Some aspects on the impact of parasitic infections in animals used as laboratory animals and their inpact on the parasites

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All animals normally harbour parasites.
Parasites are not living in mutual symbiosis with the host animal, they are taking advantage of it, although the host often appears to be clinically healthy. In the post-parasite relationship both parts will have to respond to each other. In the host this will, among other things, have effects on the immune system (*Lloyd et al.* 1988). The host-parasite relationship is always on the cost of the host. These parasites have in many cases evolved together with its normal host. The effect of parasitism on social behaviour of the host and the evolutionary theory of parasitism has recently been reviewed by *Curio* (1988).
There are several comprehensive books and papers published on parasites of domestic and laboratory animals and on clinical symptoms treatment of parasitic infections as well as prophylactic regimes (*Flynn* 1973, *Baker et al.* 1980, *Friedhoff et al.* 1983, *Wiethe & Hasslinger* 1986, *Collins* 1988, *Kunstyr* 1989). The morphology, life cycle, host range, clinical and pathological pictures are described. One should, however, be aware of the fact that observations usually were made on conventionally bred animals, not taking into account that factors such as strain, age, concomitant infections, food composition etc. may have a decisive effect on the outcome of a parasite infection.
In the present review parasites discussed belong to the helminths, arthropods and protozoa. The purpose is to elucidate some factors of the host having influence on the parasites and some effects of the parasites on the host. Both being part of a host-parasite relationship. Parasites can have an effect on the host and thus influence experimental results. Factors of the host, such as age, sex, immunological responsiveness e.t.c., or external factors such as food compositions will have an effect on the parasite burden. If the experimental situation then selects for such factors, the final result may be influenced.

Resistance
Parasites are generally host specific in the sense that a parasite species develops to maturity within only a specific host species or a closely related species. The nature of this host specificity is not known. Natural resistance in strains of inbred mice, variations in resistance related to sex and age, and mechanisms of natural resistance have been considered by *Albright & Albright* (1984). Some organisms, such as invasive amoebas are poorly adapted to their hosts and will give rise to a rapidly progressive disease contrasting to well adopted organisms such as *Toxoplasma gondii* which cause a chronic, long lasting infection. Heritable resistance to the stomach worm of sheep, *Hæmonchus contortus* was described already in 1955 by *Whitlock* (1955) and the hereditability was recently calculated to be 0.29 (*Albers et al.* 1984).
For the same sheep parasite the ability to undergo selfcure of the worm burden of the gastrointestinal tract, "expelling their worms", is shown to be related to hemoglobulin type (*Altaif & Dargie* 1978a). It is possible that the greater resistance of the HbAA sheep compared with HbBB sheep may be related to the differential oxygen-carrying capacity of the Hb types but there also

seems to be immunological differences linked with the Hb type (*Mehlhorn* 1988).
Much of the differences of susceptibility between strains of the same animal species have been found to be associated with genetic differences between the immune responses (*Wakelin* 1984, *Kennedy et al.* 1991). The regulation of gastrointestinal nematodes in mice and rats have been studied with the parasites *Trichinella spiralis* and *Nippostrongylus brasiliensis,* respectively. Most information is obtained from studies with *T. spiralis.* Experiments with high and low responders of outbred mice have shown that the regulating immune system is T-cell dependent. T-cell dependent responses to trematodes, cestodes and nematodes have been explored in the nude mice (*Mitchell & Holmes* 1982).
That the antibody response may influence the outcome of any infection is well known. Even the kinetics of the antibody response are shown to be determined by differences in mouse strain. This has been experimentally demonstrated for the development of resistance to the metacestode of *Taenia tæniæformis,* which normally develops in the liver of mouse and rat. In mice, strains such as C3H are highly susceptible while C57Bl/C are almost resistant, as very few metacestodes grow to mature cysticerci in their livers. Protective immunity against the growing metacestode involves the activity of complement-fixing IgG antibodies, but as the metacestode grows, it acquires anticomplement properties which inhibit the effect of complement-fixing antibodies. Thus, a strain of mice which fails to produce protective antibodies rapidly enough to affect the larvae before they become insusceptible, will harbour lots of metacestodes, although the same mice later will have a high specific antibody titer. The difference between a rapid (and protective) antibody response of the young C57Bl/C mouse and a delayed, and thus non protective response of the young C3H mouse, is a matter of a few days (*Mitchell* 1982). This knowledge is now resulting in vaccines for zoonotic metacestodes in sheep (*Sciutto et al.* 1990).
Strain differences in immune response have been shown to explain susceptibility to *Giardia muris* in C3H/He mice which became chronically infected, while BALB/C mice rapidly resolved the infection (*Erlich et al.* 1983). In another experiment (*Garcia et al.* 1983) BALB/C were susceptible to *Schistosoma japonicum* infection, while 50 % of 129/J mice showed an innate resistance to the same parasite. There are, of cource, also strain differences reported between parasite strains of the same species e.g. *T. spiralis* infections in the Chinese hamster (*Takada & Tada* 1987) or in humans (*Wakelin & Blackwell* 1988).
Genetically determined differences in an animal species to one parasite species will, however, not be valid for all parasites. Guinea pigs raised for susceptibility or resistance to the common intestinal roundworm of sheep, *Trichostrongylus colubriformis,* were infected with the common guinea pig mite, *Trixacarus cavie.* Compared with the nematode-susceptible guinea pigs, nematod-resistent animals had larger populations of mites and developed a more severe dermatitis with greater mast cell hyperplasia and many more infiltrating eosinophils (*Rothwell et al.* 1989).

Sex and Hormones

Not only the immune responses may play a role in the host-parasite relationship, but also hormones should be taken into account. Sex hormones (testosterone and oestradiol) were shown to influence the population dynamics of *Leishmania donovani* parasites in hamsters (*Anuradha et al.* 1990) or of *Heligmosomoides polygyrus* in free living wood mice (*Gregory et al.* 1990).
The delicate balance between a parasite and its host may be altered by hormones. Hormones produced by the host, corticosteroids, sex hormones, thyroid hormones, and insulin can be advantageous or disadvantageous for parasites as summarized by *Spinder* (1988).

An increase in the number of trichostrongylid ova in faeces during the periparturient period has been observed mostly in ewes, sows, and goats and is a well known phenomenon. It appears to be the result of elevated serum prolactin levels followed by a decrease in parasite-specific immune responses (*Urquhart et al.* 1987). The migration routes of *Toxocara* larvae in its final host, cats or dogs, are, i.a. dependent of sex(hormones) growth-hormone and previous *Toxocara* infection (*Soulsby* 1982). Probably, the galactogenic transmission of the common roundworm of the cat, *Toxocara mystax* (*cati*), in its paratenic host, the mouse (*Schon & Stoye* 1986), is influenced by hormones, too.
The fur mite, *Myobia musculi,* was found more frequently on male rats, and *Myocoptes masculinus* was found more often on conventionally raised female rats. No explanation was given (*Kang et al.* 1988).

Age
It is generally observed that young animals are more susceptible to parasites and parasitic diseases than adults. However, the susceptibility is usually not a question of age but of acquired resistance. Development of resistance to *Ostertagia ostertagia* in cattle and to other gastro-intestinal helminths requires approximately six months of continuous infection. If infection is interrupted earlier protective resistance will not develop (*Urquhart et al.* 1987). The inverse age resistance of cattle to the blood parasite *Babesia divergens* has not been fully explained, but no similar condition is demonstrable for the related parasites *Babesia microti* or *B. rodhani.* It should be noted that conclusions from one host-parasite system may not be regarded as generally valid for another, however, closely related (*Kreier et al.* 1983).

Food
Food and food composition have a direct influence on parasites, which indirectly, influence the host. Experiments varying the relative amount of protein in food for rabbits infected with *Obeliscoides cuniculi* showed that the development of the nematod in its histotrophic phase and in its fecundity was impaired in association with low protein diet. In addition, mild anaemia caused by the worms and changes of the mucosal immune response to the infection were related to the level of dietary protein (*Sinski et al.* 1988). Comparable findings were demonstrated in mice infected with another nematode, *Heligmosomoides polygyrus* (*Slater* 1988).
The fibre content of the food has been shown to have a significant effect on the number of *Giardia lamblia* trophozoites established in the small intestine of gerbils. Fewer parasites were established in animals on a high (20 %) fibre diet compared to those on a low (5 %) diet; probably a result of an increased mucus secretion (*Leitch et al.* 1989).

Vitamins
Deficiency of vitamin B2 and B6 will affect the growth of the cestode *Hymenolepis diminuta,* and most tapeworms of the order *Pseudophyllidea* will accumulate vitamin B12 (*Barret* 1981). Rats, accidently infected with *H. diminuta* showed great variations in the uptake of these vitamins from different kinds of food (*Christensson,* unpubl.).

Season
Even the season of the year is reported to have an effect on the result of experiment with the tapeworm *Hymenolepis diminuta* (*Choromanski* 1981). Seasonal occurrence of gastro-intestinal parasites of ruminants is amply described and partially explained by environmental conditioning of the infective larvae (*Soulsby* 1982).

Effects of parasites on the host
A parasite infection will activate the cellular and humoral immune system. The host will react with all its normal responses to foreign proteins. All classes of humoral immunoglobulins (IgA, IgE, IgM etc.) will be produced

(*Tizard* 1982). Nematodes and cestodes in particular will cause an IgE response. Parasites of these classes in the gut will also give rise to a local immunity in the intestinal wall. In the skin, ectoparasites will induce an immunological response.

Helminths also activate eosinophils and mast cells. Much of the immunity to helminths is therefore assumed to be dependent on T-cells. The role of T-cell subsets and cytokines in parasitic infections has been reviewed by *Cox & Liew* (1992).

As mentioned previously, lots of clinical symptoms and pathological lesions have been reported in the literature and will not be repeated here. However, some of the more recent reports are of interest. The effects described may to some extent be genetically influenced (*Grencis* 1990).

Pinworms are in most animal species considered to be of low pathogens. Of young Lobund-Wistar germ free rats infected with *Syphacia obvelata* and noninfected controls, the latter grew faster, and the weights at 6 weeks post infection were in average 12 % greater than those of their infected counterparts. It was concluded that pinworm infected rats are not suitable for growth studies (*Wagner* 1988).

Infection with the tapeworm *Hymenolepis citelli* caused a 2 % drop in dry food digestibility in *Peromyscus leucopus* (white-footed mice) (*Munger & Karasov* 1989).

Gastro-intestinal worms of sheep and horses are regularly expelled, in sheep known as "selfcure" (*Soulsby* 1982, Urquhart et al. 1987). This is also demonstrated in rats infected with *Nippostrongylus brasiliensis.* The role of mast cells and goblet cells to bring about the physical expulsion of the worms was discussed by *Levy & Frondoza* (1983).

Parasite infection costs energy which may increase food consumption or cause loss of weight as demonstrated in rabbits infected with *Sarcoptes scabei* (*Arlian et al.* 1988).

Intestinal parasites may cause morphological changes of the gut wall. The intestinal wall of conventionally raised sheep moderately infected with *Nematodirus spp.* will be 2–3 times thicker than normal due to oedema. Gnotobiotic piglets raised and kept behind barrier were infected with 10,000 larvae of *Ascaris suum.* The thickness of the muscular layer of the small intestine in infected animals was twice that of non-infected controls (*Christensson,* unpubl.). In mice experimentally infected with the trematode *Ecinostoma trivolvis* there was a marked increase of collagen in the intestinal musculature (*Weinstein & Fried* 1991). Infection with the nematode *Trichostrongylus colubriformis* in rabbits was shown to cause shortened villi and dilated crypts of the small intestine (*Hoste et al.* 1988). Infection of mice with *Hymenolepis diminuta* affected the number of eosinophils and non-eosinophilic cells in the lamina propria with IgE and IgA on their surface (*Vorst et al.* 1988). Interactions between the gut flora, coccidia and other intestinal parasites has also been reported (*Yvoré* 1989).

Parasitism of the gut will affect the organ not only while the parasites are present, but also because of lesions, which will persist even after the parasites have left. These interactions between the parasites and the host and its gut flora are discussed with particular reference to laboratory animals and animal production methods by *Worms* (1983).

The effect of a primary infection with parasites may be transient, severe in the acute phase but subclinical after becoming chronic. Mice experimentally infected with 10^3 cysts of *Giardia muris* or *Spironucleus muris* revealed a general immunosuppression the days 10 and 21 post infection. This period corresponded with the time of maximum parasite trophozoite levels in the small intestine (*Brett* 1983).

The parasite fauna of an animal population changes with time, mostly because of developing immunity. The dynamics may, however, depend on other factors such as concurrent infection. Experiments with the two species of pinworms in mice showed that the

worm burden of *Aspiculuris tetraptera* was established at a higher level than that of *Syphacia obvelata* when the two were introduced simultaneously (*Scott & Gibbs* 1986).
Infection with ectoparasites will also cause an immune response. Dogs infected with sarcoptic mange will show a humoral antibody response within 2–4 weeks (*Bornstein & Zakrisson* 1990). In mice infected with lice, increased cellular aggregation was shown in the subcutaneous tissues beneath the area where the lice lived. A reduction of blood vessels was also observed, leading to reduction of the number of lice (*Mehlhorn* 1988).
Low grade infections may exist unnoticed for a long time in well reared conventionally kept colonies. The use of virus antibody-free and ectoparasite-free mice as sentinels kept on the dirty bedding of the animals in the tested colony revealed not only mouse hepatitis virus, but also *Myobia musculi* of which there had been no trace before (*Thigpen et al.* 1989). Mites may be brought into a breeding colony by phoresis, i.e. being transported to hanging onto the legs of flying *Diptera* (*Kuhlhorn* 1982).

Remarks and conclusions

Parasites affect their host and the host responses have effects on the parasite population. There will be great individual variations due to genetic factors, sex, age, levels of immune response, concurrent infections, food, and other influences.
Parasite infection in animals may result in changes of the host such as alterations of their immune responsiveness, thickening of the gut wall, reduction of the number of skin vessels.
Animals grown parasite-free, e.g. raised behind barriers, in most cases are as susceptible as young animals. If these animals are put into an infected environment they will easily contract any parasite infection (*Scott & Gibbs* 1986). The animals will then pass through the acute and chronic phases of contracted infections. The experimental use of the animals under this period of parasitic development will probably have a greater influence on the results than using chronically infected animals from the beginning.
If parasitized or previously parasitized animals are used, this should be reported as well as the animal strain used. In long-term experiments the fluctuations of the parasite burdens should be monitored.
Parasite free animals can only be maintained behind barriers. The status of freedom has to be examined with intervals because parasites can be introduced by uncontrolled entrance of free living rodents who may spread parasite eggs, by flying dipteras which are vectors of blood parasites or by phoresis which may bring other arthropods and nematode larvae over the barrier.
In the dust of a room there may also be parasite eggs which may be dispersed by air, e.g. eggs of pinworms (*Soulsby* 1982).
Health monitoring of barrier or non-barrier bred animals should always include parasitological examination. Sampling procedures should be made so that the groups most likely to contract parasites, e.g. young animals, are always represented in the investigation. Techniques for examination should be chosen so that the most likely parasite species will be found. The gastro-intestinal tract should be examined for protozoans and helminths and the skin and fur for ectoparasites. Blood smears may be examined for parasites and richettsia in some colonies. Serological tests may be applied for *Toxoplasma gondii* and *Encephalitozoon cuniculi.* Pooled sample technies can be used for demonstration of gastro-intestinal helminths, fæcal parasite ova or oocysts.
Sentinels of known susceptible breeds kept on the dirty bedding used by a colony at test seems to be a way to amplify, propagate and thereby faciliate revealing low grade infections.

References

Albers GAA, SE Burgess, DB Adams, JSF Barker, LF Le Jambre & LR Piper: Breeding *Haemonchus contortus* resistant sheep – problems and prospects. In: JK Dineen & PM Outteridge (eds.): Immunogenic approaches to the control of endoparasites. CSIRO Australia 1984, 41–52.

Albright JF & JW Albright: Natural resistance to animal parasites. Immunobiology of parasites and parasitic infections. In: JJ Marchalonis (ed.): Contemp Top Immunol. 1984, *12,* 1–52.

Altaif KI & JD Dargie: Genetic resistance to helminths: the influence of breed and haemoglobin type of the response of sheep to primary infections with *Hæmonchus contortus.* Parasitology 1978, *77,* 161–175.

Anuradha R Pal & JC Katiyar: Sex-influenced population kinetics of *Leishmania donovani* in hamsters. Indian J. Exp. Biol. 1990, *28,* (9) 876–879.

Arlian LG, M Ahmed, DL Vyszenski-Moher, SA Estes & S Achar: Energetic relationships of *Sarcotes scabei var. canis* (*Acari: Sarcoptidae*) with the laboratory rabbit. J. Med. Entomol. 1988, *25,* (1) 57–63.

Baker HJ, JR Lindsey & SH Weisbroth: The laboratory rat. Research applications. American College of Laboratory Animal Medicine Series. Academic Press, New York 1980, *II,* 276.

Barret J: Biochemistry of parasitic helminths. 1. Worms, intestinal and parasitic. 1981.

Bornstein S & G Zakrisson: Ny diagnostik av hundens rävskabb. Svensk Veterinärtidning 1990, *42,* (4) 180–181.

Brett SJ: Immunodepression in *Giardia muris* and *Spironucleus muris* infections in mice. Parasitology 1983, *87,* (3) 507–515.

Choromanski L: The influence of the season on experimental infection of mice and rats with *Hymenolepis diminuta.* Zwierzeta Lab. 1981, *18,* (2) 81–84.

Collins BR: Common diseases and medical management of rodents and lagomorphs. In: Exotic animals, New York; Churchil Livingstone Inc. (eds. ER Jacobson & GV Kollias, Jr.), 1988, 261–316.

Cox FEG & EY Liew: T-cell Subsets and Cytokines in Parasitic Infections. Parasitol. Tod. 1992, *8,* (11) 371–373.

Curio E: Behaviour and Parasitism. Parasitology in Focus, Ed. H. Mehlhorn. Berlin Springer Verlag 1988, 149–158.

Erlich JH, RF Anders, IC Roberts-Thomson, JW Schrader & GF Mitchell: An examination of differences in serum antibody specificities and hypersensitivity reactions as contributing factors to chronic infection with the intestinal protozoan parasite, *Giardia muris,* in mice. Aust. J. Exp. Biol. Med. Sci.1983, (5) 599–615.

Flynn RJ: Parasites of laboratory animals. Printed in Ames, Iowa 40010, USA, Iowa State University Press 1973, 884.

Friedhoff KT, KI, MJ & BK: Parasitological problems in colonies of small laboratory animals. In: K Bonath (ed): Schwerpunkte der Infektionsuberwachung. Berlin und Hamburg, Paul Parey 1983, 40–54.

Garcia EG, WU Tiu & GF Mitchell: Innate resistance to *Schistosoma jasponicum* in a proportion of 129/J mice. J. Parasitol. 1983, *69,* (3) 613–615.

Gregory RD, AE Keymer & JR Clarke: Genetics, sex and exposure: the ecology of *Heligmosomoides polygyrus* (Nematoda) in the wood mouse. J. Anim. Ecol. 1990, *59,* (1) 363–378.

Grencis RK: Genetically determined variation in host response and susceptibility to pathological damage. Parasites: immunity and pathology. The consequences of parasitic infection in mammals. In: JM Behnke (ed): Parasites: Immunity and pathology. The consequences of Parasitic in mammals, London, Taylor & Francis Ltd. 1990, 120–145.

Hoste H, D Kerboeuf & AL Parodi: Trichostrongylus colubriformis: effects on villi and crypts along the whole small intestine in infected rabbits. Exp. Parasitol. 1988, *67,* (1) 39–46.

Kang YB, BJ So & KS Park: Trial for evidence of parasites in germfree mice. Korean J. Vet. Public Health 1988, *12,* (2) 143–150.

Kennedy MW, DL Wassom, AE McIntosh & JC Thomas: H-2 (I-A) control of the antibody repertoire to secreted antigens of *Trichinella spiralis* in infection and its relevance to resistance and susceptibility. Immunol. 1991, *73,* (1) 36–43.

Kreier JP, HS Abdalla & MR Oliveira: An evaluation of various rodent-Babesia systems for study of immunization against bovine babesiosis. Rev. Cienc. Biomed. 1983, *4,* 19–28.

Kuhlhorn F: On the Diptera fauna in the hutches of experimental animals in consideration of the hygienic aspects. 2. Rabbits (*Oryctolagus cuniculus f. domesticus L.*). Anz. Schadlingskun. Pflanzenschutz Umweltschutz 1982, *55,* (7) 103–107.

Kunstyr I: Mikrobiologische Diagnostik bei Laboratoriumstieren. Society for Laboratory Animal Science/Working Committee for Hygiene 1989. *11.*

Leitch GJ, GS Visvesvara, SP Wahlquist & CT Harmon: Dietary fiber and giardiasis: dietary fiber reduces rate of intestinal infection by *Giardia lamblia* in the gerbil. A. J. Trop. Med. Hyg. 1989, *41,* (5) 512–520.

Levy DA & C Frondoza: Immunity to intestinal parasites: role of mast cells and goblet cells. Fed. Proc., Fed. Am. Soc. Exp. Biol. 1983, *42,* (6) 1750–1755.

Lloyd S & EJL Soulsby: Immunological Responses of the Host. Parasitology in Focus, Ed. H Mehlhorn. Springer Verlag, Berlin 1988, 619–647.

Mehlhorn H: Parasitology in Focus. Springer Verlag, Berlin 1988.

Mitchell GF: Genetic variation in resistance of mice to *Taenia taeniaformis:* analysis of host-protective immunity and immune evation. In: A Flisser, K Willms, JP Lachette, C Ridaura, F Beltoun & C Larralde C Ridacera & F Beltran (eds.): Cysticercosis: Present state of knowledge and perspectives. Academic Press Onc., New York 1982, 575–584.

Mitchell GF & MC Holmes: Nude mice in the study of susceptibility and responses to infection with metazoan and protozoan parasites. In: Proceedings of the 3rd International Workshop on nude mice, Montana State University, Bozeman, Montana, 6–9 September 1979. Vol. I, Invited lectures, infection, immunology, ND Reed (ed.), page 1–9, New York, Gustav Fischer. 1982, *1.*

Munger JC & WH Karasov: Sublethal parasites and host energy budgets: tapeworm infection in white-footed mice. Ecol. 1989, *70,* (4) 904–921.

Rothwell TLW, SE Pope & GH Collins: Trixacarus caviae infection of guinea pigs with genetically determined differences in susceptibility to *Trichostrongylus culibriformis* infection. Int. J. Parasitol. 1989, *19,* (3) 347–348.

Schon J & M Stoye: Primary and galactogenic infection with *Toxocara mystax* Zeder 1800 (*Anisakidae*) in the mouse. J. Vet. Med. 1986, *33,* (6) 397–412.

Sciutto E, G Fragoso, L Trueba, D Lemus, RM Montoya, ML Diaz, T Govezensky, C Lomeli, G Tapia & C Larralde: Cysticercosis vaccine: cross protecting immunity with *T. solium* antigens against experimental murine *T. crassiceps* cysticercosis. Parasite Immunol. 1990, *12,* (6) 687–696.

Scott, ME & HC Gibbs: Long-term population dynamics of pinworms (*Syphacia obvelata* and *Aspiculuris tetraptera*) in mice. J. Parasitol. 1986, *72,* (5) 652–662.

Sinski E, B Bezubik, H Wedrychowicz, J Szklarczyk & M Doligalska: Effect of host nutrition on immunity and local immune response of rabbits to *Obeliscoides cuniculi.* In: Nuclear techniques in the study and control of parasitic disease of livestock. Proceedings of the final research co-ordination meeting on the use of nuclear techniques in the study and control of parasitic diseases of farm animals, IAEA, Vienna. 1988, 107–121.

Slater AFG: The influence of dietary protein on the experimental epidemiology of *Heligmosomoides polygyrus* (*Nematoda*) in the laboratory mouse. Proceedings of the Royal Society of London, Biological Sciences. 1988, *234,* (1275) 239–254.

Soulsby EJL: Helminths, Arthropods and Protozoa of Domesticated Animals. Bailliere Tindal, London 1982.

Spindler KD: Parasites and Hormones. Parasitology in Focus, Ed. H Mehlhorn. Springer Verlag, Berlin 1988, 465– 473.

Takada N & T. Tada: Evaluation of the natural resistance of Chinese hamsters to trichina infection II. Intestinal distribution and fecundity of adult worms associated with the course of expulsion. Japanese J. Parasitol. 1987, *36,* (5) 322–327.

Thigpen JE, EH Lebetkin, ML Dawes, HL Amyx, GF Caviness, BA Sawyer & DE Blackmore: The use of dirty bedding for detection of murine pathogens in sentinel mice. Lab. Anim. Sci. 1989, *39,* (4) 324–327.

Tizard I: An Introduction to Veterinary Immunology, WB Saunders Company, Canada 1982, 226–241.

Urquhart GM, J Armour, JL Duncan, AM Dunn & FW Jennings: Vet. Parasitol. Longman Scientific & Technical, England 1987, 286.

Vorst, E v d, H Dhont, JY Cesbron, M Capron, JP Dessaint & A Capron: Influence of an *Hymenolepis diminuta* infection on IgE and IgA bound to mouse intestinal eosinophils. Int. Arch. Allergy Appl. Immunol. 1988, *87,* (3) 281–285.

Wagner M: The effect of infection with the pinworm (*Syphacia muris*) on rat growth. Lab. Anim. Sci. 1988, *38,* (4) 476–478.

Wakelin D: Evasion of the immune response: survival within low responder individuals of the host population. Parasitology 1984, *88,* (4) 637–657.

Wakelin DM & JM Blackwell: Genetics of resistance to bacterial and parasitic infection. Taylor & Francis, London 1988, 287.

Weinstein MS & B Fried: The expulsion of *Echinostoma trivolvis* and retention of *Echinostoma caproni* in the ICR mouse: pathological effects. Int. J. Parasitol. 1991, *21,* (2) 255–257.

Whitlock JH: A study of inheritance of resistance to trichostrongyloidosis in sheep. Cornell Vet. 1955, *45,* 422–439.

Wiethe T & MA Hasslinger: On the infestation of laboratory mice by ectoparasites and possible treatment methods. Prakt. Tierarzt. 1986, *67,* (12) 1084–1086.

Worms MJ: The needs of the parasitologist. In: Microbiological standardisation of laboratory animals. Ellis Horwood Ltd, UK 1983, 57–65.

Yvore P: Interactions between coccidia, gut flora and intestinal parasites in mammals. Coccidia and intestinal coccidiomorphs. Proceedings of the Vth International Coccidiosis Conference. Tours (France) 17–20 October 1989, 183–192.