological examination, 3 calves (case Nos. 7, 8, 9) which did not recover were euthanized, necropsied and then examined microscopically using hematoxylin-eosin stain.

## RESULTS

*Clinical and pathological findings in calves with WMD*: All 10 calves with WMD showed motor disturbances such as recumbency and stiffness. Six cases of tachycardia and anorexia, 4 cases of diarrhea and 5 cases of skeletal muscle hardening were also observed. The temperatures of all 10 calves were normal. Eight of the 10 calves were injected intramuscularly with 200–300 mg/day of dl-α-tocopherol acetate or 2–3m*l*/day of E-Se (containing 2.5 mg Se and 68 mg dl-α-tocopherol acetate/m*l*, Burns, USA) for a period of 2 to 3 days. After the

Table 3. Serum enzyme activities of calves with white muscle disease and their dams

| Case No. | Calves | | | | Dams | | | |
|---|---|---|---|---|---|---|---|---|
| | GOT (KU)[a] | GPT (KU) | CPK (IU)[b] | LDH (WU)[c] | GOT (KU) | GPT (KU) | CPK (IU) | LDH (WU) |
| 1 | 17,996 | 970 | 38,870 | 60,590 | 87 | 17 | 20 | 2,284 |
| 2 | 1,360 | 320 | 12,500 | 15,650 | NT[d] | NT | NT | NT |
| 3 | 7,000 | 2,200 | 66,960 | 46,000 | 70 | 31 | 12 | 1,811 |
| 4 | 4,672 | 591 | 49,164 | 31,600 | 64 | 28 | 111 | 1,445 |
| 5 | 2,107 | 1,074 | 2,898 | 12,310 | 61 | 23 | 8 | 1,442 |
| 6 | 3,229 | 1,485 | 21,180 | 18,710 | 122 | 33 | 308 | 1,897 |
| 7 | 2,250 | 650 | 45,275 | 19,480 | 60 | 18 | 71 | 1,959 |
| 8 | 3,018 | 346 | 46,490 | 23,620 | 75 | 17 | 129 | 2,165 |
| 9 | 14,450 | 2,452 | 95,040 | 70,900 | 120 | 24 | 538 | 4,709 |
| 10 | 6,675 | 862 | 93,690 | 42,390 | 55 | 31 | 85 | 1,964 |
| M | 6,276 | 1,095 | 47,207 | 34,125 | 79 | 25 | 142 | 2,085 |
| ±SD | ±5,628 | ± 738 | ±31,201 | ±20,133 | ±25 | ± 6 | ±174 | ±1,058 |

a) Karmen Unit. b) International Unit. c) Wroblewski Unit. d) Not tested.

treatment 7 of the 8 calves recovered within 2 to 5 days, but case No. 9 did not recover and was euthanized. In the other 2 cases treated with fluid transfusions and antibiotics, case No. 7 died and case No. 8 was euthanized. These 3 calves which had not recovered were subsequently necropsied.

By the necrospy, lesions were found to be limited to the skeletal muscles of the whole body at sites where the muscle fibers had a pale color. Microscopically, severe degeneration of muscle fibers with swelling, fragmentatioin, hyalinosis, granulation and vacuolation were observed as lesions of the skeletal muscles. These degenerative changes of the muscles were also found in the diaphragm and tongue.

*Serum enzyme activities*: At the first examination, 10 calves affected with WMD showed marked increases in serum enzyme activity (Table 3); 6,276±5,628 Karmen unit (KU) (range of 1,360 to 17,996 KU) in GOT, 1,095±738 KU (320–2,452 KU) in GPT, 47,207±31,201 International unit (IU) (2,898–95,040 IU) in CPK, and 34,125±20,133 Wroblewski unit (WU) (12,390–70,900 WU) in LDH.

Four of the 9 dams of calves affected with WMD showed over 100 IU of CPK activity, especially two of which (22%) over 300 IU. One of these dams had over 2,500 WU in LDH with normal GOT and GPT activities.

*Serum tocopherol, selenium levels and blood GSH-Px activity*: The mean serum tocopherol concentration of the 10 affected calves was low levels, 72.1±50.3 μg/100m*l* (29.4–186.5 μg/100m*l*), in particular, 7 of which (70%) showing extremely low levels below 70 μg/100m*l*. The mean serum selenium level of the affected calves was 13.1±7.8 ppb (4.1–31.8 ppb), which were below 35 ppb, and 9 of which showed lower than 20 ppb. Similarly, the mean blood GSH-Px activity of the affected calves was also low with an average of 12.4±2.3 e.u (10.3–15.6 e.u) (Table 4). Serum tocopherol, selenium concentrations and blood GSH-Px activity of those calves which recovered due to dl-α-tocopherol and E-Se injections were high more than 100 μg/100m*l*, 35 ppb and 30 e.u, respectively. In contrast, serum selenium concentration of control Holstein calves from the Tokachi District was higher than that of the affected calves with an average of 56.3±12.8 ppb (42.1–77.2 ppb), and blood GSH-Px activity

Table 4. Serum tocopherol and selenium levels and blood GSH-Px activities of calves with white muscle disease and their dams

| Case No. | Calves | | | Dams | | |
|---|---|---|---|---|---|---|
| | Tocopherol (μg/100ml) | Selenium (ppb) | GSH-Px (e.u) | Tocopherol (μg/100ml) | Selenium (ppb) | GSH-Px (e.u) |
| 1 | 29.4 | 15.8 | NT[a)] | 113.4 | 12.9 | NT |
| 2 | 65.2 | 31.8 | NT | NT | NT | NT |
| 3 | 125.9 | 9.3 | NT | 155.9 | 9.6 | NT |
| 4 | 186.5 | 8.9 | NT | 155.0 | 9.7 | NT |
| 5 | 41.8 | 12.4 | 13.9 | 205.9 | 7.9 | 12.8 |
| 6 | 33.6 | 14.0 | 15.6 | 283.9 | 13.1 | 13.1 |
| 7 | 38.1 | 18.0 | NT | 228.7 | 10.0 | NT |
| 8 | 69.3 | 7.0 | 10.6 | 295.7 | 7.4 | 10.7 |
| 9 | 40.0 | 9.6 | 10.3 | 203.1 | 10.3 | 13.7 |
| 10 | 90.0 | 4.1 | 11.5 | 127.3 | 10.8 | 10.4 |
| M | 72.1 | 13.1 | 12.4 | 196.5 | 10.1 | 12.1 |
| ±SD | ±50.3 | ± 7.8 | ± 2.3 | ± 64.9 | ± 1.9 | ± 1.5 |

a) Not tested.

Table 5. Serum selenium levels and blood GSH-Px activities of normal Holstein cows and calves in Gunma prefecture and Tokachi district

| | | Cow | | Calf |
|---|---|---|---|---|
| | | Gunma prefect. (N=15) | Tokachi district (N=5) | Tokachi district (N=8) |
| Selenium (ppb) | M±SD | 66.7±10.9 | 64.2± 6.5 | 56.3±12.8 |
| | Range | 53.0−93.0 | 56.5−70.5 | 42.1−77.2 |
| GSH-Px (e.u) | M±SD | | | 47.8± 6.7 |
| | Range | | | 33.6−56.8 |

averaged 47.8±6.7 e.u (33.6–56.8 e.u) (Table 5).

The mean serum tocopherol concentration of the dams was 196.5±64.9 μg/100m*l* (113.4–295.7 μg/100m*l*), but 2 dams of them (22%) showed below 150 μg/100m*l*. The serum selenium levels of the 9 dams were very low, under 20 ppb, with an average of 10.1±1.9 ppb (7.4–13.1 ppb) (Table 4). In comparison, those of control Holstein cows from Gunma Prefecture and the Tokachi District were higher with an average of 66.7±10.9 ppb (53.0–93.0 ppb) and 64.2±6.5 ppb (56.6–70.5 ppb), respectively (Table 5). Five of the 9 dams also showed low blood GSH-Px activity with an average of 12.1±1.5 e.u (10.4–13.7 e.u).

The relationship between serum selenium concentration and blood GSH-Px activity was examined in 103 cattle, which consisted of 93 cattle which had been fed with various feed systems in Tokachi, 5 calves affected with WMD, and 5 of their dams. There was a significant correlation between serum selenium concentration (X) and blood GSH-Px activity (Y) ($r=0.81$, $Y=0.686X+5.37$, $p<0.01$).

*α-Tocopherol and selenium contents in organs*: α-Tocopherol and selenium contents in the heart, skeletal muscle (M. longissimus cervicis), liver, spleen, lungs, adrenal glands, and kidneys of 2 affected calves (case Nos. 8, 9) and 3 control Holstein calves were measured (Table 6).

Table 6. α-Tocopherol and selenium levels in organs of calves with white muscle disease and healthy Holstein calves

| Organs | α-Tocopherol (μg/g) | | Selenium (ppb) | | |
|---|---|---|---|---|---|
| | Case No.8 | Normal calves (N=3) | Case No.8 | Case No.9 | Normal calves (N=3) |
| Heart | 0.97 | 1.79±1.40 | 24.0 | 55.0 | 126.5±32.2 |
| Skeletal muscle[a)] | 0.13 | 1.15±0.56 | 24.9 | 32.0 | 47.4±13.3 |
| Liver | 0.82 | 2.15±0.59 | 33.9 | 59.9 | 219.2±72.4 |
| Spleen | 0.33 | 1.49±1.00 | 64.9 | 111.2 | 206.2±34.7 |
| Lung | 0.56 | 2.01±0.84 | 23.3 | 58.3 | 114.6±35.7 |
| Adrenal gland | 0.99 | 5.53±0.67 | NT[b)] | 128.0 | 206.2±34.5 |
| Kidney | 0.81 | 2.05±2.09 | | | |
| Kidney medulla | | | 91.0 | 123.5 | 220.1±33.1 |
| Kidney cortex | | | 495.0 | 869.7 | 656.0±57.0 |

a) M. longissimus cervicis. b) Not tested.

α-Tocopherol contents in the organs of case No. 8, which had not been administered with tocopherol, were much lower than those of the control calves. The selenium contents in the organs of the 2 affected calves were extremely low in comparison with the control calves, especially in the liver.

*α-Tocopherol and selenium contents in feed*: The α-Tocopherol and selenium contents in feedstuffs supplied to dams in the 10 farms are shown in Table 7. The feed supplied mainly consisted of roughage such as hay, bean stalks, beet pulp, rice straw and corn silage. The α-tocopherol content of feedstuffs was low, with a range of 0.4 to 3.9 mg/100g dry matter (DM), with the exception of the hay found at 2 farms showing less than 3 mg/100g DM. The selenium contents of feedstuffs ranged from 9.0 to 104.9 ppb in DM. Excepting the hay of farm B, the feed selenium contents were below 100 ppb in DM with the greater part of the samples having extremely low levels under 50 ppb in DM.

Table 7. α-Tocopherol and selenium concentration in feedstuffs of the farms with white muscle disease

| Farm | Diet | α-Tocopherol (mg/100g DM) | Selenium (ppb in DM) |
|---|---|---|---|
| A | Hay | 2.1 | 48.4 |
| B | Hay | 1.0 | 104.9 |
| | Paddy straw | 2.3 | 72.3 |
| | Bean hulls | 0.6 | 17.1 |
| C | Hay | 2.5 | 17.4 |
| | Corn silage | 1.1 | 33.0 |
| D | Hay | 1.2 | 34.7 |
| | Beat pulp | 1.0 | 27.6 |
| E | Hay | 1.6 | 9.0 |
| F | Hay | 2.2 | 22.9 |
| G | Hay | 3.8 | 30.4 |
| H | Hay | 3.9 | 40.9 |
| I | Hay | 1.1 | 13.2 |
| | Corn silage | 0.3 | 36.9 |
| | Bean hulls | 2.0 | 22.9 |
| J | Hay | 0.7 | 41.8 |
| | Bean hulls | 0.6 | 38.0 |

## DISCUSSION

The reports about the cause of WMD in calves have varied, having attributed it to tocopherol deficiency [7, 14, 16], selenium deficiency [21] or both [27, 33]. In Japan, low levels of tocopherol have been reported, but selenium levels have not been examined [11, 12, 30, 31]. In this study, the tocopherol and selenium levels in 10 WMD calves with clinical symptoms of recumbency, stiffness and remarkable increases in serum enzyme activities (GOT, GPT, CPK, LDH) by severe myodegeneration were discussed.

Some researches have been reported on the normal blood selenium concentration in cattle [4, 15, 24, 29, 33]. Although the results have not been in complete agreement, the lowest limit of normal serum selenium levels have generally been considered to be about 35 ppb [29]. It was thought to be remarkable low value that the mean serum selenium level in the 10 WMD calves was 13.1±7.8 ppb. All of the calves showed deficient levels below 35 ppb, 9 of which having extremely significant low levels under 20 ppb. Selenium contents in the organs of case Nos. 8 and 9 were also very low compared with control calves, confirming that there was a selenium deficiency.

Selenium is an essential component of GSH-Px, an enzyme which plays a role in the protection of cellular membranes from oxidative damage [25]. And this study revealed a significant correlation between serum selenium levels and blood GSH-Px activity. Accordingly, the measurement of blood GSH-Px activity in cattle can be considered to be a useful screening test for serum selenium levels, because the direct measurement of serum selenium levels is very complicated.

The predominant role of tocopherol which interacts with GSH-Px appears to be as an antioxidant. It functions for preventing lipid oxidation and for prolonging the biologic life of polyunsaturated fatty acids (PUFAs). This action has been equated with stabilization of cell membranes, since PUFAs are important membrane constituents. Sharman [26] indicated that the level of serum tocopherol deficiencies in calves is under 70 $\mu$g/100m*l*. In this study, the serum tocopherol levels of the calves with WMD were low with an average of 72.1±50.3 $\mu$g/100m*l*, and 70% of the calves showed deficient levels below 70 $\mu$g/100m*l*. $\alpha$-Tocopherol level in the organs of case No. 8 was also found to be very low. These results confirm the deficiency in the selenium and tocopherol concentrations in the affected calves.

Because almost all of the affected calves in this study were still suckling, it was expected that their selenium and tocopherol levels would depend on those of their dams. Therefore, the selenium and tocopherol levels in the serum of 9 dams and in feedstuffs supplied in the farms were investigated. The mean serum selenium level of the 9 dams was 10.1±1.9 ppb, and all the dams showed extremely deficient levels under 20 ppb with 5 dams showing low blood GSH-Px activity. Asakawa *et al.* [5, 6] reported that the soil and forage in Japan have a low selenium content and that Hokkaido is a representative selenium-deficient area. The feedstuffs which had been given to the dams of the affected calves consisted of roughages produced in each farm, and the selenium content of the feedstuffs was low, being under 100 ppb in DM. In particular, most of the feedstuffs were extremely deficient in selenium below 50 ppb in DM [2, 3]. These results indicated that the selenium deficiency in the affected calves was caused by an insufficiency of selenium in the diet supplied to their dams.

The serum tocopherol levels of the dams were low averaging 196.5±64.9 $\mu$g/100m*l*, and 2 of the 9 dams (22%) had extremely low levels under 150 $\mu$g/100m*l*, which are considered to be deficient levels in adult cattle [26]. The $\alpha$-Tocopherol contents in almost all of the feedstuffs was lower than 3 mg/100 g DM, the level considered to be deficient [28]. Generally, although there is a high concentration of tocopherol in green grass and fresh hay, it has been reported that drying or long storage induce a decrease in tocopherol by its oxidation [8, 10, 11, 32]. Therefore, serum tocopherol level of cattle for the housing period was lower than that for the grazing period [17]. In this study, since the outbreak of WMD almost occurred at the end of the housing period, it was considered that the tocopherol content in the feedstuffs was reduced to very low levels.

Thus, it was concluded that the WMD in the calves described in this study resulted from the latent selenium deficiency in the dams and the marked decrease in tocopherol content of the feedstuffs during the housing period from winter to spring.

Recently, it was reported that nutritional muscular degeneration caused by deficiency in selenium and tocopherol occurred not only in calves but also in young cattle and heifers [8, 9, 10, 19, 32]. Researchers have described that the subclinical muscular dystrophy had been found in many of the calves kept in stables with the WMD calves [12, 30]. Therefore, in the present investigation, the serum enzyme activity of dams was examined and 44% of the dams was observed to have high CPK activities over 100 IU with 1 dam having over 2,500 WU in LDH. Accordingly, it was suggested that subclinical nutritional muscular dystrophy also existed in the dams of the calves with WMD and that its existence should be suspected the crisis in other cattle kept in the stables with affected calves.

REFERENCES


1. Abe, K. and Katsui, G. 1975. Spectrophotometry of serum tocopherol. *J. Jap. Soc. Nutr. Food. Sci.* 28: 277–280 (in Japanese).
2. Allen, W. M., Parr, W. H., Bradley, R., Swannack, K., Barton, C. K. Q., and Tyler, R. 1974. Loss of vitamin E in stored cereals in relation to a myopathy of yearling cattle. *Vet. Med.* 20: 373–375.
3. Ammerman, C. B. and Miller, S. M. 1975. Selenium in ruminant nutrition: A review. *J. Dairy Sci.* 58: 1561–1577.
4. Anderson, P. H., Berrett, S., and Patterson, D. S. P. 1976. Some observation on "paralytic myoglobinuria" of cattle in Britain. *Vet. Rec.* 99: 316–318.
5. Asakawa, T., Kushizaki, M., and Ishizawa, Z. 1977. Studies on distribution and changes of selenium in grassland. I. *Jap. Soc. Soil. Sci. & Plant Nutr.* 48: 287–292 (in Japanese).
6. Asakawa, T., Kushizaki, M., and Ishizawa, Z. 1977. Studies on distribution and changes of selenium in grassland. II. *Jap. Soc. Soil. Sci. & Plant Nutr.* 48: 293–296 (in Japanese).
7. Blaxter, K. L. 1953. Muscular dystrophy. *Vet. Rec.* 65: 835–838.
8. Gitter, M., Bradley, R., and Pepper, R. 1978. Nutritional myodegeneration in dairy cows. *Vet. Rec.* 103: 24–26.
9. Hunter, A. G. and Boyd, J. H. 1977. Degenerative myopathy in housed yearling bulls. *Vet. Rec.* 100: 104–106.
10. Hutchinson, L. J., Scholz, R. W., and Drake, T. R. 1982. Nutritional myodegeneration in group of Chianina heifers. *J. Amer. Vet. Med. Assoc.* 181: 581–584.
11. Ichijo, S., Hasegawa, M., Kim, D., Kimata, T., Ogawa, S., Konishi, T., Ono, T., Morishita, T., and Tanaka, I. 1981. Clinical and clinicopathological findings on leukomyoma in calves. *J. Jpn. Vet. Med. Assoc.* 34: 573–579 (in Japanese).
12. Ichijo, S., Inada, I., Osame, S., Uchida, E., Sarashina, T., Murase, T., Uchisugi, H., Miyakoshi, M., Fukawa, Y., and Tanaka, I. 1984. Clinical and clinicopathological findings on leukomyoma in calves. II. Serum chemical findings on calves kept in contaminated barns. *J. Jpn. Vet. Med. Assoc.* 37: 145–150 (in Japanese).
13. Katsui, G. 1981. Determination method of vitamin E. 4. High performance liquid chromatography. *Vitamins.* 55: 267–273 (in Japanese).
14. Keith, T. B. and Schneider, A. P. 1957. Muscular dystrophy in calves and lambs. I. The relation of environmental conditions to incidence. *J. Amer. Vet. Med. Assoc.* 131: 519–522.
15. Langlands, J. P., Wilkins, J. F., Bowles, J. E., Smith, A. J., and Webb, R. F. 1981. Selenium concentration in the blood of ruminants grazing in Northern New South Wales. I. Analysis of samples collected in the National Brucellosis Eradication Scheme. *Aust. J. Agric. Res.* 32: 511–521.
16. Maplesden, D. C. and Loosli, J. K. 1960. Nutritional muscular dystrophy in calves. II. Addition of selenium and tocopherol to a basal, dystrophogetic diet containing cod-liver oil. *J. Dairy Sci.* 43: 645–653.
17. Matsumoto, N., Ichijo, S., and Konishi, T. 1985. Changes of serum tocopherol in normal cattle. *J. Jpn. Vet. Med. Assoc.* 38: 170–174 (in Japanese).
18. McDonnough, L. T. 1953. Vitamin E deficiency among dairy calves fed on coconut meal. *Vet. Rec.* 65: 425–426.
19. McMurray, C. H. and McEldowney, P. K. 1977. A possible prophylaxis and model for nutritional degenerative myopathy in young cattle. *Brit. Vet. J.* 133: 535–542.
20. Muth, O. H. 1955. White muscle disease

(myopathy) in lambs and calves. I. Occurrence and nature of the disease under Oregon condition. *J. Amer. Vet. Med. Assoc.* 126: 355–361.
21. Muth, O. H. 1963. White muscle disease, a selenium-responsive myopathy. *J. Amer. Vet. Med. Assoc.* 142: 272–277.
22. Olson, O. E., Palmer, I. S., and Cary, E. E. 1975. Modification of the official fluorometric method for selenium in plants. *J. AOAC.* 58: 115–121.
23. Paglia, O. E. and Valentine, W. N. 1967. Studies on the quantitative and qualitative characterization of erythrocyte glutathione peroxidase. *J. Lab. Clin. Med.* 70: 158–169.
24. Perry, T. W., Caldwell, D. M., and Peterson, R. C. 1976. Selenium content of feeds and effect of dietary selenium on hair and blood serum. *J. Dairy Sci.* 59: 760–763.
25. Rotruck, J. T., Pope, A. L., Ganther, H. E., Swanson, A. B., Hafemen, D. G., and Hoekstra, W. G. 1973. Selenium: Biochemical role as a component of glutathione peroxidase. *Science* 179: 588–590.
26. Sharman, G. A. M. 1954. Muscular dystrophy of beef calves in the north of Scotland. *Vet. Rec.* 66: 275–279.
27. Sharman, G. A. M. 1959. Prevention of enzootic muscular dystrophy by selenium administration. *Vet. Rec.* 71: 536.
28. Sorensen, P. H. 1973. Basic principles involved in the supplementation of compound feeds with vitamin E. *Acta. Agric. Scand. Supple.* 19: 177–180.
29. Stevens, J. B., Olson, W. G., Kraemer, R., and Archambeau, J. 1985. Serum selenium concentrations and glutathione peroxidase activities in cattle grazing forages of various selenium concentration.*Am. J. Vet. Res.* 46: 1556–1560.
30. Takahashi, H., Koiwa, M., Yasuda, M., Ishimi, T., Nomura, T., Minami, Y., and Umeo, K. 1984. Clinical observations on white muscle disease of calves occurred collectively in a feedlot. *J. Vet. Clinic.* No. 250: 11–17 (in Japanese).
31. Takahashi, R., Ishiki, S., Hayashi, M., Miho, G., Nakagawa, H., Takekuma, T., and Satoh, K. 1984. An outbreak of white muscle disease in calves of the indigenous Japanese black breed. *J. Jpn. Vet. Med. Assoc.* 37: 779–783 (in Japanese).
32. Van Vleet, J. F., Crawley, R. R., and Amstutz, H. E. 1977. Myodegeneration associated with selenium-vitamin E deficiency in a pregnant heifer. *J. Amer. Vet. Med. Assoc.* 171: 443–445.
33. Van Vleet, J. F. 1980. Current knowledge of selenium-vitamin E deficiency in domestic animals. *J. Amer. Vet. Med. Assoc.* 176: 321–325.

## 要　約

子牛白筋症におけるトコフェロール,セレニウム値および血液グルタチオンペルオキシダーゼ活性値について：星野順彦・一条　茂・納　敏・高橋英二（帯広畜産大学家畜内科学教室）――子牛白筋症10例とその母牛９例について，とくに血清トコフェロール（Toc），セレニウム（Se）値並びに血液グルタチオンペルオキシダーゼ（GSH-Px）活性値の検討を行った．子牛白筋症の主な臨床症状では，いずれも起立困難または歩行困難などの運動障害が認められた．血清酵素活性値（GOT, GPT, CPK, LDH）は全例が著しい上昇を示し，さらに母牛においても活性値の上昇を示す例が認められた．発病子牛の血清 Toc 値は低値であり，とくに70％の例では70μg/100m*l* 以下の欠乏値を示した．血清 Se 値は全例が35 ppb 以下の欠乏値を示し，かつ血液 GSH-Px 活性値も低値であった．さらに臓器中の α-Toc と Se 濃度においても著しい低値が認められた．白筋症子牛の母牛の血清 Toc 値は低値を示し，22％が150μg/100m*l* 以下の欠乏値であった．血清 Se 値では全例が20ppb 以下の極端な欠乏値を示し，血液 GSH-Px 活性値も同様に著しい低値であった．発生農家における給与飼料中の α-Toc 含量は，ほとんどが３ mg/100 g DM 以下の著しい低値であり，Se 含量も50 ppb DM 以下の極端な欠乏値であった．以上の成績から，白筋症子牛はいずれも母牛への Toc と Se の給与不足による栄養性筋変性症と考えられた．