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# The development of *Hymenolepis diminuta* tapeworms of inbred line WMS il1 in rats of the WAG alb. race in primary and secondary infections of varying intensity

M. STRADOWSKI

Department of General Biology and Parasitology, Medical Academy, Chalubinskiego 5, 02-004 Warsaw, Poland,
E-mail: *mstrad@poczta.onet.pl*

**Summary**

Following its termination by the antihelminthic Yomesan, a primary infection due to the administration of 50 *H. diminuta* cysticercoids has no significant influence on the abundance, length or dry mass of tapeworms in a secondary infection of 7, 13 and 42 days, or 8 months duration in WAG alb. rats. Crowded primary and secondary tapeworm infections experienced only slightly greater proportional losses than low-abundance populations. In no period of study was there any destrobilation of tapeworms.

Key words: *Hymenolepis diminuta*; primary and secondary infection; numbers of tapeworms; length and dry weight of tapeworms

## Introduction

Many studies point to the relative large loss of *H. diminuta* tapeworms from multiple infections. Thus, for example, the supply of 25 or 30 cysticercoids to rats was followed by a dramatic fall in numbers of worms shortly after infection (Harris and Turton, 1973) and (Chappell and Pike, 1976), respectively. In contrast, no losses occurred in 5-worm infections in these same experiments (Harris and Turton, 1973; Chappell and Pike, 1976). 1 to 3 months after dosing with between 40 to 100 cysticercoids, the intestines of rats have been found to contain around 10 or fever tapeworms (Hesselberg and Andreassen, 1975; Pike and Chappell, 1981; Featherston and Copeman, 1990; Andreassen, 1991; Featherston *et al*., 1992). In turn, in other studies, 20 days after 100 cysticercoids were given only half remained (Hindsbo *et al*., 1982), while 2 days after 300 larvae were supplied only a quarter of the number of tapeworms remained (Roberts, 1961). In connection with the decline in the number of tapeworms (Pike and Chappell, 1981; Featherston *et al*., 1992) and destrobilation (Featherston *et al*., 1992), the mass of a crowded population declines dramatically. The importance of the race of rats will be referred to in further discussion.

The number of tapeworms declines much more rapidly in a secondary infection than in primary one (Andreassen and Hopkins, 1980), while the dry mass of population falls more abruptly (Andreassen and Hopkins, 1980; Hopkins and Andreassen, 1991).

Some authors have noted the destrobilation of tapeworms in crowded primary or secondary populations or in both (Hindsbo *et al*., 1982; Featherston *et al*., 1992). Taylor and Hole (1997), presumed that the initial stage of destrobilation is revealed in the narrowing of the strobila beyond the neck. Crowded populations of other *Hymenolepis* species like *H. citelli* also show a rapid reduction in the number of tapeworms with declining biomass and destrobilation (Alghali, 1986).

Some authors consider the nature of the *H. diminuta* population in course of primary, secondary or supplementary infection to be a manifestation of the crowding effect (Chandler, 1939; Roberts and Mong, 1968), while others attempt to link the issue with reactions on the part of the host organism (ChoromaĔski, 1980; Machnicka and Choromanski, 1983; Featherston and Copeman, 1990; Featherston *et al*., 1992; Ishih, 1992, 1994; Taylor and Hoole, 1997; Dwinell *et al*., 1998). In effect the precise causes of the abrupt decline in the numbers and mass of tapeworms, as well of destrobilation, remains unknown.

Irrespective of these as yet not unambiguously explained parasite-host interactions, the complex and still poorly-known structure of tapeworm infrapopulations still requires recognition - including in the inbred line *H. diminuta* WMS il1.

The aim of the work described here was thus to study the abundance, lengths and masses of tapeworms in different

periods of primary and secondary infections, following the supply of rats of the WAG alb. race with defined numbers of cysticercoids of *H. diminuta* WMS il1. Attention was paid to the presence or absence of final proglottids, in which the excretory canals joined, which allow the rate of development of tapeworms to be assessed.

**Material and Methods**

*H. diminuta* cysticercoids deriving from generations 56 – 58 of the WMS il 1 inbred line were dissected from *Tribolium destructor* and transferred to an appropriately excavated depression in the cooked white of a hen's egg, and fed to 5 months old male rats of the WAG alb. strain. Further research was confined to those rats which ate (most often swallowed) the egg white with cysticercoids after less than 20 minutes of exposure to it following 24 – 36 hours without food. Research on the course of the secondary infection first involved the infection of 5-week-old rats with a dose of 50 cysticercoids, and then their deworming after 3 months by supplying 50 mg of crushed yomesan in 2 $cm^3$ of water twice a day for 12 successive days. To ensure uniform conditions in the experiment, this antihelminthic was also given to 4.5-month-old control rats which had not previously been infected.

The elimination of the primary infection was confirmed by both coproscopic studies of all rats and autopsy of 12 rats 7 – 8 days after the last dose of yomesan. Eight days after this last dose, the control near 5-month-old rats not infected hitherto were supplied with 6, 50 or 110 tapeworm cysticercoids each, as were the rats whose primary infections had been eliminated on between the $87^{th}$ and $92^{nd}$ days. Rats were fed murigran and autopsied after different intervals to allow for the study of tapeworms from the $7^{th}$, $13^{th}$ or $42^{nd}$ days of infection, or after 8 months. To determine the presence of very small tapeworms, the mucosa of the intestines of rats were detached with a scissors and scalpel and examined using a stereoscopic microscope. All the tapeworms obtained were transferred to physiological solution (0.9 % NaCl), counted, studied for signs of ongoing or completed destrobilation and maintained at 25 ± 1°C. The length of tapeworms was measured after individuals has ceased to move. They were in glass bowls of diameter 20 cm, covered to prevent evaporation of water and changes in NaCl concentration. For precise measurement, longer tapeworms were cut by scalpel and laid out along a ruler beneath the bowl. On account of the large number of tapeworms obtained following dosing with 110 cysticercoids, measurement was confined to the length of 60 – 70 randomly selected individuals from each population. Finally, all of the tapeworms were rinsed in distilled water, dried for 48 hours in aluminium foil at a temperature of 95 ± 1°C

Table 1. Intensity of infection with *Hymenolepis diminuta* tapeworm at different time after infection

| Dose of cystic. | Age of Worms | Primary (p) or secondary (s) infection | No. of tapeworms recovered | | | | | |
|---|---|---|---|---|---|---|---|---|
| | | | from | to | av. | total | ± SD | %* |
| 6 | 7 days | p | 5 | 6 | 5.8 | 35 | 0.4a | 96.7 |
| 6 | 7 days | s | 5 | 6 | 5.8 | 35 | 0.4a | 96.7 |
| 6 | 13 days | p | 5 | 6 | 5.7 | 34 | 0.5b | 95.0 |
| 6 | 13 days | s | 6 | 6 | 6.0 | 36 | 0.0b | 100.0 |
| 6 | 42 days | p | 4 | 6 | 5.3 | 32 | 0.8c | 88.3 |
| 6 | 42 days | s | 5 | 6 | 5.7 | 34 | 0.5d | 95.0 |
| 6 | 8 months | p | 4 | 6 | 5.2 | 31 | 0.8e | 86.7 |
| 6 | 8 months | s | 4 | 6 | 5.0 | 30 | 0.6e | 83.3 |
| 50 | 7 days | p | 41 | 50 | 47.0 | 282 | 3.3f | 94.0 |
| 50 | 7 days | s | 45 | 49 | 46.5 | 279 | 1.8f | 93.0 |
| 50 | 13 days | p | 43 | 48 | 45.7 | 274 | 2.0g | 91.0 |
| 50 | 13 days | s | 44 | 49 | 45.8 | 275 | 1.9g | 91.6 |
| 50 | 42 days | p | 42 | 48 | 44.7 | 268 | 2.7h | 89.4 |
| 50 | 42 days | s | 42 | 47 | 45.2 | 271 | 2.1h | 90.4 |
| 50 | 8 month | p | 37 | 44 | 40.8 | 245 | 2.4i | 81.6 |
| 50 | 8 months | s | 33 | 43 | 37.8 | 227 | 3.7i | 75.6 |
| 110 | 7 days | p | 87 | 105 | 93.8 | 563 | 7.0j | 85.3 |
| 110 | 7 days | s | 84 | 101 | 90.8 | 545 | 6.7k | 82.5 |
| 110 | 13 days | p | 87 | 97 | 93.5 | 561 | 3.8l | 85.0 |
| 110 | 13 days | s | 85 | 97 | 91.2 | 547 | 4.9l | 82.9 |
| 110 | 42 days | p | 85 | 95 | 89.7 | 538 | 3.3m | 81.5 |
| 110 | 42 days | s | 74 | 96 | 88.2 | 529 | 7.8m | 80.2 |
| 110 | 8 months | p | 65 | 80 | 73.0 | 365 | 6.4n | 66.4 |
| 110 | 8 months | s | 64 | 82 | 71.3 | 428 | 6.7n | 64.8 |

Number of rats per group = 6. Explanations: cystic. - cysticercoids; av. - average; * - related to no. of cysticercoids administered; figures with the same letters are not significantly different at the following levels: a, b, e, f, g, h, i, l, m, n: $P > 0.05$; (Figures c, d, j, k, are significantly different ($P < 0.1$)

and weighed to give the dry mass of the different populations. The dry mass of these populations calculated earlier – after 32 hours of desiccation – was identical, hence 48 hours desiccation was entirely sufficient. Statistical analysis of results was performed with the use of Student and Wilcoxon and Cochran-Cox tests.

**Results**

In general the numbers of tapeworms of the same age discover in the primary infection did not differ from those noted in the secondary infection (Tab. 1). The only exception concerned the number of 42-day old and 7-day old worms obtained following dosing with 6 and 110 cysticercoids (Tab.1). A decline in the number of tapeworms in relation to the number of cysticercoids supplied to rats is most visible over time in crowded populations. However, even here this is not an abrupt process (Tab. 1). All 7 – 13 day tapeworms had terminal proglottids in which the excretory ducts were joined - thereby exhibiting a feature characteristic of the early stage of development still prior to apolysis (Fig. 1). In contrast, after 8 months tapeworms lacked such proglottids, terminating instead in uterine ones. The 42-day-old populations obtained from rats initially dosed with 110 cysticercoids included 4 worms with a terminal proglottid of lingulate shape that arises first during ontogenesis and in which excretory ducts converge. There were 2 such tapeworms in the analogous secondary infections. All other 42-day-old tapeworms within both the crowded

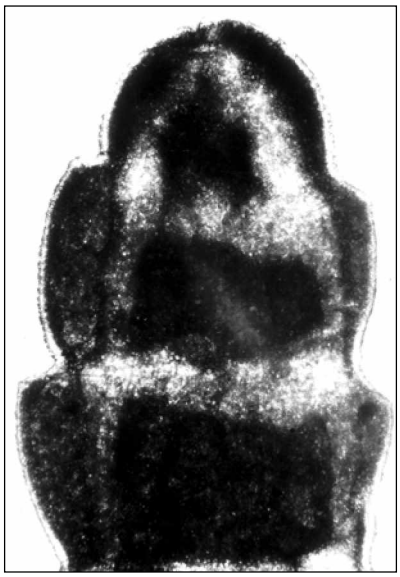

Fig. 1. End fragment with terminal, ligulate proglottid in a 13-day-old *Hymenolepis diminuta* tapeworm from a secondary infection induced by a dose of 50 cysticercoids

Tab. 2. Length of *Hymenolepis diminuta* in relation to the age of tapeworms, mean intensity and primary or secondary infection

| Age of worms | Primary (p) or secondary (s) infection | Mean intensity of infection | No. of investigated worms | Worm length in mm | | | |
|---|---|---|---|---|---|---|---|
| | | | | from | to | mean | ± SD |
| 7 days | p | 5.8 | 32 | 91 | 132 | 107.8 | 12.6a |
| 7 days | s | 5.8 | 32 | 84 | 128 | 104.6 | 13.5a |
| 13 days | p | 5.7 | 32 | 367 | 476 | 440.2 | 28.6b |
| 13 days | s | 6.0 | 36 | 376 | 481 | 438.2 | 25.8b |
| 42 days | p | 5.3 | 32 | 496 | 630 | 574.5 | 32.5c |
| 42 days | s | 5.7 | 32 | 509 | 638 | 572.6 | 30.0c |
| 8 mo. | p | 5.2 | 31 | 623 | 698 | 669.6 | 22.5d |
| 8 mo. | s | 5.0 | 28 | 637 | 705 | 671.5 | 21.2d |
| 7 days | p | 47.0 | 242 | 54 | 89 | 71.7 | 7.3e |
| 7 days | s | 46.5 | 247 | 56 | 86 | 74.0 | 6.7f |
| 13 days | p | 45.7 | 253 | 128 | 244 | 180.8 | 25.4g |
| 13 days | s | 45.8 | 244 | 125 | 253 | 185.5 | 27.1g |
| 42 days | p | 44.7 | 213 | 173 | 254 | 212.6 | 18.1h |
| 42 days | s | 45.2 | 181 | 163 | 256 | 203.7 | 20.0i |
| 8 mo. | p | 40.8 | 236 | 175 | 265 | 221.8 | 18.1j |
| 8 mo. | s | 37.8 | 226 | 132 | 292 | 222.9 | 18.6j |
| 7 days | p | 93.8 | 305 | 27 | 74 | 50.1 | 11.6l |
| 7 days | s | 90.8 | 307 | 29 | 77 | 47.8 | 11.6l |
| 13 days | p | 93.5 | 387 | 76 | 164 | 119.2 | 17.7m |
| 13 days | s | 91.2 | 37 | 84 | 153 | 116.9 | 15.0m |
| 42 days | p | 89.7 | 382 | 120 | 178 | 142.1 | 11.6n |
| 42 days | s | 88.2 | 361 | 123 | 184 | 147.0 | 12.3o |
| 8 mo. | p | 73.0 | 380 | 117 | 237 | 169.9 | 29.2p |
| 8 mo. | s | 71.3 | 349 | 116 | 247 | 173.3 | 29.6p |

Number of rats per group = 6. Explanations: figures with the same letters are not significantly different at the following levels: a, b, c, d, g, j, m, p: $P > 0.05$. Significantly different data are: e, f ($P = 0.014$; h, i ($P < 0.01$; k, l ($P < 0.03$); n, o, ($P < 0.01$). Other explanations as in Table 1.

and low-density populations had a terminal part of the strobila that lacked a terminal lingulate proglottid, while uterine proglottids were present. After none of the studied periods in the range 7 days to 8 months inclusive was there even a single recorded case of destrobilation. In spite of the detachment from the strobila of uterine proglottids 42-day old tapeworms were longer than 13-day-old ones, which had a complete strobila with the teminal proglottid arising first in the course of ontogenesis (Tab. 2). The length of tapeworms increased further after the 42nd day of infection, because the longest individuals were noted in the 8-month-old populations (Tab. 2).

In general the lengths of tapeworms of the same age discover in the primary infection did not differ from those recorded in the secondary infection. The only exception concerned the length of 7-day old and 42-day old worms obtained dosing with 50 cysticercoids, as well as the length of 7-day old and 42-day old worms obtained dosing with 110 cysticercoids (Tab. 2).

The dry mass of an population increases with the duration of infection, in spite of a decline in the number of tapeworms. This points to long-term growth in the mass of particular individuals (Tab. 3). The dry weight of populations of the same age and similar intensity of infection in the primary infection did not differ from those noted in these secondary infection (Tab. 3).

## Discussion

The work of many of the authors cited in the introduction has attested to an abrupt decline in the numbers of tapeworms in a crowded population of *H. diminuta*, as well as to a sudden decline in the dry mass of the population after an initial increase, and to quite frequent destrobilation of tapeworms. However none of these properties was characteristic of the population obtained from the *H. diminuta* WMS il1, be they primary or secondary infections, because the decline in the numbers of tapeworms was not so sudden as among worms studied by other authors, with the dry mass of the populations increasing up to the end of the study period in the 8th month of infection. Nor did any of the tapeworms show signs of destrobilation. Although the influence of the primary infection on the secondary infection diminishes with time studies by Andreassen and Hopkins (1980) seem to indicate that the interval in studies on *H. diminuta* WMS il1 referred to is short enough to reveal a possible influence of one infection on the other.

*H. diminuta* is not so closely associated with the circulatory system as many other helminths are. However, as Martin and Holland (1984) note, its contact with tissues of the host is distinct, and leads to atrophy of the cells of rat intestines. Dwinell *et al.* (1998) reported hypertrophy of the circular and longitudinal muscles of the intestines of

Tab. 3. Dry weight of *Hymenolepis diminuta* in relation to the age of tapeworms, mean intensity and primary or secondary infection

| Age of worms | Primary (p) or secondary (s) infection | Mean intensity of infection | No. of investigated populations | Populations dry weight in mg | | | |
|---|---|---|---|---|---|---|---|
| | | | | from | to | mean | ± SD |
| 7 days | p | 5.8 | 6 | 15 | 25 | 20.3 | 3.5a |
| 7 days | s | 5.8 | 6 | 20 | 27 | 23.2 | 2.6a |
| 13 days | p | 5.7 | 6 | 380 | 436 | 410.3 | 21.9b |
| 13 days | s | 6.0 | 6 | 394 | 446 | 418.8 | 19.0b |
| 42 days | p | 5.3 | 6 | 794 | 853 | 827.0 | 21.6c |
| 42 days | s | 5.7 | 6 | 787 | 847 | 818.2 | 22.8c |
| 8 mo. | p | 5.2 | 5 | 1231 | 1303 | 1252.8 | 30.8d |
| 8 mo. | s | 5.0 | 5 | 1197 | 1278 | 1238.4 | 36.1d |
| 7 days | p | 47.0 | 6 | 48 | 61 | 56.3 | 4.9e |
| 7 days | s | 46.5 | 6 | 46 | 58 | 50.7 | 4.8e |
| 13 days | p | 45.7 | 6 | 494 | 530 | 512.8 | 13.3f |
| 13 days | s | 45.8 | 6 | 479 | 523 | 505.8 | 19.7f |
| 42 days | p | 44.7 | 6 | 857 | 893 | 875.2 | 14.1g |
| 42 days | s | 45.2 | 6 | 858 | 918 | 888.2 | 23.9g |
| 8 mo. | p | 40.8 | 6 | 1278 | 1313 | 1299.8 | 13.6h |
| 8 mo. | s | 37.8 | 6 | 1287 | 1323 | 1306.5 | 14.6h |
| 7 days | p | 93.8 | 6 | 75 | 83 | 78.2 | 3.2i |
| 7 days | s | 90.8 | 6 | 71 | 81 | 76.5 | 4.1i |
| 13 days | p | 93.5 | 6 | 496 | 611 | 553.5 | 42.6j |
| 13 days | s | 91.2 | 6 | 514 | 614 | 571.5 | 38.3j |
| 42 days | p | 89.7 | 6 | 947 | 1076 | 1008.2 | 50.2k |
| 42 days | s | 88.2 | 6 | 962 | 1065 | 1020.7 | 38.8k |
| 8 mo. | p | 73.0 | 6 | 1217 | 1354 | 1294.7 | 46.5l |
| 8 mo. | s | 71.3 | 6 | 1193 | 1324 | 1263.2 | 49.9l |

Explanations: figures with the same letters are not significantly different at the $P > 0.05$ level. Other explanations as in Table 1.

rats infected by this tapeworm, though there was no unambiguous link with the structure of the population.

Particularly promising for futhure research is the discovery of a clear influence of the race of rat on the nature of the *H. diminuta* population. In turns out that the provision of just 10 cysticercoids leads to a rapid reduction in numbers of tapeworms in rats of the TM race (Ishih *et al.*, 1992) and the DA race (Ishih 1992, 1994) - the opposite effect to that noted in other races (Ishih 1992, 1994; Ishih *et al.*, 1992). In connection with this, it would in future be of value to make comparisons regarding the course of infection by *H. diminuta* in the races of rat studied by these authors. At present, all that may be done is to use the literature and the author's own results for comparisons on the basis of experimentation with a similar race of host and the related *H. diminuta* WMS il1 tapeworm. In the work done by Andreassen and Hopkins (1980), the authors used rats of the Wistar race from the WAG alb. race derives, as well as the RICE race of tapeworms which gave rise to the inbred line WMS il1. Such comparisons are in fact found to produce an unexpected result, since 7 days of primary and secondary infection in rats of the WAG alb. race led to the discovery of 94 % and 93 % of tapeworms respectively, as compared with the 50 cysticercoids supplied (Tab. 1); while the corresponding values found in the work of Andreassen and Hopkins (1980) were 58 % and 27 %. The dry masses of the respective populations in the primary and secondary infections (Tab. 3) are respectively 6 and 1914 times as great as in the work by Andreassen and Hopkins (1980). In addition, Andreassen and Hopkins (1980) described the presence of destrobilation, which was not recorded in *H. diminuta* WMS il1.

Analysis of this comparison brings no answer to the question as to whether the cause of such different results lies in differences between the Wistar and WAG alb. races of rat, in spite of the fact that the former derives from the latter, or may in fact be a reflection of the inbred nature of *H. diminuta* WMS il1,which was obtained in conditions whose principle is given by Stradowski, 1994.

On the basis of the results obtained by others and by the author, it is possible to distinguish different types of H. diminuta populations according to degrees of reduction in the numbers of tapeworms and degree of development of strobilae:

1) an abrupt decline in the numbers of worms in populations numbering at least 10, in rats of the races DA (Ishih, 1992, 1994) and TM (Ishih *et al.*, 1992) - as opposed to a completely different situation in other races compared (Ishih, 1992, 1994; Ishih *et al.*, 1992).

2) an abrupt decline in number and dry masses of RICE strain worms in Wistar rats in primary and secondary infection (Andreassen and Hopkins, 1980) which was discussed above. An abrupt decline in the number of worms in populations initially numbering 40 – 100 individuals - as a result of which the number declines to around 10 in the course of only 1 – 3 months (Hesselberg and Andreeassen, 1975; Pike and Chappell, 1981; Andreassen, 1991; Featherston *et al.*, 1992; Featherston and Copeman, 1990), with described destrobilation of tapeworms (Hindsbo *et al.*, 1982; Featherston *et al.*, 1992).

3) a slower decline in the number of tapeworms than in group 2 as presented in rats of the Holtzman race (Roberts and Mong, 1968).

4) a very slow decline in the number of tapeworms in populations rising after dosing with 6 – 50 cysticercoids, along with a greater, though still not abrupt, decline in the number of tapeworms in populations obtained from 110 cysticercoids (Tab. 1). In no case was any destrobilation noted, and the length and mass of tapeworms increased over a long period of time (Tabs. 2, 3). It also results from work on *H. diminuta* WMS il1 conducted in WAG alb. rats that the number of worms does not fall abruptly in either sparse or crowded populations studied up to 10$^{th}$ month of infection (Stradowski, 1998)

The presented classification is certainly lacking in much of the important information, because the authors do not always detail the race of the host and tapeworm, while the conditions in which the work was carried out were often not comparable. Nevertheless, the comparison may facilitate the process by which methodologies for studying the issues presented are devised. Indicated above all is the value of using rats of the WAG alb. and DA races in direct comparative research.

**References**


ALGHALI, S. T. O. (1986): *Hymenolepis citelli* and *H. diminuta*: Worm burdens in homologous and heterologous infections in rats. *Exp. Parasitol.*, 62: 14 – 23

ANDREASSEN, J. (1991): Immunity to adult cestodes: Basic knowledge and vaccination problems. A review. *Parassitologia*, 33: 45 – 53

ANDREASSEN, J., HOPKINS, C. A. (1980): Immunologically mediated rejection of *Hymenolepis diminuta* by its normal host, the rat. *J. Parasitol.*, 66: 898 – 903

CHAPPELL, L. H., PIKE, W. W. (1976): Loss of *Hymenolepis diminuta* from the rat. *Int. J. Parasitol.*, 6: 333 – 339

CHANDLER, A. C. (1939): The effects of number and age of worms on development of primary and secondary infections with *Hymenolepis diminuta* in rats, and an investigation into the true nature of "premunition" in tapeworm infections. *A. J. Hyg.*, 29: 105 – 114

CHOROMAĆSKI, L. (1980): Humoral and cellular responses in mice and rats infected with *Hymenolepis diminuta*. *Bull. Acad. Pol. Sci. Biol.*, 28: 707 – 713

DWINELL, M. B., WISE, R. M., BASS, P., OAKS, J. A. (1998): *Hymenolepis diminuta*: Mucosal mastocytosis and intestinal smooth muscle hypertrophy occur in tapeworm-infected rats. *Exp. Parasitol.*, 89: 92 – 102

FEATHERSTON, D. W., COPEMAN, C. N. (1990): Mucosal mast cells in Spraque-Dawley rats infected with *Hymenolepis diminuta* tapeworms. *Int. J. Parasitol.*, 20: 401 – 403

FEATHERSTON, D. W., WAKELIN, D., LAMMAS, D. A. (1992): Inflammatory responses in the intestine during tapeworm infections. Mucosal mast cells and mucosal mast cells proteases in Spraque-Dawley rats infected with *Hy-*

*menolepis diminuta. Int. J. Parasitol.,* 22: 961 – 966
HARRIS, W. G., TURTON, J. A. (1973): Antibody response to tapeworm *(Hymenolepis diminuta)* in the rat. *Nature,* 246: 521 – 522
HESSELBERG, C. A., ANDREASSEN, J. (1975): Some influences of population density on *Hymenolepis diminuta* in rats. *Parasitology,* 71: 517 – 523
HINDSBO, O., ANDREASSEN, J., RUTTENBERG, J. (1982): Immunological and histopathological reactions of the rat against the tapeworm *Hymenolepis diminuta* and the effects of antithymocyte serum. *Parasit. Immunol.,* 4: 59 – 76
HOPKINS, C. A., ANDREASSEN, J., (1991): Inhibition of growth of a tapeworm *Hymenolepis diminuta* in its normal host (rat). *Int. J. Parasitol.,* 21: 47 – 55
ISHIH, A. (1992): Mucosal mast cell response to *Hymenolepis diminuta* infection in different rat strains. *Int. J. Parasitol.,* 22: 1033 – 1035
ISHIH, A. (1994): Worm burden and mucosal mast cell response in DA and F344\N rat strains infected with *Hymenolepis diminuta*. *Int. J. Parasitol.,* 24: 295 – 298
ISHIH, A., NISHIMURA, M., SANO, M. (1992): Differential establishment and survival of *Hymenolepis diminuta* in syngeneic and outbred rat strains. *J. Helminthol.,* 66: 132 – 136
MACHNICKA, B., CHOROMANSKI, L. (1983): Immune response and immunodepression in *Hymenolepis diminuta* infection in rats. *Zts. Parasitenk.,* 69: 239 – 245
MARTIN, J., HOLLAND, C. (1984): Scanning electron microscope studies of the mucosa of rats infected with *Hymenolepis diminuta (Cestoda). J. Helminthol.,* 58: 93 – 99
PIKE, W. W., CHAPPELL, L. H. (1981): *Hymenolepis diminuta*: Worm loss and worm weight loss in long-term infections of the rat. *Exp. Parasitol.,* 51: 35 – 41
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
ROBERTS, L. S., MONG, F. N. (1968): Developmental physiology of cestodes . III. Development of *Hymenolepis diminuta* in superinfections. *J. Parasitol.,* 54: 55 – 62
STRADOWSKI, M. (1994): Effects of inbreeding in *Hymenolepis diminuta (Cestoda). Acta Parasitol.,* 39: 146 – 149
STRADOWSKI, M. (1998): Influence of crowding of *Hymenolepis diminuta* (WMS il1) on structure of strobila. *Acta Parasitol.,* 43: 30 – 35
TAYLOR, K., HOOLE, D. (1997): Interactions between rat C-reactive protein and adult *Hymenolepis diminuta. Parasitology*: 297 – 302

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