[Jpn. J. Parasitol., Vol. 38, No. 1, 46—53, February, 1989]

# Viable Egg Production of *Taenia crassiceps* Developed in the Intestine of Prednisolone-treated Golden Hamsters

HIROSHI SATO AND MASAO KAMIYA

(Accepted for publication; December 23, 1988)

## Abstract

Gravid *Taenia crassiceps* was recovered in the intestine of golden hamsters, *Mesocricetus auratus*, which were injected subcutaneously with 5 mg of prednisolone tertiary-butylacetate at 2 to 4 day intervals, from day 4 (Group I) or 0 (Group II) before administration of 30 cysticerci. Hamsters died or were sacrificed at 10 to 44 days PI, and mean recovery rate of sexually mature or gravid worms were 10.0% (Group I) or 17.3% (Group II) of administered cysticerci. Length and proglottisation of worms and proglottis release in feces were comparable to those of the worms grown in dogs. Egg counts showed that the number of eggs *per* proglottis obtained from hamsters ($18 \times 10^3$) were much more than that from dogs ($10—12 \times 10^3$). Its viability was demonstrated by artificial hatching test and experimental infection of eggs to red-backed voles, *Clethrionomys rufocanus bedfordiae,* and Mongolian gerbils. The fact that prednisolone-treated golden hamsters can harbor normal gravids of *T. crassiceps* with viable eggs assures a possibility that a complete life-cycle of carnivorous taeniid cestodes is reproduced in the laboratory using experimental small rodents alone.

**Key words:** *Taenia crassiceps,* golden hamster, *Mesocricetus auratus,* prednisolone, egg production, experimental model

## Introduction

Definitive hosts of *Taenia crassiceps* (Zeder, 1800) Rudolphi, 1810 in nature are various carnivores including the fox, wolf, dog and lynx, and many species of rodents play a role of the intermediate host (Abuladze, 1964; Freeman, 1962).

Kroeze and Freeman (1982) noted the unpublished observation of Chau and Freeman that, when cysticerci of *T. crassiceps* in an infected mouse were eaten by a second mouse, some of the metacestodes survived in the intestine of the latter for a week or more. And they conducted experimental oral administration of cysticerci of *T. crassiceps* to mice and showed that some of the worms survived in the small intestine up to 16 days and others penetrated through the intestinal wall and reached the peritoneal cavity within 24 hours.

This phenomenon was also observed in various rodents and the survival time in their intestines was as follows (Kitaoka, 1988; Saitoh, 1987); 11 days in Mongolian gerbils and red-backed voles, *Clethrionomys rufocanus bedfordiae,* 7 days in Chinese hamsters, 28 days or exceptionally 40 days in golden hamsters, *Mesocricetus auratus.* Proglottisation and development of genital primordia were observed in worms in the intestine, and also in the peritoneal cavity, although they did not become sexually mature (Kroeze and Freeman, 1983; Saitoh, 1987), except for golden hamsters in which the cestodes matured sexually (Kitaoka, 1988). Cortisone acetate-treated golden hamsters kept higher recovery rate of the worms in the intestine than non-treated ones, but eggs isolated

*Department of Parasitology, Faculty of Veterinary Medicine, Hokkaido University, Sapporo 060, Japan*

This work was supported by grants from the Ministry of Education, Science and Culture, Japan (Nos. 61880009 and 63480086) and Nissan Science Foundation.

佐藤　宏　神谷正男（北海道大学獣医学部家畜寄生虫病学講座）

from worms in golden hamsters were not viable (Kitaoka, 1988).

The production of viable eggs of taeniid cestodes in the unnatural host has not been confirmed in the previous reports (Gnezdilov, 1957; Kitaoka, 1988; Verster, 1971, 1974). In the present study, we conducted oral administration of cysticerci of *T. crassiceps* to prednisolone-treated golden hamsters and dogs to evaluate egg production and viability or infectivity of eggs.

## Materials and Methods

*Animal and treatment.* Ten conventional male golden hamsters, 5 weeks of age, purchased from a commercial breeder (Japan Laboratory Animals, Inc., Tokyo) and 2 mongrel dogs, 1 female and 1 male, about 1 month of age, were used. The former were divided into 2 groups with 5 animals each. Hamsters of Group I were injected subcutaneously in the dorsal region with 5 mg of prednisolone tertiary-butylacetate (PTBA) (Suspension of Codelcortone® -T.B.A., MERCK & CO., INC., Rahway, N. J., U.S.A.) at −4, −2, 0, 3, 6, 9, 12, 16, 20, 24, 28, 32 days PI. Those of Group II were treated similarly after the day of infection. The hamsters and dogs were kept in a conventional circumstance and given commercial pellets (CE-2 and CD-5, respectively, CLEA JAPAN, INC., Tokyo) and water *ad libitum*.

*Parasite and administration of cysticerci. T. crassiceps,* isolated from *Microtus montebelli* in Nagano Prefecture, Japan in 1985 (Miyaji, 1987), has been maintained by the serial peritoneal passage of metacestodes, cysticerci, using Mongolian gerbils in this laboratory, thereafter. The cysticerci used in the present study were removed from the peritoneal cavity of the animal infected 12 months before and placed into Petri dishes containing physiological saline for counting under a dissection microscope.

All hamsters of Groups I and II, lightly anesthetized with diethyl ether, were administered 30 fully developed cysticerci by Pasteur pipette with appropriate diameter for both passing of cysticerci and oral inoculation into posterior esophagus of hamsters. Dogs I and II were given 50 or 300 cysticerci using polyethylene tube, respectively.

*Recovery and examination of worms.* Two hamsters each of Groups I and II and 2 dogs were killed at 35 days PI. The others of Groups I and II were left until they died, and autopsied as soon as their death, as shown in Table 1. At autopsy, the intestine was removed from the carcass, and opened longitudinally. In hamsters, after the point of attachment was determined, the worm was placed and washed in numbered Petri dishes with physiological saline, while in dogs determination of attachment point and collection of worms were made every 10 cm of the small intestine opened longitudinally.

The length was measured in all worms collected from hamsters, and in 7 and 18 worms collected randomly from dogs I and II, respectively, after they were completely relaxed in distilled water for several hours (Roberts, 1961). After measurement, worms were fixed by hot 10% formalin and stained with acetocarmine. Morphological observations and proglottis counts of worms were made using whole-mounted specimens.

*Fecal examination.* In order to determine the initial day of proglottis shedding and their number, feces of animals were collected and examined every 12 hours from 24 days PI to the end of experiment.

*Egg counts.* Last 2 gravid proglottides were removed from the worms before formalin fixation at autopsy of 35 days PI and kept in antibiotic saline containing 1,000 I.U./ml penicillin, 10mg/ml streptomycin and 125 $\mu$g/ml fungizone for 2 weeks at 4°C. Certain numbers of gravid proglottides *i.e.*, 35 from hamsters of Group II, 40 and 50 from dog I, and 30 from dog II, were chopped with scissors in a Petri dish and filtered through 200-mesh using a pestle for crushing pieces of proglottides to free the eggs. The eggs were washed by centrifugation at 1,500 rpm for 10 minutes, and kept in the saline. The egg concentration in 40 ml egg suspension was measured by counting eggs in 20 $\mu$l 5 times, and the number of eggs *per* proglottis was calculated.

*Viability and Infectivity of eggs.* Viability of eggs, which had been stored in antibiotic saline for 2 weeks, was assessed using a artificial intestinal juice (Silverman, 1954). Infectivity of these eggs was determined by oral inoculation of 1,000 eggs, or subcutaneous and intraperitoneal injections of 1,000 oncospheres enclosed by the oncospheral membrane to female red-backed voles caught in the field and to female Mongolian gerbils, 5 weeks of age, bred in this laboratory. The animals infected with eggs obtained from hamsters were autopsied at 21 days PI and those infected with eggs from a dog were autopsied at 17—21 days PI.

## Result

*Worm recovery.* As shown in Table 1, various numbers of cysticerci developed into adult worms in the small intestine of both PTBA-treated hamsters and dogs. Mean recovery rate of sexually mature worms from hamsters of Groups I and II were 10.0% and 17.3%, respectively. Very rarely, normally segmented mature adults grown in the intestine were found in the peritoneal cavity. Mean attachment points of worms in the small intestine at 35 days PI were anterior 10% and 38% (range 10—57%) in 2 hamsters of Group I, 38% and 40% (0—68%) in 2 hamsters of Group II, 42% (24—56%) in dog I, and 65% (42—91%) in dog II.

*Maturation, worm length and proglottisation.* Worms from a hamster of Group I, which died at 10 days PI, were sexually mature and had fully developed testes, ovaries and vitellia. Worms at 22 days PI had markedly developed uterus containing immature eggs, and those at 25 days PI had several proglottides containing shelled eggs. At 35 days PI and thereafter most of worms from both hamsters and dogs possessed gravid proglottides (Fig. 1). As shown in Table 2, there was no abnormality of growth, maturation and proglottisation of worms grown in PTBA-treated golden hamsters in comparison with those of dogs. Difference observed among them might be explained by the "crowding effect" as discussed later.

*Proglottis release.* First proglottis appeared in feces at 25 day PI in hamsters of Group II and in dog II, and at 26 day PI in others. After 29 days PI, collected eggs were hatched *in vitro*. Total number of proglottides collected from feces

Table 1. Recovery of adult *T. crassiceps* from the intestine of golden hamsters and dogs

| Host and No. | | Number of cysticerci administered | Days of autopsy (PI) | Number of adult worms rocovered from intestine | Recovery rate (%) | Rate of mature worms included (%) |
|---|---|---|---|---|---|---|
| | 1 | 30 | 10D* | 6 | 20.0 | 83.3 |
| Golden | 2 | 30 | 25D | 4 | 13.3 | 100 |
| Hamster | 3 | 30 | 25D | 1 | 3.3 | 100 |
| Group I | 4 | 30 | 35K | 1 | 3.3 | 100 |
| | 5 | 30 | 35K | 4 | 13.3 | 100 |
| | 1 | 30 | 22D | 6 | 20.0 | 33.3 |
| Golden | 2 | 30 | 35K | 3 | 10.0 | 100 |
| Hamster | 3 | 30 | 35K | 17 | 56.6 | 76.5 |
| Group II | 4 | 30 | 40K | 2 | 6.7 | 100 |
| | 5 | 30 | 44K | 6 | 20.0 | 100 |
| Dog I | | 50 | 35K | 34 | 68.0 | 100 |
| Dog II | | 300 | 35K | 273 | 91.0 | 100 |

*D; dead, K; killed.

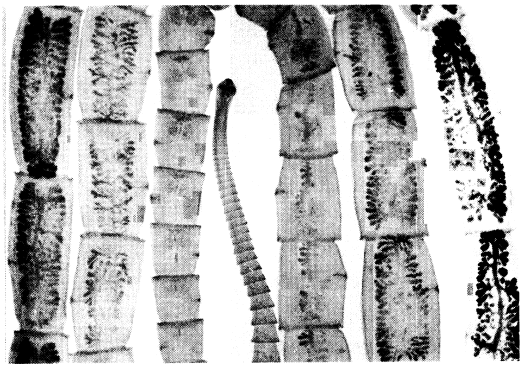

Fig. 1. Gravid *T. crassiceps* developed in the intestine of PTBA-treated golden hamster (No. 3) of Group II. × 3.21, stained with acetocarmine.

from the first day of shedding to 34 days PI and the number of proglottides collected from feces *per* worm *per* day from 29 to 34 days PI were as follows; 39 and 0.93 in hamsters of Group I (almost 2 animals), 136 and 0.73 in hamsters of Group II (4 animals), 113 and 0.37 in dog I, and 369 and 0.21 in dog II (Fig. 2). The rate of proglottis release in dog II, which was administered with 300 cysticerci, were apparently lower than others, and hamsters of both Groups I and II had a high rate of proglottis release.

*Egg count.* Mean number of eggs *per* proglottis isolated from hamsters of Group II, and dogs I and II at 35 days PI were $18 \times 10^3$, $10 \times 10^3$ and $12 \times 10^3$, respectively.

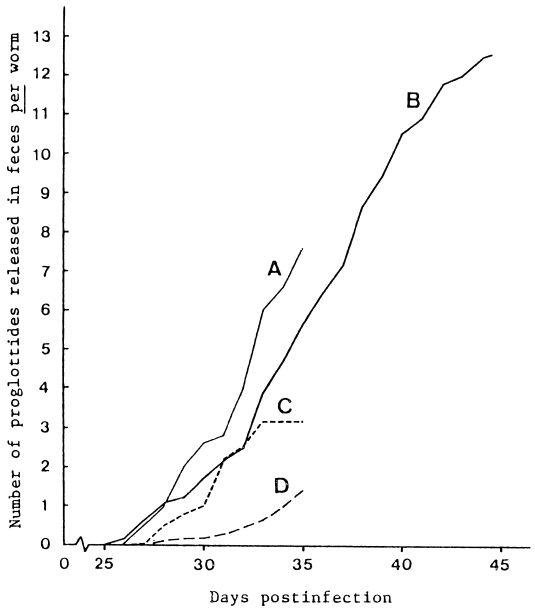


Fig. 2. Comparison of accumulated number of proglottides released *per* worm *per* day in feces of golden hamsters of Groups I (A) and II (B), and dogs I (C) and II (D).

*Viability of eggs.* Eggs collected from gravid worms in hamsters and dogs exhibited the characteristic dark brown coloring, and had a thick radially striated embryophores and well-developed hexacanth embryos (Fig. 3A). As stated by Freeman (1962), eggs of *T. crassiceps* did not need pretreatment with gastric juice, and

Table 2. Worm length and strobilar constitution of *T. crassiceps* grown in golden hamsters and dogs

| Host* | | Age of worms (days) | Number of worms examined* | Worm length (mm) | Number of Proglottides | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | | | Sexually immature | Sexually mature | Uterus dilated, but not gravid | Containing shelled eggs | Total |
| Hamster Group II | 2 | 35 | 3 | 286±50 | 43.1±2.1 | 22.0±3.0 | 29.5±2.1 | 16.5±0.7 | 108.0±7.5 |
| | 3 | 35 | 13 | 245±16 | 40.4±1.9 | 12.1±1.2 | 25.9±3.7 | 10.1±1.4 | 89.4±2.5 |
| | 5 | 44 | 6 | 246±38 | 43.5±2.7 | 22.0±1.4 | 29 & 30† | 3 & 1† | 91.8±3.4 |
| Dog I | | 35 | 7 | 262±40 | 40.3±1.7 | 20.6±3.3 | 22.1±1.6 | 7.3±6.3 | 88.9±3.3 |
| Dog II | | 35 | 18 | 215±42 | 39.1±2.1 | 14.9±1.5 | 17.4±3.1 | 7.6±2.4 | 79.0±4.7 |

* Host and total number of worms recovered were shown in Table 1. All examined worms were gravid except for those recovered from the golden hamster dead at 44 days PI (No. 5).

† Determined only from two worms.

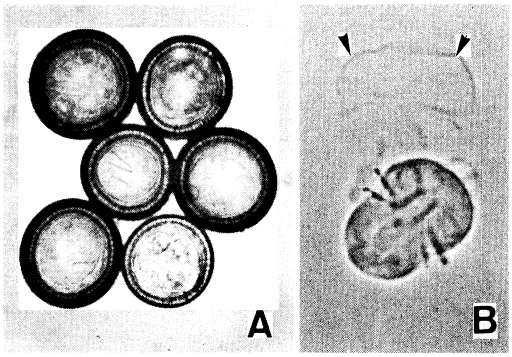


Fig. 3. Eggs and an oncosphere isolated from gravid proglottides of PTBA-treated golden hamsters of Group II. A. Normally developed eggs with thick radially-striated embryophores except for one egg. Hooklets were perceptible in some of them. ×420. B. A hatched and activated oncosphere. Arrow heads indicate its oncospheral membrane. ×650.

most of the eggs from 35-day-old gravid worms developed in PTBA-treated golden hamsters and dogs started to hatch soon after immersion in artificial intestinal juice. Most of these eggs hatched within 20 minutes, however, activation of oncospheres (Fig. 3B) was very rare in either sample even after a long time. Those oncospheres remained to be enclosed by the oncospheral membrane and exhibited a characteristic egg-shape appearance with normally arranged hooklets, indicating viability (Silverman, 1954).

The eggs of *T. crassiceps* obtained from hamsters of Group II and dog II at 35 days PI and kept at 4°C for 2 weeks were shown to be infective to voles and gerbils (Table 3). By oral inoculation of 1,000 eggs from hamsters, 3 of 5 gerbils were infected and 1—6 cysticerci were recovered from the pleural and peritoneal cavities and the subcutis in axillae, although none of 3 voles were infected. One to 15 cysticerci were recovered from 5 of 6 voles that were orally inoculated with 1,000 eggs from a dog. By subcutaneous and intraperitoneal injections of 1,000 oncospheres, 2—58 and 3—10 cysticerci, respectively, were recovered from all 3 voles, while 78 and 85 cysticerci in the subcutis and 5 and 12 cysticerci in the peritoneal cavity were found in 2 gerbils. Recovered cysticeci were morphologically normal.

## Discussion

Present study shows that one of the taeniid cestode, *T. crassiceps,* which normally grows in the intestine of carnivores, can also mature sexually in the intestine of prednisolone-treated golden hamsters and shed gravid proglottides containing viable eggs in its feces. This fact assures a possibility that the overall life-cycle of carnivorous taeniid cestode is reproduced in the laboratory merely using rodents.

Up to date, this kind of attempts to rear taeniid cestodes in the intestine of laboratory small rodents have been made by several authors with the purpose of producing eggs of human tapeworms, *T. solium* and *T. saginata* (Gnezdilov, 1957; Verster, 1971 and 1974) or making

Table 3. Experimental infection of eggs of *T. crassiceps* grown in a dog and golden hamsters for 35 days to red-backed voles and Mongolian gerbils

| Egg origin | Host infected | Infection of eggs | | |
|---|---|---|---|---|
| | | Oral inoculation | Subcutaneous injection | Intraperitoneal injection |
| Dog | Red-backed vole | 5/6* | NE† | NE |
| Dog | Mongolian gerbil | NE | NE | NE |
| Hamster | Red-backed vole | 0/3 | 3/3‡ | 3/3‡ |
| Hamster | Mongolian gerbil | 3/5 | 2/2‡ | 2/2‡ |

* Number of positive hosts/number examined.
† Not examined.
‡ Injection of oncospheres.

comparison of growth and development in different definitive hosts (Hutchison, 1959).

Verster (1971) was attracted by the result of Gnezdilov (1957) that *T. solium* might establish in the golden hamster, and he conducted experimental administration of cysticerci of *T. solium* and *T. saginata* to the prednisolone-treated animals. He showed appreciable increase in susceptibility to infection with adult *T. solium* and, in less degree, *T. saginata* in prednisolone-treated golden hamsters, although he failed to obtain gravid worms. In the latter, he compared the effect of various treatments, *viz.* antilymphocytic serum, whole body irradiation, or chemical immunosuppressants, to the susceptibility of golden hamsters to *T. solium* infection, and demonstrated all of those treatments greatly increased the susceptibility of those animals (Verster, 1974).

Hutchison (1959) found that *T. taeniaeformis* survived for 48 hours in the intestine of the albino rat in the search for more easily maintained laboratory animals to act as definitive hosts for this cestode instead of cats.

Administration of protoscoleces of *Echinococcus* species was also performed in a variety of small animals; ground squirrels, *Citellus citellus* (Žuković *et al.*, 1975), mice, cotton rats, rats, Dzungarian hamsters, golden hamsters (Kovalenko, 1976), and nude mice (Kamiya *et al.*, 1982). Kovalenko (1976) used some species of neonatal and juvenile laboratory rodents with or without cortisone treatment, and observed segmentation and formation of genital primordia of *E. multilocularis* reared in the intestine of cortisone-treated cotton rats for 17 days. He suggested the possibility of rearing *E. multilocularis* and *E. granulosus* in the intestine of laboratory small animals using antiphlogistics.

Apart from taeniid cestodes mentioned above, laboratory small rodents, especially golden hamsters, have been used commonly as a kind of taxonomic tool for morphological and/or developmental comparison of *Diphyllobothrium* species in the same definitive host (Andersen, 1972 and 1978; Bråton, 1966; Yamane *et al.*, 1988).

In the present study, the recovery rate of *T. crassiceps* in the intestine of hamsters of Groups I and II was 3.3—20.0% (mean 10.6%) and 6.7—56.6% (mean 22.7%), respectively (Table 1). Most of worms recovered were sexually mature and/or gravid. The recovery rate of adult worms was much higher in dogs (68.0% in dog I and 91.0% in dog II) than in hamsters. However, this high rate of worm recovery in dogs used in the present study might be attributed to their age, because they were weanling puppies. In a previous experiment using more old puppies, 10—37 worms (mean number 24.3) were recovered when 100 cysticerci were administered (Miyaji, 1987).

In addition to the loss of administered cysticerci by passing through the intestinal tract without attachment to its wall, another loss of worms in hamsters is attributed to the migration of some cysticerci to the peritoneal cavity. This unique phenomenon of *T. crassiceps* has been noted in a variety of rodents including golden hamsters (Kitaoka, 1988; Kroeze and Freeman, 1982 and 1983; Saitoh, 1987). Kroeze and Freeman (1982) reported that similar proportions of different doses of worms reached the peritoneal cavity within the first 24 hours after administration of cysticerci regardless of the size of the inoculum and sex or strain of mice used.

In the present study, some worms were also present in the peritoneal cavity. It is not shown whether the number of worms reaching the peritoneal cavity is affected or not by PTBA treatment. However, adverse effect of PTBA pretreatment to early worm establishment in the intestine was demonstrated in Mongolian gerbils, which were administered with *T. crassiceps* cysticerci similarly to the present study (Sato and Kamiya, unpublished). Gerbils treated with PTBA just after the administration of cysticerci had a higher recovery of adult worms in the intestine than those given PTBA pre-treatment.

Morphology, maturation, growth and proglottisation of adult *T. crassiceps* recovered from the intestine of PTBA-treated golden hamsters were normal and comparable to those from dogs (Fig. 1 and Table 2). As shown in Table 2, the

worms collected from dog II that harbored 273 adults are markedly smaller and have smaller number of proglottides than others. The number of immature proglottides is almost consistent in worms from each host, while appreciable difference exists in the number of mature and/or gravid proglottides. It is well known that growth in length and weight, and proglottisation of *Hymenolepis diminuta* in its definitive host reflect the effect of the population density within the host, commonly called the "crowding effect" (Roberts, 1961). This concept may be applicable to the present results.

Viable egg production and proglottis release in feces of taeniid cestodes reared in laboratory rodents have not been observed until the present study on *T. crassiceps* and on *E. multilocularis* (Kamiya and Sato, unpublished). Those of *Diphyllobothrium* spp. reared in golden hamsters have been investigated showing a very low rate of viable eggs (Andersen, 1972; Bråten, 1966; Meyer and Vik, 1963). Bråten (1966) described the viable eggs did not exceed 11% in *D. norvegicum* reared in the intestine of golden hamsters.

In the present study, gravid *T. crassiceps* reared in the intestine of golden hamsters had 18 $\times$ $10^3$ eggs *per* proglottis and released 0.73 or 0.93 proglottis *per* worm *per* day in feces. This egg number was apparently much more than those reared in the intestine of dog, because the egg number *per* proglottis in the latter was estimated to be 10 or 12 $\times$ $10^3$ in the present study and 13 $\times$ $10^3$ in a previous study (Miyaji, 1987).

The rate of establishment of the eggs obtained from gravid worms in hamsters is within the normal range (Bilqees, 1979; Miyaji, 1987). By subcutaneous and intraperitoneal injections of 1,000 oncospheres of *T. crassiceps* from dogs, mean recovery number (range) of cysticerci after 2 weeks PI was 29.5 (8—51) and 5.4 (up to 11) in female red-backed voles (Miyaji, 1987). Low rate of establishment of eggs seems to be one of characteristics of *T. crassiceps* itself (Bilquees, 1979; Miyaji, 1987).

Due to high rate of proglottis release and production of viable eggs with infectivity as well as high recovery rate of adult worms, and the ease of maintaining the animals, the use of golden hamsters as an experimental definitive host is valuable from the point of view of practical utility. And the use of adult-phased taeniid cestode/laboratory small rodent system will offer an indication to actual mechanism of host-parasite relationship, as already established *Hymenolepis*/laboratory small rodent systems (Ito and Smyth, 1987).

**References**


1) Abuladze, K. I. (1964): Taeniata of man and animals and the diseases caused by them. In Essentials of Cestodology, Vol. 4, ed. by K. I. Skrjabin, Akad. Nauk SSSR, Moscow (translated by Israel Program for Scientific Translation, Jersalem, 1970), 59—67.
2) Andersen, K. (1972): Studies of the helminth fauna of Norway. XXIV: The morphology of *Diphyllobothrium ditremum* (Creplin, 1825) from the golden hamster (*Mesocricetus auratus* [Waterhouse, 1839]) and a comparison with *D. dendriticum* (Nitzsch, 1824) and *D. latum* (L., 1758) from the same final host. Norw. J. Zool., 20, 255—264.
3) Andersen, K. (1978): The development of the tapeworm *Diphyllobothrium latum* (L. 1756) (Cestoda: Pseudophyllidea) in its definitive hosts, with special references to the growth patterns of *D. dendriticum* (Zitzsch, 1824) and *D. ditremum* (Creplin, 1827). Parasitology, 77, 111—120.
4) Bilqees, F. M. (1979): Effect of penicillin on the host tissue reaction in the development of *Taenia crassiceps* (Zeder, 1800). Acta Parasitol. Pol., 369—375.
5) Bråten, T. (1966): Studies on the helminth fauna of Norway. VII. Growth, fecundity, and fertility of *Diphyllobothrium norvegicum* Vik, (Cestoda) in the golden hamster. Nytt. Mag. Zool., 13, 39—51.
6) Freeman, R. S. (1962): Studies on the biology of *Taenia crassiceps* (Zeder, 1800) Rudolphi, 1810 (Cestoda). Can. J. Zool., 40, 969—990.
7) Gnezdilov, V. G. (1957): The golden hamster (*Mesocricetus auratus* Waterhouse) as a potential definitive host of the tapeworm *Taenia solium*. (In Russian). Zool. Zh., 36, 1770—1773.
8) Hutchison, W. K. (1959): Studies on *Hydatigera* (*Taenia*) *taeniaeformis*. II. Growth of the adult phase. Exp. Parasitol., 8, 557—567.
9) Ito, A. and Smyth, J. D. (1987): Adult cestodes; immunology of the lumen-dwelling cestode infections. In Immunology, Immunopathology and Immunoprophylaxis of Parasitic Infections, Vol. 2, ed. by E. J. L. Soulsby, CRC Press, Florida, 115—163.

10) Kamiya, M., Oku, Y., Kamiya, H. and Nomura, T. (1982): Characteristic responses of nude mice in Angiostrongyliasis and Echinococcosis. In Proceedings of the Third International Workshop on Nude Mice, Gustav Fischer, New York, 133—145.
11) Kitaoka, M. (1988): Golden hamsters as a definitive host of *Taenia crassiceps* (Cestoda). Master thesis, Hokkaido University. (Abstract was published in Jpn. J. Vet. Res., 36, 159.)
12) Kovalenko, F. P. (1976): Survival of *Echinococcus multilocularis* and *E. granulosus* protoscoleces in the intestine of laboratory rodents. (in Russian) Med. Parazitol. Parazit. Bolezn., 45, 350—352.
13) Kroeze, W. K. and Freeman, R. S. (1982): *Taenia crassiceps*: Fate of cysticerci following ingestion by the mouse. Exp. Parasitol., 54, 425—431.
14) Kroeze, W. K. and Freeman, R. S. (1983): Growth and development of *Taenia crassiceps* (Cestoda) in the small intestine and peritoneal cavity of mice following oral infection. Can. J. Zool., 61, 1598—1604.
15) Meyer, M. C. and Vik, R. (1963): The life-cycle of *Diphyllobothrium sebago* (Ward, 1910). J. Parasitol., 49, 962—968.
16) Miyaji, S. (1987): Development and proliferation of *Taenia crassiceps* in intermediate and definitive hosts. Master thesis, Hokkaido University. (Abstract was published in Jpn. J. Vet. Res., 35, 138.)
17) Roberts, L. S. (1961): The influence of population density on patterns and physiology of growth in *Hymenolepis diminuta* (Cestoda: Cyclophyllidea) in the definitive host. Exp. Parasitol., 11, 332—371.
18) Saitoh, R. (1987): Fate of cysticerci of *Taenia crassiceps* following oral infection in small mammals. Master thesis, Hokkaido University. (Abstract was published in Jpn. J. Vet. Res., 35, 145.)
19) Silverman, P. H. (1954): Studies on the biology of some tapeworms of the genus *Taenia*. I. Factors affecting hatching and activation of taeniid ova, and some criteria of their viability. Ann. Trop. Med. Parasitol., 48, 207—215.
20) Verster, A. (1971): Preliminary reports on the golden hamster as a definitive host of *Taenia solium* Linnaeus, 1758 and *Taenia saginata* Goeze, 1782. Onderstepoort J. Vet. Res., 38, 63—64.
21) Verster, A. (1974): The golden hamster as a definitive host of *Taenia solium* and *Taenia saginata*. Onderstepoort J. Vet. Res., 41, 23—28.
22) Yamane, Y., Bylund, G., Abe, K., Fukumoto, S. and Yazaki, S. (1988): Early development of four *Diphyllobothrium* species in the final host. Parasitol. Res., 74, 463—468.
23) Žuković, M., Wikerhauser, T., Džakula, N. and Yelewere, G. (1975): O razvoju trakavice *Echinococcus granulosus* u tekunici (*Citellus citellus*). Acta Parasitol. Iugosl., 6, 67—71.