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Growth-Promoting Effect of *Spirometra erinacei* (Rudolphi, 1819) Plerocercoids in Young Mice

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Introduction

It is known that some substances produced by parasites cause metabolic disturbances in their hosts, and growth factors secreted by parasites have recently attracted much attention. Fisher (1963), for example, reported that *Tribolium* larvae infected with *Nosema* sp. grew larger than uninfected controls, and Lincicome (1963) demonstrated that rats infected with *Trypanosoma lewisi* and mice infected with *T. musculi* gained weight more rapidly than controls. These observations suggest that the parasites stimulate the host to produce some hormone or some substance with hormone-like activity.

Mueller (1963) found that mice infected with *Spirometra mansonoides* plerocercoids showed increased body weight and skeletal growth, suggesting that *S. mansonoides* plerocercoids secreted a physiologically active substance. He reported (1965, 1968a, 1968b, 1974) that deer mice, hamsters, propylthiouracil-treated rats, thyroidectomized rats and hypophysectomized rats infected with the plerocercoids also showed increased body weight. Subsequently, the same effect of this parasite was demonstrated in other animals such as rats, castrate-rats, hypophysectomized castrate-rats, hypophysectomized mice (Steelman *et al.*, 1971; 1972), alloxan-diabetic rats (Ruegamer and Mueller, 1973), diabetic hypophysectomized rats (Phares *et al.*, 1978) and even lizards (Phares *et al.*, 1974).

Although several groups have studied the mechanism of the increase in body weight of rodents infected with *S. mansonoides* plerocercoids, it is not clear whether the increase is due to real growth or simple obesity. Meyer *et al.* (1965) reported that stimulation of lipogenesis was the primary cause of the increase, whereas Steelman *et al.* (1971) stressed that although experimental animals infected with the plerocercoids had an increased lipid content, the chief reason for increase in body weight was skeletal and muscular growth.

S. erinacei is the only species of the genus *Spirometra* found in Japan: *S. mansonoides* has not yet been recorded. Hirai *et al.* (1978) reported for the first time that *S. erinacei* plerocercoids enhanced weight-gain in mice, but the mechanism of this effect was unknown.

The present study was undertaken to investigate the mechanism of enhancement of weight-gain by *S. erinacei* plerocercoids in young male mice. First changes in weight of the main organs involved in the weight-gain were followed with time, and

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then the glycogen, triglyceride, protein and nucleic acid contents were measured for analysis at the cellular level. In addition, the head-body length and the width of the tibial cartilage were measured as indices of skeletal growth.

Materials and Methods

Plerocercoids of *S. erinacei* were collected from two species of snake, *Elaphe quadrivirgata* and *Rabdophis tigrinus,* captured in the south of Ehime Prefecture, Japan. Adult worms were obtained experimentally by feeding plerocercoids to cats, and these worms were identified as *S. erinacei* from their morphological characters.

Male ICR mice, approximately 20 g in weight at 4 weeks of age, were provided from Nihon Clea Co., Tokyo. Groups of 5 experimental and 5 control mice were used in each experiment. Mice in experimental groups were injected under the skin of the back with 10 scoleces in 0.4 ml of physiological saline solution containing 1000 u/ml penicillin-G and 0.5 mg/ml streptomycin. Animals in control groups received a similar injection of saline containing antibiotics without the worms. Mice were ear-coded, kept in cages lined with shavings and given pelleted mouse food (MF, Oriental Yeast Co.) and water *ad lib.*

Analytical grade commercial chemicals were used.

Differences between the two groups were assessed by unpaired Student's *t*-test.

Experiment I: Pairs of animals were killed by cervical dislocation on days 0, 5, 10, 15, 21, 28, 35 and 42 after the injection. The body weight and head-body length were recorded and then plerocercoids and organs were removed. The wet weights of the plerocercoids, bilateral anterior tibial muscles, liver, bilateral kidneys, spleen, bilateral epididymal fat pads and bilateral testes were recorded.

Experiment II: The amount of whole body triglyceride was measured on days 14, 28 and 42 after infection. The body and bilateral epididymal fat pads were weighed. Then the whole body including the epididymal fat pads was homogenized in a blender, and extracted with 20 volumes of n-hexane-ethyl ether-ethanol (5:5:2, v/v). Triglyceride was determined by the method of Fletcher (1968).

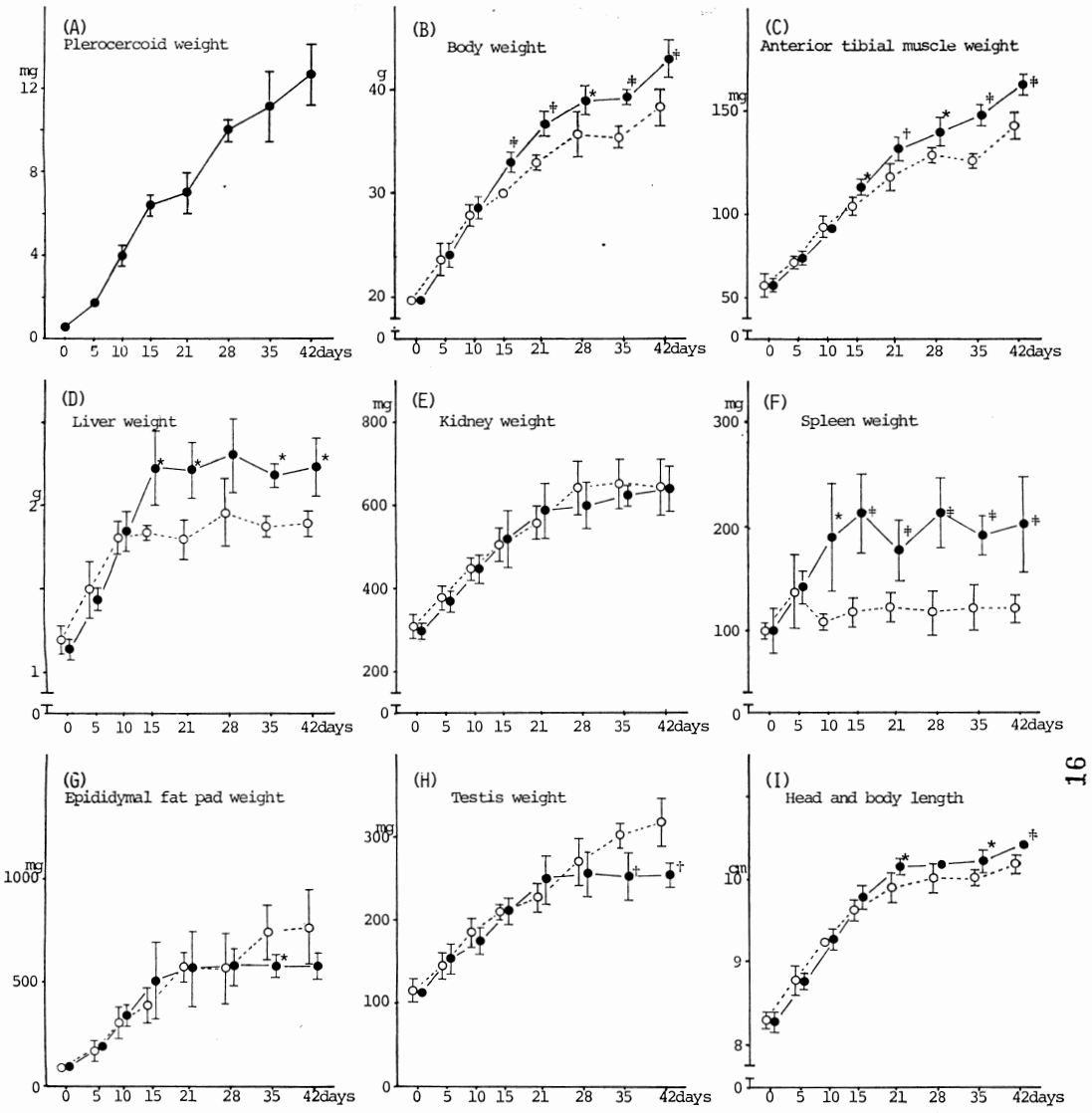
Experiment III: The triglyceride and glycogen contents of the liver and anterior tibial muscle were determined on days 14, 28 and 42 after infection. Triglyceride was measured by the method of Fletcher. Glycogen was measured by the method of Seifter *et al.* (1950); the samples were frozen in dry ice, weighed and glucose was used as a standard.

Experiment IV: The DNA, RNA and protein contents of the liver and anterior tibial muscle were determined on days 14, 28 and 42 after infection. The DNA, RNA and protein contents were extracted by a modification of the method of Schimidt-Thannhauser-Schneider by Mizuno (1969), and DNA and RNA were measured by the absorption at 260 nm. Protein was measured by the method of Lowry *et al.* (1951) with bovine serum-albumin as a standard.

Experiment V: The width of tibial caltilage was determined on days 14, 28 and 42 after infection. The tibia was fixed in 10% formalin, treated with 5% HNO_3 and then stood in 5% Na_2SO_4 for one day. Specimens were dehydrated in an ethanol series, embedded in paraffin, sectioned and stained with toluidine-blue. The width of tibial cartilage in sections of the proximal tibia was evaluated.

Results

Experiment I: The body weights, organ weights and head-body lengths of the ex-



16

Fig. 1 Effects of plerocercoids infection on organ weights in mice
○·····○ Control group ●——● Experimental group
Vertical lines represent standard deviations.
The significance of differences between means for the experimental and control groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

perimental and the control groups are summarized in Fig. 1 and Table 1. The weight of plerocercoids per mouse increased linearly with time to 127 mg on day 42 after infection, which was equivalent to 2.8% of the increased weight-gain over that of controls (Fig. 1A). The body weights of both controls and experimental animals increased rapidly until day 21, and then less rapidly. Until day 10 after in-

Table 1 Effects of plerocercoid infection on relative weights of organs

		0 days	5 days	10 days	15 days	21 days	28 days	35 days	42 days
Anterior tibial muscle	C.	3.36±0.33	3.24±0.18	3.38±0.23	3.48±0.17	3.56±0.17	3.62±0.22	3.55±0.05	3.75±0.21
	E.	3.35±0.20	3.28±0.15	3.29±0.15	3.44±0.23	3.62±0.13	3.60±0.13	3.76±0.16*	3.79±0.14
Liver	C.	60.6±4.9	63.0±4.5	64.9±2.2	61.8±1.0	54.1±3.1	54.6±2.5	52.6±1.4	49.2±1.8
	E.	57.4±1.8	59.5±2.9	65.0±2.2	67.3±6.6	60.4±4.6*	58.9±5.5	55.6±1.6*	51.8±3.4
Kidney	C.	15.8±1.3	16.1±0.9	16.1±0.5	16.9±1.3	16.8±1.1	18.1±2.2	18.3±1.4	16.7±1.3
	E.	15.2±1.0	15.4±0.6	15.9±0.9	15.7±1.9	16.1±2.2	15.5±1.7	15.9±0.9†	14.9±1.4
Spleen	C.	4.96±0.36	5.74±1.40	3.87±0.24	3.88±0.54	3.72±0.51	3.24±0.51	3.41±0.58	3.18±0.44
	E.	4.99±0.99	5.92±0.61	6.66±1.68†	6.47±1.15‡	4.84±0.93*	5.50±1.05‡	4.87±0.46†	4.71±1.11*
Epididymal fat pad	C.	4.6±1.1	6.8±2.2	10.9±2.9	12.9±3.1	17.4±2.5	15.9±5.5	20.7±3.6	19.7±4.8
	E.	4.5±0.6	8.0±1.2	11.6±1.9	15.2±6.4	15.4±5.1	14.7±2.2	14.5±1.4†	13.3±2.0*
Testis	C.	5.81±0.74	6.14±0.76	6.67±0.69	7.09±0.44	6.92±0.46	7.57±0.68	8.51±0.65	8.33±1.07
	E.	5.71±0.23	6.44±0.79	6.15±0.68	6.44±0.62	6.83±0.93	6.58±0.82	6.44±0.75‡	5.94±0.34‡

Relative weight is the ratio of organ weight to body weight (mg/g).
Values are means ± standard deviations.
The significance of differences between the means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

Table 2 Effects of plerocercoid infection on lipid metabolism in mice

		14 days		28 days		42 days	
		Absolute (mg)	Relative (mg/g Body wt.)	Absolute (mg)	Relative (mg/g Body wt.)	Absolute (mg)	Relative (mg/g Body wt.)
Epididymal fat pad	C.	504± 62	16.6±2.0	644±116	18.9±4.2	732±73	19.7±1.5
	E.	424± 48	13.5±2.2*	552± 55	14.8±2.2	690±95	15.5±1.8†
Whole body triglyceride	C.	2640±280	86.8±7.6	2950±530	86.6±16.7	3220± 290	89.3± 8.8
	E.	2370±190	75.1±7.8	2660±390	70.7± 8.2	3740±1100	84.1±22.6

Values are means ± standard deviations.
The significance of differences between means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$

Table 3 Effects of plerocercoid infection on triglyceride and glycogen contents of the liver and anterior tibial muscle of mice

		14 days		28 days		42 days	
		Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)
Liver triglyceride	C.	23.5±8.5	12.0±3.3	19.6±6.3	9.9±3.0	21.3±6.0	10.7±2.1
	E.	25.1±3.3	11.2±0.6	22.5±2.5	9.5±0.8	23.4±2.2	10.1±1.6
Liver glycogen	C.	90±22	46.3±10.3	93±24	47.2±12.7	99±18	50.6±9.7
	E.	106±24	47.2±9.9	109±9	46.3±3.9	87±9	37.2±3.3*
Anterior tibial muscle triglyceride	C.	0.476±0.138	4.17±1.30	0.481±0.059	3.96±0.52	0.500±0.068	3.66±0.47
	E.	0.475±0.112	4.12±1.18	0.570±0.061	4.05±0.09	0.625±0.029†	3.71±0.49
Anterior tibial muscle glycogen	C.	0.541±0.060	4.73±0.64	0.579±0.047	4.78±0.55	0.584±0.047	4.27±0.18
	E.	0.665±0.088	5.72±0.76	0.640±0.038	4.58±0.40	0.743±0.072†	4.56±0.27

Values are means ± standard deviations.
The significance of differences between means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

fection, the weight-gains of the two groups were similar, but from day 15 the experimental group began to gain weight more rapidly ($P<0.001$). The difference in body weights of the two groups increased with time, being as much as 4.5 g on day 42 after infection (Fig. 1B). As shown in Fig. 1C, the experimental mice showed a marked increase in weight of the anterior tibial muscle with age, its weight being much more than in controls from 15 days after infection ($P<0.05$). As there was no difference in the ratio of the anterior tibial muscle weight to the body weight, there was a close correlation between the increases in body and muscle weights (Table 1). The liver weight increased more rapidly in the experimental group than in the controls from day 15 ($P<0.05$), but as the difference in the liver weights in the two groups did not increase above 0.4 g after day 15, no correlation was observed between the increases in body and liver weights (Fig. 1D). No difference was found in the kidney weights of the experimental and control groups (Fig. 1E), and the ratio

Table 4 Effects of plerocercoid infection on DNA, RNA and protein contents of the anterior tibial muscle of mice

		14 days		28 days		42 days	
		Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)
DNA	C.	0.106±0.004	1.02±0.06	0.110±0.015	0.85± 0.07	0.137±0.011	1.02±0.07
	E.	0.112±0.008	1.01±0.07	0.133±0.013*	0.94± 0.08	0.170±0.009‡	1.03±0.07
RNA	C.	3.39±0.66	32.0±6.0	5.03±1.36	39.1±10.0	4.17±1.13	31.0±8.3
	E.	3.33±0.71	30.3±7.4	5.63±1.48	39.6±10.4	5.77±1.43	35.0±8.1
Protein	C.	10.7±0.5	101±2	13.9±1.2	108±3	14.3±1.0	106±5
	E.	11.4±0.9	103±5	15.4±1.2	108±5	17.9±1.5†	109±4
Protein/DNA (mg/mg)	C.	100±5		127±9		105± 8	
	E.	102±4		116±9		106±10	

Values are means ± standard deviations.
The significance of differences between means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

Table 5 Effects of plerocercoid infection on DNA, RNA and protein contents of liver of mice

		14 days		28 days		42 days	
		Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)	Absolute (mg)	Relative (mg/g Organ wt.)
DNA	C.	4.16±0.30	2.26±0.14	4.19±0.36	2.12±0.16	4.22±0.39	2.35±0.19
	E.	4.90±0.36†	2.26±0.13	4.97±0.47*	2.12±0.06	5.21±0.34†	2.37±0.16
RNA	C.	19.6±2.0	10.6±0.4	22.2±2.1	11.2±0.6	24.1±2.4	13.4±0.8
	E.	25.6±1.6‡	11.8±0.3‡	27.3±2.6†	11.7±0.4	28.3±1.7†	12.9±0.8
Protein	C.	237±21	129±2	254±19	129±6	256±20	142±11
	E.	277±12†	128±4	295±21*	126±5	310±14†	141± 8
Protein/DNA (mg/mg)	C.	57.2±3.4		60.9±4.5		60.6±2.3	
	E.	56.6±3.1		59.6±2.6		59.5±2.0	

Values are means ± standard deviations.
The significance of differences between means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

of the kidney weight to the body weight was less in the experimental group than in the control group (Table 1). The spleen weight increased more rapidly in the experimental group than in the controls at first ($P<0.05$), but no further increase was found after day 10 (Fig. 1F). The weight of the epididymal fat pad varied greatly in different individuals, but was less in the experimental group than in the control group on days 35 and 42 ($P<0.05$) (Fig. 1G). The relative weight of the epididymal fat pad was less in the experimental group than in the controls after day 21 ($P<0.05$) (Table 1). The weight of the testis was less in the experimental group than in the controls after day 35 (Fig. 1H). Head-body length increased

Table 6 Effects of plerocercoid infection on cartilage of mice

		14 days	28 days	42 days
Head and body length (cm)	C.	9.58±0.08	10.02±0.04	10.00±0.07
	E.	9.82±0.11†	10.26±0.05‡	10.24±0.05‡
Tibial cartilage width (μm)	C.	202± 8	159±13	128±9
	E.	227±14†	164± 8	135±8

Values are means ± standard deviations.
The significance of differences between means for experimental (E.) and control (C.) groups was tested by Student's *t*-test.
*: $P<0.05$ †: $P<0.01$ ‡: $P<0.001$

rapidly in both groups until day 21 (7 weeks of age) and then more slowly; the value for the experimental group was significantly more after day 21 ($P<0.05$), but subsequently the difference did not increase (Fig. 1I).

Experiment II: The triglyceride contents of the whole body in the two groups were similar (Table 2). A close correlation ($\gamma=0.752$) was observed between the triglyceride content of the whole body and the weight of the epididymal fat pad ($P<0.01$).

Experiment III: The triglyceride and glycogen contents of the liver and anterior tibial muscle are summarized in Table 3. The triglyceride contents of the liver and muscle were higher in the experimental group on days 28 and 42 than those in the control group ($P<0.05$). The glycogen content of the muscle was higher in the experimental group ($P<0.01$). The relative triglyceride and glycogen contents (mg/g organ weight) were similar in the two groups.

Experiment IV: The DNA, RNA and protein contents in the anterior tibial muscle are summarized in Table 4. The DNA content was higher in the experimental group than in the controls on days 28 and 42 ($P<0.05$). The protein content of the experimental group was higher on day 42 ($P<0.05$). The relative DNA and protein contents of the two groups were similar. Although the RNA content was slightly higher in the experimental group on days 28 and 42, this difference was not significant and was probably due to variation in individuals. Therefore, the DNA, RNA and protein contents increased in proportion to increase in muscle weight in the experimental group. As shown in Table 5, the DNA, RNA and protein contents of the liver in the experimental mice were higher than those of controls after 14 days, but the relative quantities of DNA, RNA and protein were not significantly different from those of the control groups and the ratio of protein to DNA was also not significantly different in the two groups.

Experiment V: As summarized in Table 6, the width of tibial cartilage was approximately 200 μm on day 14 when the head-body length was increasing rapidly, and later it decreased with age. Though the cartilage width decreased with age in the experimental group, it was significantly more than that of the control on day 14 after infection ($P<0.01$).

Discussion

Mueller (1963) first showed that the plerocercoid of *S. mansonoides* promoted weight-gain in rodents. Subsequently, he showed that the plerocercoid of other species of the genus *Spirometra* also stimulated weight-gain in some rodents. For instance he demonstrated that plerocer-

coids from Malay, Formosa and Australia stimulated increase in body weight of mice (Mueller, 1965; 1970). Although his morphological descriptions are not sufficient, these plerocercoids seem to be *S. erinacei* judging from the morphological character of the uteri of the adults.

Hirai *et al.* (1978) first reported that the plerocercoids of *S. erinacei* stimulated weight-gain of mice. According to their report, the increase in body weight of female ICR mice, about 11 g, began 3 weeks after infection. This report shows that the plerocercoids of *S. erinacei* enhance weight-gain of male ICR mice of 4 weeks old. Infected mice showed a marked increase in body weight from 15 to 42 days after infection, as reported by Hirai *et al.* (1978). Thus male as well as female ICR mice show increased weight-gain for at least 6 weeks after infection with plerocercoids of *S. erinacei.*

The reason for the increased weight-gain has been studied in hamsters, rats and hypophysectomized rats infected with *S. mansonoides* plerocercoids. Hamsters infected with the plerocercoids showed a marked increase in lipid content and in lipogenesis (Meyer *et al.,* 1965; Phares and and Carrol, 1977). Rats infected with the plerocercoids also showed an increase in the weight of the epididymal fat pad, but without increase in body weight (Glitzer and Steelman, 1971). However, it was not clear whether the increase in lipid content was the main reason for the increase in body weight, because the weights of the bone and muscle were not measured. The effects of infection on various organs of hypophysectomized rats, in which plerocercoids greatly enhanced increase in body weight, were also studied (Steelman *et al.,* 1970; 1971). Results showed that plerocercoids stimulated increases in weight of the muscle, bone, liver, kidney and epididymal fat pad. As the increases in weight of the bone and muscle were approximately proportional to that of body weight, they concluded that the increases in weight of these two organs were mainly responsible for the increase in body weight. However, their studies did not show whether the increase in body weight was caused by skeletal and muscular growth or by obesity.

There is only one report of studies on the mechanism of enhanced weight-gain in animals infected with plerocercoids from Malay and Formosa: Glitzer and Steelman (1970) reported on the organ weights of growing rats infected with plerocercoids from Malay and Formosa. They reported that the body weight and the weight of the epididymal fat pad increased but that the weights of the kidney and liver did not increase significantly. However, they did not examine the weights of the bone and muscle, which are related to increase in body weight, and did not study the changes in weight of organs with time after infection. Therefore, in this work we observed the changes in weight of various organs with time after infection. The weight of plerocercoids which constituted only 2.8% of the increase in weight, could not be the main reason for this increase in body weight. The head-body length and the weights of the muscle and liver began to increase from day 15 after infection, when significant increase in body weight began. The increase in weight of the muscle was especially closed proportional to the increase in body weight. As the spleen weight did not increase after day 10, the increase in the weight of the spleen also was not a main factor. Furthermore the weights of the kidney, testis and epididymal fat pad and the triglyceride content of the whole body did not increase, and so these organs and obesity were not related to the mechanism of accelerating increase in body weight. Results clearly showed that infection with *S. erinacei* plerocercoids stimulated increase in body weight by skeletal growth and increases in

muscle and liver weights.

Thus change in the chemical composition in the organs showing increasing weight-gain becomes of interest. Steelman *et al.* (1970, 1971) reported that *S. mansonoides* plerocercoids stimulated protein synthesis in muscle, ornithine decarboxylase activity in liver and cell division in tibial cartilage. From these results they concluded that *S. mansonoides* plerocercoids promotes the growth of the host. Since actual growth involves increase in cell number of the organ (Tsushima, 1980), their conclusion requires confirmation. Because, they did not show increase in cell number of the muscle.

Therefore, in this work detailed analyses were made of the muscle, liver and bone which were the main organs responsible for increase in body weight. Results showed that the DNA, RNA and protein contents of the muscle and liver, and the triglyceride and glycogen contents of the muscle of mice infected with *S. erinacei* plerocercoids increased. However, their contents relative to DNA did not increase. Since the DNA content of the diploid nucleus is constant in all cells and virtually all cellular DNA is chromosomal in origin, the ratio of protein to DNA is commonly used as an index of cell size. Thus the results indicate that the increase in the muscle and liver weights resulted from hyperplasia of these organs. As tibial cartilage width increased, increase in head-body length seemed to be due to enhanced cell division of the cartilage. Probably the low response of cartilage cells owing to physiological epiphysial-closure does not result in a significant increase in width in the period from 28 to 42 days after infection. The results show that *S. erinacei* plerocercoids stimulated cell division in the muscle, liver and epiphysial cartilage. Thus an important action of plerocercoids seems to be to increase the cell number.

Salmon and Daughaday (1957) suggested that the stimulation of growth by growth hormone was mediated by somatomedins, the substances that promote cell-growth in the cartilage and liver cells and myoblasts (Van Wyk *et al.*, 1971; Dulak and Temin, 1973; Ewton and Florini, 1980). Because plerocercoids enhance the mitotic activity of muscle, liver and cartilage cells, it seems probable that they stimulate secretion of growth factors.

S. erinacei and *S. mansonoides* plerocercoids are similar to each other in their effects on the bone, muscle and liver, but apparently differ in their effects on lipid metabolism. However, different hosts were used in previous studies on these two species and so this difference in effects requires comfirmation.

Summary

Intact male ICR mice of 4 weeks age were infected with *S. erinacei* plerocercoids collected in Ehime Prefecture, Japan, to investigate the mechanism of its effect in promoting weight-gain. The body weight, weights of the main organs and head-body length were measured with time after infection. On day 42 after infection the average weight-gain of infected mice was 4.5 g more than that of control mice. In the same period, the average weight of plerocercoids was 127 mg, or only 2.8% of the increased weight-gain of infected mice. The increase in body weight of infected mice was mainly due to increase in muscle and liver weights. The DNA, RNA and protein contents of their muscle and liver were increased, but their protein/DNA ratio was not increased. Head-body length and width of tibial cartilage were also increased, but there was no increase in lipid content, as observed in hosts infected with *S. mansonoides* plerocercoids. Thus *S. erinacei* plerocercoids stimulate cell division in the muscle, liver and epiphysial cartilage.

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マンソン裂頭条虫擬充尾虫の幼若マウスに対する成長促進作用について

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愛媛県において捕獲したシマヘビ，ヤマカガシより採取したマンソン裂頭条虫 *Spirometra erinacei* の擬充尾虫の10頭節を健康な4週令 ICR 系雄マウスの皮下に注入して，体重，頭胴長，擬充尾虫および諸臓器の湿重量を42日間経時的に測定した．実験群は対照群に比較して，感染後42日目で 4.5 g の体重増加が認められた．感染後42日目の擬充尾虫湿重量は 127 mg で，体重増加量の 2.8% にしか相当せず，擬充尾虫の重量は宿主の体重増加に余り貢献していなかった．感染群の頭胴長および前脛骨筋，肝臓と脾臓の重量は増加していたが，腎臓，副睾丸脂肪組織，睾丸の重量増加は認められなかった．そして，重量増加が顕著であった前脛骨筋と肝臓では，DNA，RNA と蛋白質量の増加が認められたが，細胞容量の指標である DNA 当りの蛋白質量には両群に有意な差は認められなかった．また，感染後14日目で実験群の脛骨近位端の軟骨幅が対照群よりも有意に増大していた．しかし，今回の実験では，*S. mansonoides* 擬充尾虫で観察されている脂質増加作用は認められなかった．以上のことから，マンソン裂頭条虫擬充尾虫は，主に幼若な雄マウスの筋肉，肝臓および骨端軟骨の細胞に細胞分裂を引きおこすことによって，宿主の成長を促進していることが明らかになった．