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EFFECTS OF VOLUNTARY RESISTANCE EXERCISE (CLIMBING) ON IRON STATUS IN SEVERELY AND MILDLY IRON DEFICIENT RATS

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Abstract

To evaluate the effects of long-term voluntary resistance exercise (climbing) compared with aerobic exercise (swimming) on iron status in severely (4 mg Fe/kg diet) and mildly (18-29 mg Fe/kg diet) iron deficient rats, we trained male Wistar rats for 8 weeks to climb a wire-mesh tower (ϕ 20 cm × 200 cm, CLIMB) and swim in a plastic pool (ϕ 50 cm × 50 cm, SWIM). These were compared to sedentary (SED) rats. After the experimental period, blood hemoglobin level, hematocrit, plasma iron concentration and transferrin saturation were significantly lower in the 4 mg Fe/kg diet fed rats than in the 40 mg Fe/kg diet fed rats for SED, SWIM and CLIMB groups (experiment 1). Hemoglobin and hematocrit levels were significantly higher in the CLIMB group than the SED group in 4 mg Fe/kg diet fed rats (experiment 1). On the other hand, neither exercise affected iron status in mildly iron deficient rats (experiment 2). Iron content in the liver, spleen, heart and kidney rdecreased with iron levels. These results suggest that long-term resistance exercise inproves iron status in severely iron deficient rats more than aerobic exercise, probably due to an increase in the capacity for heme biosynthesis. Resistance exercise may be a useful therapy for iron deficient anemia.

Key word : resistance exercise, aerobic exercise, iron deficiency, hemoglobin, heme biosynthesis, rat

Introduction

Iron deficiency continues to be a significant nutritional problem around the world.[1,2] It has deleterious effects on work performance, immune function, sympathetic and endocrinal metabolism, and thermoregulatory performance.[3-6] Although the hematologic and functional consequences of iron deficiency have been classically ascribed to dietary or pathologic origins[2], previous studies suggest that chronic exercise can detrimentally alter body iron physiology. Decreased hematocrit, hemoglobin and serum iron, and in creased erythrocyte fragility can occur with aerobic exercise.[7-10] Thus far, animal studies[11,12] examining iron deficiency and aerobic exercise interactions have demonstrated that exercise lessens the impact of moderate iron deficiency on essential body iron components, such as hemoglobin. Hisaoka and Shibuya[13] demonstrated that hemoglobin, hematocrit, and red blood cell volume were significantly higher in swimming rats than control rats. Tobin and Beard[14] reported that running failed to alter hemoglobin, hematocrit and red blood cell mass in iron deficient and control rats.

On the other hand, few studies have looked at the effects of resistance exercise on body iron status. We previously demonstrated that mild resistance exercise improved non-anemic iron deficiency without iron supplementation in young women.[15] Recently, we designed a new voluntary resistance training model, in which rats climbed a vertical tower.[16] Three weeks of climbing increased the activity of d-aminolevulinic acid dehydratase, the marker enzyme for heme biosynthesis, in the bone marrow of severely iron deficient rats, whereas blood hemoglobin concentration was not affected.[17,18] These findings suggest that 3 weeks of resistance exercise increases heme biosynthesis, but neither exercise improved severe iron deficiencies.[17,18] However, we assume that the length of the experiment and the level of iron deficiency influence the iron status.

The purpose of the present study was to determine whether resistance exercise for a longer period (8 weeks) induces improvement in essential body iron components in severely and mildly (marginal) iron deficient rats. We also compared the effectiveness of resistance versus aerobic exercise (swimming).

Materials and methods

Experiment 1 Effects of voluntary resistance exercise on iron status in severely iron deficient rats compared to aerobic exercise

Animals and experimental design

Thirty-six male Wistar rats (3 weeks old) were obtained from Japan SLC, Inc. (Shizuoka, Japan). Rats were fed CE-2, commercial rodent diet (CLEA Japan, Tokyo), and water ad libitum through age 5 weeks. All animals were individually housed in an animal room at 24±1℃, with lights on from 8 am to 8 pm. Half of the animals were as signed to a diet based on AIN-76[19,20], with 4 mg Fe/kg, and the other half were assigned to an identical diet with 40 mg Fe/kg (Table 1). Both groups of rats were ran-domly divided into three subgroups, sedentary (SED), swim ming exercise (SWIM) and climbing exercise (CLIMB). Each group (n=6/group) was meal-fed the diet at 8 : 00 to 9 : 00 h and 17 : 00 to 19 : 00 h and given free access to water for 8 weeks. After the 8-week experimental period, the rats were fasted overnight and killed by decapitation under light ether anesthesia. Liver, spleen, heart and kidney were quickly removed and stored at -40℃ until analysis.

The voluntary resistance training model, CLIMB, was a modification of that described by Yarasheski et al.[21] and Duncan et al.[22]. Rats of the CLIMB group were housed in metal cages containing wire-mesh towers (ϕ 20 cm × 200 cm) with water bottles set on the top of the tower.[16-18] On the other hand, the rats of the SWIM group were trained from 8 am to 9 am everyday in a plastic pool (ϕ 50 cm × 50 cm) with water maintained at 33-35℃. The voluntary swimming exercise was performed in the manner described previously.[23,24]

Analysis

Blood hemoglobin concentration was determined colorimetrically using a hemoglobin B-Test kit purchased from Wako Pure Chemical Industries (Osaka, Japan). Hematocrit was measured by centrifugation of blood collected into heparinized microcapillary tubes. Plasma iron concentration and total iron binding capacity (TIBC) were determined by the method of the International Nutritional Anemia Consultative Group.[25] Transferrin saturation was calculated by plasma iron concentration and TIBC.[25]

Iron content in the liver, spleen, heart and kidney were measured after acid hydrolysis using an atomic absorption spectrophotometer (Model Z-5000, Hitachi, Tokyo, Japan).

Table 1 Composition of experimental diets

	Experiment 1		Experiment 2	
Iron level (mg/kg diet)	4	40	18	29
Ingredients		g/kg diet		
Corn starch	649.8	649.8	649.8	649.8
Casein	200.0	200.0	200.0	200.0
Corn oil	50.0	50.0	50.0	50.0
Cellulose	50.0	50.0	50.0	50.0
Vitamin mixture[1]	10.0	10.0	10.0	10.0
Mineral mixture with iron[1]	0.0	35.0	12.0	23.0
Mineral mixture without iron[1]	35.0	0.0	23.0	12.0
Choline chloride	2.0	2.0	2.0	2.0
DL-Methionine	3.0	3.0	3.0	3.0
Butylated hydroxytoluene	0.2	0.2	0.2	0.2
Total	1000.0	1000.0	1000.0	1000.0

[1]Based on the AIN-76A.

Experiment 2 Effects of voluntary resistance exercise on iron status in mildly iron deficient rats compared to aerobic exercise

Animals and experimental design

Thirty-six male Wistar rats (3 weeks old) were obtained from Japan SLC, Inc. (Shizuoka, Japan). Rats were fed CE-2, commercial rodent diet (CLEA Japan, Tokyo), and water ad libitum through age 5 weeks. Half of the animals were assigned to a diet based on AIN-76 (19, 20), with 18 mg Fe/kg, and the other half were assigned to a diet with 29 mg Fe/kg (Table 1). Both diet groups were then randomly divided into SED, SWIM and CLIMB groups. Housing conditions were the same as experiment 1.

The training methods for climbing and swimming were the same as described in experiment 1.

Analysis

Blood and plasma iron status, and tissue iron content were assayed as in experiment 1.

Statistics

Data were expressed as means ± SE. All data were analyzed by two-way analysis of variance (ANOVA) and Fisher's PLSD tests. Differences were considered statistically significant at $p<0.05$.

Results

Experiment 1

Body weight, food intake and food efficiency

Swimming exercise reduced body weight gain in both 4 mg Fe/kg diet and 40 mg Fe/kg diet groups (SWIM-4 and SWIM-40 groups), though food intake was approximately the same in all experimental groups (Table 2). Food efficiency was significantly lower ($p<0.05$) in the SWIM group than in the SED and CLIMB groups for both 4 mg Fe/kg diet and 40 mg Fe/kg diet groups (Table 2).

Table 2 Effects of exercise and dietary iron level on weight gain, food intake and food efficiency in rats[1]

Group[2]	Weight gain	Food intake	Food efficiency
	(g)	(g/day)	(%)
SED-4	91±9	7.2±0.3	22.1±1.2
SWIM-4	55±7#	7.5±0.3	13.0±1.1#
CLIMB-4	107±15	8.2±0.4	21.5±1.1
SED-40	113±10	8.4±0.4	23.8±0.9
SWIM-40	70±8#	7.4±0.4	16.4±1.1*#
CLIMB-40	103±10	8.2±0.5	22.4±1.0

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
*Statistically significant difference ($p<0.05$) from 4 mg Fe/kg diet group (two-way ANOVA and Fisher's PLSD tests).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively.

Hemoglobin, hematocrit, plasma iron and transferrin saturation

Blood hemoglobin level, hematocrit, plasma iron concentration and transferrin saturation were significantly lower ($p<0.05$) in the 4 mg Fe/kg fed rats than in the 40 mg Fe/kg diet fed rats for SED, SWIM and CLIMB groups (Table 3). Hemoglobin and hematicrit levels were significantly higher ($p<0.05$) in the CLIMB-4 group than the SED-4group (Table 3). TIBC and transferrin saturation was significantly higher ($p<0.05$) in the CLIMB-4 group than the SED-4 and SWIM-4 groups (Table 3).

Table 3 Effects of exercise and dietary iron level on iron status indices in rats[1]

Group[2]	Hemoglobin	Hematocrit	Plasma iron	TIBC	Transferrin saturation
	(g/100ml)	(%)	(mg/ml)	(mg/ml)	(%)
SED-4	7.6±0.1	29.2±0.7	2.5±0.1	8.3±0.5	30.8±1.9
SWIM-4	7.8±0.2	31.1±1.0	2.7±0.1	8.5±0.7	31.9±1.8
CLIMB-4	8.4±0.5#	32.1±1.9#	2.7±0.1	6.2±0.4#	43.6±2.5#
SED-40	13.2±0.2*	46.1±0.7*	3.4±0.2*	5.1±0.3*	66.8±1.6*
SWIM-40	13.2±0.3*	45.0±0.7*	3.4±0.2*	5.5±0.2*	61.8±2.1*
CLIMB-40	12.7±0.2*	43.9±0.7*	3.4±0.1*	5.0±0.3*	68.6±2.9*

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
*Statistically significant difference ($p<0.05$) from 4 mg Fe/kg diet group (two-way ANOVA and Fisher's PLSD tests).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively.

Table 4 Effects of exercise and dietary iron level on tissue weights in rats[1]

Group[2]	Liver	Spleen	Heart	Kidney
	(g)	(mg)	(mg)	(g)
SED-4	4.4±0.3	443±42	493±25	1.1±0.1
SWIM-4	3.6±0.2	315±19#	472±19	0.9±0.1
CLIMB-4	4.1±0.2	395±28	453±25	1.1±0.1
SED-40	5.8±0.4*	422±20	504±27	1.3±0.1*
SWIM-40	4.3±0.4#	319±26#	470±23	1.0±0.1#
CLIMB-40	5.1±0.4	400±10	481±22	1.2±0.1

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
*Statistically significant difference ($p<0.05$) from 4 mg Fe/kg diet group (two-way ANOVA and Fisher's PLSD tests).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively.

Liver, spleen, heart and kidney weights

Swimming exercise lowered liver, spleen and kidney wei-ghts in 40 mg Fe/kg diet fed rats (Table 4). In the SED group, liver and kidney weights were significantly lower ($p<0.05$) in the 4 mg Fe/kg diet group than in the 40 mg Fe/kg diet group (Table 4).

Tissue iron content

Iron content in the liver, spleen, heart and kidney was significantly lower ($p<0.05$) in the 4 mg Fe/kg diet group than in the 40 mg Fe/kg group (Table 5). Heart iron content was significantly higher ($p<0.05$) in the CLIMB group than in the SED group (Table 5). In the 40 mg Fe/kg diet, swimming increased liver and spleen iron content, whereas climbing did not influence iron levels in these tissues.

Experiment 2

Body weight, food intake and food efficiency

Swimming increased food intake and reduced food efficiency in both the 18 mg Fe/kg diet and 29 mg Fe/kg diet groups, whereas body weight was approximately the same in all experimental groups (Table 6). No differences the between 18 mg Fe/kg and 29 mg Fe/kg groups were found in body weight and food intake (Table 6).

Hemoglobin, hematocrit, plasma iron and transferrin saturation

Blood hemoglobin level, hematocrit, plasma iron concentration and transferrin saturation were approximately the same in all experimental groups (Table 7).

Table 5 Effects of exercise and dietary iron level on tissue iron content in rats[1]

Group[2]	Liver	Spleen	Heart	Kidney
	(mg/g)	(mg/g)	(mg/g)	(mg/g)
SED-4	31±1	185±12	46±1	32±2
SWIM-4	32±1	185±5	49±1#	33±2
CLIMB-4	33±1	170±13	54±1	33±1
SED-40	74±2*	582±27*	68±2*	68±5*
SWIM-40	85±3*#	723±38*#	72±2*	68±6*
CLIMB-40	76±5*	520±47*	76±5*#	63±7*

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
*Statistically significant difference ($p<0.05$) from 4 mg Fe/kg diet group (two-way ANOVA and Fisher's PLSD tests).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively.

Table 6 Effects of exercise and dietary iron level on weight gain, food intake and food efficiency in rats[1]

Group[2]	Weight gain	Food intake	Food efficiency
	(g)	(g/day)	(%)
SED-18	142±6	9.9±0.3	25.5±0.5
SWIM-18	125±6	10.9±0.3	20.4±0.5#
CLIMB-18	149±9	10.1±0.5	26.4±0.6
SED-29	135±6	9.3±0.4	26.1±0.2
SWIM-29	133±6	11.2±0.2#	21.3±0.6#
CLIMB-29	143±14	10.1±0.5	25.0±1.5

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively (wo-way ANOVA and Fisher's PLSD tests).

Table 7 Effects of exercise and dietary iron level on iron status indices in rats[1]

Group[2]	Hemoglobin	Hematocrit	Plasma iron	TIBC	Transferrin saturation
	(g/100ml)	(%)	(mg/ml)	(mg/ml)	(%)
SED-18	15.1±0.2	45.5±0.8	1.7±0.2	5.2±0.6	34.3±6.9
SWIM-18	13.9±0.5	42.4±1.7	2.0±0.3	5.8±0.2	34.4±2.9
CLIMB-18	15.0±0.2	45.0±0.6	1.6±0.2	5.1±0.1	31.4±5.0
SED-29	15.0±0.1	44.9±0.6	1.8±0.1	4.7±0.4	40.2±4.2
SWIM-29	15.0±0.2	45.4±0.6	2.0±0.2	5.5±0.5	37.6±4.9
CLIMB-29	15.3±0.3	44.2±1.4	1.7±0.1	5.6±0.5	30.8±3.2

[1]Values are means±SE for 6 rats.
[2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise. Numbers indicate dietary iron levels (mg Fe/kg diet).

Table 8 Effects of exercise and dietary iron level on tissue weights in rats[1]

Group[2]	Liver	Spleen	Heart	Kidney
	(g)	(mg)	(mg)	(g)
SED-18	6.3±0.2	460±19	565±12	1.5±0.1
SWIM-18	5.9±0.2	449±16	627±28	1.4±0.1
CLIMB-18	6.4±0.3	463±24	568±29	1.4±0.1
SED-29	5.9±0.3	481±10	535±20	1.4±0.1
SWIM-29	6.9±0.1	456±12	614±17#	1.5±0.1
CLIMB-29	6.5±0.3	472±22	541±30	1.4±0.1

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively (wo-way ANOVA and Fisher's PLSD tests).

Liver, spleen, heart and kidney weights

Tissue weights were the same in all experimental groups except for heart (Table 8). Heart weight was significantly heavier ($p<0.05$) in the SWIM-29 group than the SED-29 and CLIMB-29 groups (Table 8).

Tissue iron content

Iron content in the liver and spleen was significantly lower ($p<0.05$) in the 18 mg Fe/kg diet group than in the 29 mg Fe/kg group (Table 9). Swimming lowered liver iron content in the 29 mg Fe/kg diet group and lowered

Table 9 Effects of exercise and dietary iron level on tissue iron content in rats[1]

Group[2]	Liver	Spleen	Heart	Kidney
	(mg/g)	(mg/g)	(mg/g)	(mg/g)
SED-18	64±4	400±41	76±2	55±5
SWIM-18	50±5	294±31#	72±2#	44±2
CLIMB-18	64±7	438±48	78±1	45±2
SED-29	106±5*	749±35*	81±2	55±1
SWIM-29	82±6*#	643±62*	78±2*	54±4*
CLIMB-29	96±10*	764±34*	82±1	57±3*

[1]Values are means±SE for 6 rats. [2]SED=sedentary, SWIM=swimming exercise, CLIMB=climbing exercise.
Numbers indicate dietary iron levels (mg Fe/kg diet).
*Statistically significant difference ($p<0.05$) from 18 mg Fe/kg diet group (two-way ANOVA and Fisher's PLSD tests).
#Statistically significant difference ($p<0.05$) from SED or SWIM group, respectively.

spleen and heart iron content in the 18 mg Fe/kg diet groups, whereas climbing did not influence tissue iron content in either the 18 mg Fe/kg or 29 mg Fe/kg diet groups (Table 9).

Discussion

This study demonstrates that long-term resistance exercise (CLIMB) training increases iron status indices such as blood hemoglobin level and hematocrit more effectively than aerobic exercise (SWIM) training in severely iron deficient rats. These results suggest that the capacity for heme biosynthesis may be dependent on resistant loads to the animal body. In previous studies we reported that resistance exercise increased the activity of d-aminolevulinic acid dehydratase, the marker enzyme for heme biosynthesis[26], in bone marrow of severely iron deficient rats.[17,18] Thus, increases in the activity of this enzyme in rat bone marrow have been linked to accelerated heme production.[26] Hemoglobin is dependent on heme synthesis in the bone marrow because iron-containing porphyrin constitutes the ring structure to which the hemoglobin is conjugated with apoprotein. Therefore, we hypothesized that the heme pathway would be accelerated by exercise-induced increases in hemoglobin concentration. Tobin and Beard[14] suggested that training in iron deficient animals was chracterized by a higher percentage of ^{59}Fe associated with red cells as compared to iron deficient sedentary rats within 1 hour after intravenous injection of ^{59}Fe. The present study supports these previous findings. On the other hand, neither resistance nor aerobic exercise affected the iron status indices in mild iron deficiency (18-29 mg Fe/kg diets, experiment 2). This is probably because the iron status indices were so close to those of normal rats (40 mg Fe/kg diet, experiment 1).

In this study, iron content in several tissues was significantly lower in the 4 mg Fe/kg diet goup than in the 40 mg Fe/kg group (experiment 1) and was significantly lower in the 29 mg Fe/kg group than in the 18 mg Fe/kg group (experiment 2). Since blood hemoglobin levels of mildly iron deficient rats are normal (experiment 2), 18-29 mg Fe/kg diets could cause non-anemic "marginal" iron deficiency. Borel et al.[27] reported that as dietary iron intake increased above 16 mg Fe/kg diet, liver iron concentrations steadily increased, and hemoglobin concentrations were maintained at normal levels. Siimes et al.[28] determined that a dietary iron intake of less than 25 mg Fe/kg diet resulted in hemoglobin concentrations of 12 mg/100ml or below. Our present findings agree with the results obtained by Borel[27] and Siimes.[28] On the other hand, while climbing had no influence on tissue iron content for any dietary iron level, swimming increased tissue iron content in all tissues except for kidney in the 40 mg Fe/kg diet group (experiment 1), and reduced iron content in the 18-29 mg Fe/kg diet groups (experiment 2). These mechanisms remain unknown, so further studies will be required.

In conclusion, our study demonstrates that long-term resistance exercise increases iron status in severely iron deficient rats more effectively than aerobic exercise, probably due to an increase of heme biosynthesis in rat bone marrow. These results suggest that resistance exercise may be a useful therapy for iron deficient anemia.

References

1. Beard JL, Finch CL. Iron Fortification of Food. New York, NY: Academic Press, 1985 ; 3-16
2. Finch CA, Huebers MD. Perspectives in iron metabolism. N Engl J Med 1982 ; **25** : 1520-1528.
3. Cook JD, Lynch SR. The liabilities of iron deficiency. Blood 1984 ; **68** : 803-809.
4. Dallman PR. Biochemical basis for the manifestations of iron deficiency. Annu Rev Nutr 1986 ; **6** : 13-40.
5. Dallman PR. Manifestations of iron deficiency. Semin Hematol 1982 ; **19** : 19-30.
6. Scrimshaw NS. Functional consequences of iron deficiency in human populations. J Nutr Sci Vitaminol 1984 ; **30** : 47-63.
7. Dressendorfer RH, Wade CE, Amsterdman EA. Development of a pseudoanemia in marathon runners during a 20-day road race. JAMA 1981 ; **246** : 1215-1218.
8. Ehn L, Carlmark B, Hoglund S. Iron status in athletes involved in intense physical activity. Med Sci Sprorts Exerc 1977 ; **12** : 61-64.
9. Radomski MW, Sabiston BH, Isoard P. Development of sports anemia in physically fit men after daily sustained submaximal exercise. Aviat Space Environ Med 1980 ; **51** : 41-45.
10. Wijn JF, De Jongeste JL, Monsterd W, Willebrand D. Hemoglobin, packed cell volume, and iron binding capacity of selected athletes during training. Nutr Metab 1971 ; **13** : 129-139.
11. Perkkio MV, Jansson LT, Henderson H, Refino CJ, Brooks GA, Dallman PR. Work performance in the iron deficient rat: improved endurance with exercise training. Am J physiol 1985 ; **249** : E306-E311.
12. Willis WT, Brooks GA, Henderson SA, Dallman PR. Effects of iron deficiency and training on mitochondrial enzymes in skeletal muscle. J Appl Physiol 1987 ; **62** : 2442-2446.
13. Hisaoka F, Shibuya M. Effects of training on hematological values (hemoglobin, hematocrit and plasma volume) in growing male and female rats. JJpn Soc Nutr Food Sci 1983 ; **36** : 141-149.
14. Tobin BW, Beard JL. Interactions of iron deficiency and exercise training in male Sprague-Dawley rats: ferrokinetics and hematology. J Nutr 1989 ; **119** : 1340-1347.
15. Matsuo T, Suzuki M. Dumbbell exercise improves non-anemic iron deficiency in young women without iron supplementation. Health Sci 2000 : **16** : 250-258.
16. Matsuo T, Mizushima Y, Fujie M, Suzuki M. Effect of voluntary resistance exercise on mitochondrial heme biosynthesis in rat liver. J Clin Biochem Nutr 1999 ; **27** : 151-159.
17. Matsuo T, Suzuki M. The effects of voluntary resistance exercise on bone marrow heme biosynthesis in iron deficient rats. Health Sci 2000 ; **16** : 250-258.
18. Matsuo T, Suzuki M. Resistance exercise increases the capacity of heme biosynthesis more than aerobic exercise in rats. J Clin Biochem Nutr, in press
19. American Institute of Nutrition. Report of the American Institute of Nutrition ad hoc committee on standards for nutritional studies. J Nutr 1977 ; **107** : 1340-1348.
20. American Institute of Nutrition. Second report of the ad hoc committee on standards for nutritional studies. J Nutr 1980 ; **110** : 1726.
21. Yarasheski KE, Lemon PW, Gilloteaux J. Effect of heavy resistance exercise training on muscle fiber composition in young rats. J Appl Physiol 1990 ; **69** : 434-437.
22. Duncan ND, Williams DA, Lynch GS. Adaptations in rat skeletal muscle following long-term resistance exercise training. Eur J Appl Physiol 1998 ; **77** : 372-378.
23. Saitoh S, Matsuo T, Suzuki M. The effects of a high carbohydrate diet on postprandial energy expenditure during exercise in rats. Eur J Appl Physiol 1993 ; **66** : 445-450.
24. Saitoh S, Shimomura Y, Tasaki Y, Suzuki M. Effect of short-term exercise training on muscle glycogen in resting conditions in rats fed a high fat diet. Eur J Appl Physiol 1992 ; **64** : 62-67.
25. International Nutritional Anemia Consultative Group. Measurements of iron status. Washington DC : The Nutrition Foundation Inc, 1985.
26. Finelli VN, Murthy L, Oeirano WB, Petering HG. d-Aminolevulinate dehydratase, a zinc dependent enzyme. Biochem Biophys ResComm 1974 ; **60** : 1418-1424.
27. Borel MJ, Smith SH, Brigham DE, Beard JL. The impact of varying degrees of iron nutriture on several

functional consequences of iron deficiency in rats. J Nutr 1991；121：729-736.
28. Siimes MA, Refino C, Dallman PR. Manifestation of iron deficiency at various levels of dietary iron intake. Am J Clin Nutr 1980；33：570-574.

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自発的レジスタンス運動（クライミング）が鉄欠乏および潜在性鉄欠乏ラットの生体内鉄状態に及ぼす影響

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要　　約

長期の自発的レジスタンス運動（クライミング）が正常（40mg Fe/kg diet），鉄欠乏（4 mg Fe/kg diet）および潜在性鉄欠乏（18-29mg Fe/kg diet）ラットの生体内鉄状態に及ぼす影響を，有酸素運動（遊泳）と比較して検討した．4週齢Wistar系雄ラットを3群に分け，そのうちの2群にクライミング運動および遊泳運動を8週間実施させ，残りを安静群とした．血中ヘモグロビン濃度，ヘマトクリット値，血漿鉄濃度，トランスフェリン飽和率は，運動の有無にかかわらず，正常群（40mg Fe/kg diet）に比べて鉄欠乏群（4 mg Fe/kg diet）で有意に低値であった（実験1）．また鉄欠乏ラットにおいて，血中ヘモグロビン濃度とヘマトクリット値は，安静群に比べてクライミング運動群で有意に高かった（実験1）．一方，潜在性鉄欠乏（18-29mg Fe/kg diet）ラットにおいては，いずれの運動もラットの生体内鉄状態に影響を与えなかった（実験2）．以上の結果から，長期のレジスタンス運動は有酸素運動に比べて，鉄欠乏ラットの体内鉄状態を改善することが明らかになった．これはレジスタンス運動によって，鉄欠乏ラットのヘム合成能が増加したことによるものと推察され，レジスタンス運動は鉄欠乏性貧血の治療に有効である可能性が示唆された．