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REVIEW ARTICLE

Effects of macroparasites on the energy allocation of reproducing small mammals

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Abstract Reproduction, including lactation, is the most costly activity in terms of energy expenditure in female mammals. Consequently, the energy requirements of the reproducing female may not be met at this time, especially if other energy demanding activities are occurring concomitantly. Such activities could be the activation and maintenance of an immune system in response to parasitic infestation. These protective processes are energetically demanding and require trade-off decisions among competing energy demands. In the case of a reproducing mammal, the trade-offs occur mainly between defence against parasites and reproductive costs of the host. In this paper, I discuss the effects of macroparasites on the energy allocation of reproducing small mammals.

Keywords reproduction, small mammals, macroparasites, ectoparasites and endoparasites, immune response, trade-off decisions, energy allocation

1 Introduction

More than 50% of the species on earth and more than 50% of all individual organisms are parasites or pathogens and, consequently, their biological impact is substantial (Marcogliese, 2004). They play a major role in the life history of their hosts as they can affect such variables as fecundity, survival rate, mate selection, population dynamics, and growth of the host (Tompkins and Begon, 1999; Forchhammer and Asferg, 2000; Lochmiller and Deerenberg, 2000; Zuk and Stoehr, 2002).

In general, parasites cause harm to their hosts, but not immediate death. This implies a reduction of host fitness while parasitized (Arnold and Lichtenstein, 1993; Clayton and Moore, 1997), including an increase in host mortality and/or morbidity as well as a decline in host fecundity (Tompkins and Begon, 1999; Newey and Thirgood, 2004). Parasites can affect hosts in two ways: (1) directly, such as using the energy and nutrients of hosts (Khokhlova et al., 2002); and (2) indirectly, such as increasing the activity of the immune system (Wedekind, 1992; Lochmiller and Deerenberg, 2000), modifying behaviour (Barnard et al., 1998; Kavaliers et al., 1998; Poulin, 2000; Hofstede and Fenton, 2005) and decreasing the food intake of hosts (Tripet and Richner, 1997; Kyriazakis et al., 1998; Simon et al., 2004).

Although many studies have demonstrated the detrimental effects of parasites (Munger and Karasov, 1989; Alves, 1997), some have failed to do so (Munger and Slichter, 1995; Pacejka et al., 1998; Bouslama et al., 2001; Kristan, 2004). Most studies on the physiological and ecological impacts of parasites on hosts examined endoparasites, especially helminths (Munger and Karasov, 1991; Meagher, 1998; Behnke et al., 2001; Kristan, 2002a; 2002b; Meagher and Dudek, 2002; Kristan and Hammond, 2004). Studies on ectoparasites have been done mainly on avian hosts (Arendt, 1985; Brown and Brown, 1986; Fauth et al., 1991; Moller, 1990; de Lope et al., 1993; Richner et al., 1993; Richner and Tripet, 1999), with a few on mammals (Dzieciolowski and Clarke, 1990; Butler and Roper, 1996; Chekchak et al., 2000; Khokhlova et al., 2002; 2004a; 2004b; Krasnov et al., 2004; 2005).

In this review, I will discuss the effects of macroparasites on the energy allocation of reproducing small mammals and start by defining briefly the terms macroparasites and energy budget of the host.

1.1 Macroparasites

Parasites are traditionally divided into two main groups: microparasites and macroparasites. Macroparasites, larger than microparasites, are multicellular and include flatworms (Platyhelminthes – tapeworms and flukes) and roundworms (Nematoda) as well as ticks, fleas, mites

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and fungi. Some macroparasites live in intercellular spaces or in the body cavities of their hosts. Others do not enter their hosts but live part of the time on their hosts from where they receive their nutrients. They can grow and undergo sexual reproduction in or on their hosts but multiply by releasing juvenile stages outside of their hosts. Furthermore, macroparasites often undergo complex life cycles, requiring one or more intermediate hosts for development or growth (Marcogliese, 2004). Besides the division of micro- and macroparasites, parasites can also be divided into endoparasites, those that enter the body of the host (for example, flatworms and roundworms), and ectoparasites, those that remain outside the body of the host (for example, fleas and ticks).

1.2 Energy budget of the host

Food, which is digested in the gastrointestinal tract and absorbed via the blood sysytem, provides energy to the animal. Dry matter that the animal extracts from its diet is the apparent dry matter digestibilty (ADMD, proportion) and is calculated from the dry matter intake (DMI, g) and dry matter fecal output (DMFO, g) as:

$$\mathrm{ADMD} = (\mathrm{DMI} - \mathrm{DMFO})/\mathrm{DMI}$$

The apparent digestible energy (ADE; kJ) the animal obtains from its diet is the gross energy (GE; kJ) of the diet minus the energy lost in feces (FE; kJ):

$$\mathrm{ADE} = \mathrm{GE} - \mathrm{FE}:$$

and is usually expressed as a proportion or percentage of GE. The apparent metabolizable energy (AME; kJ) is the digestible energy minus the energy in urine (UE; kJ) plus energy in combustible gases (ECG).

$$\mathrm{AME} = [\mathrm{ADE} - (\mathrm{UE} + \mathrm{ECG})]$$

and is also usually expressed as a proportion or percentage of GE (Kam and Degen 1997).

The metabolizable energy intake (MEI) is the energy available to an animal for maintenance and production (growth, milk production, etc). If the MEI of an animal: (1) equals maintenance energy requirements, then there is no change in energy content and all the chemical energy intake is dissipated as heat; (2) is below maintenance requirements, then the animal is forced to catabolize tissue from its body energy to compensate for this lack of energy; and (3) is above maintenance requirements, the animal can then add to its body reserves (Degen, 1997). These relationships may be somewhat more complicated when, for example, the intake of a parasitized animal is below the energy requirements for both maintenance and immunity costs, nonetheless, the immunity system is activated by the mobilization of body energy. The relationship between metabolizable energy intake (MEI, kJ/d), heat production (HP, kJ/d) and body energy retention (ER, kJ/d) can be presented as:

$$\mathrm{MEI} = \mathrm{HP} + \mathrm{ER}$$

where ER can be either positive or negative.

Basal metabolic rate (BMR) represents a baseline of minimal energy expenditure for the body functions of an animal while awake, and when, basically, zero energy is expended in movement, thermoregulation, combating diseases and food absorption. It is a very widespread measure of energy expenditure in animals and is usually determined by their rate of oxygen (O_2) uptake. Resting metabolic rate, fasting metabolic rate and standard metabolic rate are all similar to BMR and these terms are often used interchangebly. Average daily metabolic rate (ADMR) is the metabolizable energy intake required by a caged, laboratory animal to maintain a constant body energy content. It includes BMR, the heat increment of feeding for maintenance, some minimal locomotory costs and possibly some thermoregulatory costs (Degen et al., 1998). The field metabolic rate of an animal is its energy expenditure under free-living conditions, which includes ADMR, locomotor costs and thermoregulatory costs (Degen, 1997).

1.3 Trade-offs between immune costs and other energetic costs

Parasites can affect their hosts through the diversion of resources (Candolin and Voigt, 2001; Zuk and Stoehr, 2002). This occurs when parasitized hosts are confronted with trade-off decisions between energy costs of the immune defence system and other energy demanding processes such as maintenance, reproduction, growth and thermoregulation.

Immunity can be either innate, that is, present in a host at all times or can be acquired, that is, activated in response to a challenge. Both require energy from the host during parasite infection. In general, the metabolic costs of: 1) mounting an immune response; and 2) maintaining a competent immune system are extremely difficult to measure and, consequently, have been assessed by measuring and observing physiological changes in the host. As stated by Lochmiller and Deerenberg (2000), "These two physiological traits are not easily addressed quantitatively, particularly with respect to the former, because of the integrated and organizational characteristics of the immune system with other physiological sytems". In addition, "severity, type, and duration of infection, ambient temperature, and gender, age, and nutritional status of the host all influence the cost of mounting an immune response."

It is generally accepted that activation of an immune response and even maintenance of a competent immune

system can be very energetically demanding processes (Sheldon and Verhulst, 1996; Lochmiller and Deernberg, 2000; Zuk and Stoehr, 2002). Indirectly, this has been supported by studies demonstrating that reproductive effort and energy restriction of a host can lead to a suppression of the immune system and an increase in the risk of infection, in particular in opportunistic pathogens (Norris et al., 1994; Oppliger et al., 1996; Hudson and Dobson, 1997; Christe et al., 1998; Murray et al., 1998; Lochmiller and Dabbert, 1993). Immune challenges often suppress food intake of a host (sepsis-induced anorexia) at a time when additional energy is vitally needed, as immune cells require high levels of glucose and glutamine, in particular. The body mobilizes protein and energy reserves to support the initial acute-phase immune response with an accelerarted lipolysis, proteolysis and glycolysis and, to do so, increases metabolic rate. This can often lead to a negative nitrogen balance and loss in body mass of the host (Hasselgren and Fischer 1998).

Derting and Compton (2003) examined the costs of mounting and maintaining immune responses in wild white-footed mice (*Peromyscus leucopus*). Mice were injected with sheep red blood cells and phytohemagglutinin (in foot pads), so that their humoral and cell-mediated immune responses would be stimulated, respectively. There was no difference in the dry matter intake, the apparent dry matter digestibility, the resting metabolic rate and the average daily metabolic rate between immunochallenged and control mice. Also, white blood cells concentration did not differ between immunochallenged and control mice but feet of mice injected with phytohemagglutinin were 57% heavier than in controls, showing a significant cell-mediated immune response. The wet and dry masses of the small intestine and testes and wet mass of lungs were significantly greater in the control than the immunochallenged mice. Consequently, it was concluded that ''mounting an immune response to a mild immunochallenge was associated with change in patterns of energy allocation .. specifically, energy allocation to the small intestine and testes were reduced. White-footed mice appeared to accommodate the cost of mounting an immune response through trade-offs in energy allocation to other physiological systems rather than through increased ingestion of food energy.'' To assess the energetic costs of maintaining an immune response, immune responses were suppressed by injection of cyclophosphamide, a drug that suppresses humoral immunocompetence but not macrophages or cell-mediated immunity (Allison 2000). The same measurements as above were made on the mice. There was an immunosuppressive effect as white blood cells in the control mice were 225% higher than in injected mice. However, there was no difference between the immunosuppressed and the control mice in any of the other variables measured. It was concluded that ''the cost of maintaining a normally functioning immune system was minimal in wild adult-male white-footed mice. Significant suppression of the immune system was not associated with a significant change in RMR (resting metabolic rate) or in DMR (daily metabolic rate) or any measurable change in energy allocation to the vital, intestinal, or reproductive organs.'' In summary, from this study, it was concluded that the immune response could be energetically costly, but that the cost of its maintenance was negligible.

2 Reproduction and energy allocation

Reproduction, in particular lactation, is the most costly activity in terms of energy expenditure in female mammals. Females attempt to maximize reproductive success by producing a large number of offspring and often use their own energy reserves to maximize reproductive efforts (Degen et al., 2002). This is particularly true when other energy demanding activities, such as immune responses against parasitism, are occurring concomitantly (Kam and Degen, 1993).

In reproducing mammals, the energy cost of parasitism can be paid by the offspring by reduced litter mass and/or growth rate (Arendt, 1985; Richner et al., 1993; Hollmen et al., 1999) and/or the parents by reduced body energy and/or survival and/or future reproductive performance (Brown et al., 1995; Richner and Tripet, 1999; Fitze et al., 2004), and/or by increased levels of resource acquisition by parents (Tripet and Richner, 1997; Thomas and Shutler, 2001; Tripet et al., 2002). Many studies on parasitism and reproductive success have been done on birds and fish (Moller, 1990; Tompkins et al., 1996; Allander, 1998; Kopachena et al., 2000). However, little is known about the interactions between parasites and reproductive success in mammals (Lehmann, 1993; Murray et al., 1998; Neuhaus, 2003).

A model describing energy allocation of the total energy expenditure (TEE) of the reproducing mother during reproduction could be presented as:

$$\mathrm{TEE} = \mathrm{MEI} - (\mathrm{ER} + \text{energy for production})$$

where energy for production could be related to pregnancy (fetal energy) or lactation (milk energy). Parasitism, which can cause an increase in energy expenditure and/or a decrease in metabolizable energy intake (MEI), can lead to a decrease in energy production (fetus or milk). Alternatively, the female could mobilize of energy reserves (ER) to compensate for the increased energy demands.

If it is assumed that parasitism can have either a negative or negligible effect on the reproductive success of a host, than nine possible scenarios (Table 1) are envisioned for the parasitized reproducing females (P) compared to the non-parasitized female (N).

Table 1 Nine possible scenarios on the effect of parasitism on the reproducing female

Scenario #:	dependent variables			effect of parasites
	MEI	ΔM_b	Δm_b of litter	
1	P = N	P = N	P = N	no effect
2	P = N	P = N	P < N	no compensation
3	P = N	P < N	P = N	full compensation
4	P = N	P < N	P < N	partial compensation
5	P < N	P = N	P < N	no compensation
6	P < N	P < N	P < N	partial compensation
7	P < N	P < N	P = N	full compensation
8	P > N	P = Nor P < N	P < N	partial compensation
9	P > N	P = Nor P < N	P = N	full compensation

In these scenarios, three dependent variables are examined: metabolizable energy intake (MEI); body mass changes of the dam (ΔM_b); and growth rate of the litter (Δm_b). To compensate for energy used in fighting parasitism, infested females can increase energy consumption and/or increase the mobilization of body energy reserves (body mass loss) to the extent that they can either fully or partially support growth of their litter as compared with controls. Differences in growth rate (Δm_b) of the litter between treatment and control are indicative of differences in milk energy production. The effect of parasitim on the reproducing female can, therefore, result in one of four responses: 1) no effect of parasitism; 2) no compensation by the reproducing female; 3) partial compensation; 4) and full compensation. I did not consider a situation of improved reproductive success due to parasitism.

In scenarios one to four (Table 1), there is no difference in energy intake between parasitized and non-parasitized reproducing females. In the first, there is no effect on either the mass of the female or the litter and, therefore, no effect due to parasitism. In the second, there is no effect on the female, but the litter mass is reduced and, therefore, there is no compensation from the female for the added energy costs of parasitism. In the third, there is an effect on the female but not on the litter and, therefore, the female is compensating fully for the costs of parasitism by catabolizing body tissue. In the fourth, there is an effect on both the female and the litter and, therefore, the female is compensating partially for the added energy costs of parasitism.

In scenarios five to seven (Table 1), the energy intake of parasitized reproducing females is less than non-parasitized females. In the fifth, there is no effect on the dam but an effect on the offspring and, therefore, there is no compensation. In the sixth, there is an effect on both the dam and the offspring and, therefore, there is partial compensation. In the seventh, there is an effect on the dam but not on the offspring and, therefore, there is full compensation.

In scenarios eight and nine (Table 1), the parasitized females consume more energy than the non-parasitized females. In the eighth, there is (or is not) an effect on the dam and an effect on the offspring and, therefore, there is partial compensation. In the ninth, there is (or is not) an effect on the dam but no effect on the offspring and, therefore, there is full compensation.

2.1 Effects of macroparasites on reproductive success of free-living mammals

Neuhaus (2003) studied the effect of ectoparasites, mainly fleas, on reproductive success in free-living Columbian ground squirrels (*Spermophilus columbianus*), a social rodent living in colonies, in Alberta, Canada. These squirrels emerge from hibernation in late April when females breed. Gestation is approximately 24 days and young remain in the burrow for about 27 days. By mid to late August, all animals hibernate. All individuals in the study area carried some fleas and prevalence was high. Animals were trapped before mating and ectoparasites were removed from half the females using a commercially available flea and tick powder. Treatment was then applied weekly until the end of lactation. All females mated with between three to five males, and there was no difference in treatment. Body mass did not differ significantly between treated and untreated females when emerging from spring hibernation (458 g vs 474 g, respectively) or just after parturition (541 g vs 557 g, respectively), but was higher in treated than untreated females at weaning (560 g vs 518 g, respectively). During lactation, that is, from parturion to juvenile emergence, treated females gained 19 g while untreated females lost 40 g. Furthermore, treated females gained more mass than untreated females from emergence in spring to the emergence of young, both when litter mass was and was not included. Treated females weaned 5.25 young whereas untreated females weaned 3.6 young per litter and the number of young surviving to yearling age was higher for treated than untreated females (3.5 vs 2.0 young per litter, respectively). There was no difference between the treated and untreated groups in body mass per young at birth. But, because of the larger litters in treated than untreated females, mass of litter was higher in treated females. Neuhaus (2003) concluded that "it seems obvious that ectoparasites in my study area have a profound negative effect on individual reproductive success and on body mass." In addition, he concluded that ".. the constant presence of ectoparasites in these ground squirrels leads to a constantly lower reproductive success and survival of females and their offspring and probably affects the whole population, since all animals are infected to a certain degree." Untreated females lost body mass whereas treated females gained body mass and total litter mass was greater in the treated than untreated females. Consequently, scenarios 4, 6 or 8 could possibly describe these results. Energy intake of the female would be required to refine the prediction of the right scenario.

The effect of endoparasites on the fecundity of free-living mountain hares (*Lepus timidus*; adult body mass = 2–4 kg) in the Highlands of Scotland (Newey and Thirgood, 2004) was examined. Three groups of female mountain hares were studied: 1) treated with an anti-helminthic drug (subcutaneous injection of Ivermectin); 2) handled but untreated and 3) not handled and untreated. Following treatment, the treated group had less *Trichostrongylus retortaeformis* nematodes than the other two groups. There was no difference among groups in survival. In addition, there was no difference in body condition, measured as kidney fat index, among the three groups. However, fecundity, measured by the number of *corpora albicantia* in both ovaries, was significantly higher in the treated group than in the other two groups, showing an improved fecundity in the treated group. Scenarios 2, 5, or 8 could possibly describe these results as there is no effect on the reproducing female but there are reduced offspring. Again, energy intake of the females is not available to choose the exact scenario.

The effect of nematodes and nutrition on seasonal body condition and reproduction was studied in snowshoe hare (*Lepus americanus*; adult body mass = 1200 g) in south-central Manitoba (Murray et al., 1998). Nematodes in both males and females were reduced by a subcutaneous injection of an anti-helminthic drug (Ivermectin) and nutrition was manipulated by supplementation of commercial rabbit pellets. The hares were further divided into low, medium and high density groups. The treated hares had significantly less nematodes for four of five nematode species and the fifth nematode species (*Trichuris leporis*) was 35% lower in the treated group, but this difference was not significant. The body mass of non-treated male and female hares was significantly negatively correlated to nematode abundance in May to June only, indicating that the nematodes had a negative effect on the hares at that time. However, overall, neither parasite reduction nor food supplemenation affected the body mass of the hares although food x density interaction was significant from November to April. Food supplementation increased marrow fat level of males by 16%. However, parasite reduction and parasite reduction x food supplementation interaction had no effect. In females, neither food, parasitism nor their interaction had an effect on the marrow fat level. The reproductive status of males was determined by testes palpation mainly during May–August. The proportion of males with descended testes was similar among food treatments and parasite treatments and the interaction between them was not significant. Females had three to four litters during the breeding season that lasted between April and August. They were palpated for embryos from March to August. Food supplementation did not increase consistently the proportion of pregnant females but food supplementation x hare density interaction was significant, being higher in the moderate and high density groups. In addition, food supplemention resulted in a more rapid onset of pregnancy. Neither parasite treatment nor parasite x food interaction had an effect on pregnancy rates. Furthermore, examination of females in May–June showed that food supplemention increased the number of embryos, but the number of *corpora lutea* and conception dates were similar among treatment groups. Neither parasite reduction nor its interaction with food had an effect on either number of embryos, number of *corpora lutea* or conception dates. The authors concluded that ''the non-significant effect of nematodes on hare production may indicate the small overall cost of such parasitism to hares.'' They also concluded that the effects of parasitism might be evident with severe food restriction, which did not occur during the study. It seems as if there was no effect on the reproducing female due to parasitism in this study and if I assume that there was no effect on the litter mass, then either scenario 1 or 9 could possibly describe the results.

2.2 Effects of macroparasites on reproductive success of laboratory mammals

Kristan (2004) examined the effects of the intestinal nematode *Heligmosomoides polygyrus* on reproduction and on offspring growth in wild-derived house mice (*Mus musculus*) in which young were weaned at 20 days. Half the pups of the parasitized and unparasitized mothers were infected with nematodes and the growth rate of the pups followed for 60 days. Parasitized females had 45% larger litter sizes at birth (8.4 vs 5.8 pups) and 51% larger litters at weaning than unparasitized mothers (8.3 vs 5.5 pups). Pup loss between birth and weaning at 20 days did not differ between groups and there was no effect due to parasites on average time to first litter, on inter-litter intervals and on the success of weaning at least one pup per litter (93% and 89% for parasitized and unparasitized females, respectively). Pups at birth from parasitized mothers were larger than those from unparasitized mothers by 1.2%; however, pup growth to weaning was not affected by mother parasite infection. There was no difference in body mass or lean mass at 60 days between parasitized and unparasitized pups, but parasitized pups had 4% more fat than unparasitized pups. On a dry matter basis, parasitized pups had 5% greater liver mass, 40% greater small intestine mass but 8% smaller stomach mass and similar spleen mass than unparasitized pups. Interestingly, parasitized females produced more and larger offspring than unparasitized females in four consecutive litters; that is, improved reproductive success. In the scenarios that were presented, it was assumed that there was no effect or a negative effect on reproductive success and, therefore, results from this study do not fit any of the possible scenarios envisioned.

A similar experiment by Kristan (2002a) was done on laboratory mice (*Mus musculus*) in which the pups were

also weaned at 20 days. Pups from parasitized and unparasitized mothers received either low intensity nematode infection, high intensity infection or no infection (as controls). Body mass growth, maximum rate of body mass gain, tail length and foot growth of the pups were not affected by the maternal parasite condition, however, pups from parasitized mothers were 4% heavier than from unparasitized mothers when pups were growing at their fastest. In addition, parasitized pups grew 5% faster per day and reached their maximun growth rate 0.5 days earlier than unparasitized pups. Nonetheless, at 60 days of age, there was no effect on body mass or on body energy of the pups due to maternal or pup parasite treatment. However, in general, high intensity parasitized pups had larger livers, spleens and small intestines, but smaller kidneys than low intensity parasitized pups and unparasitized pups. This may suggest a "change in energy allocation to organs during growth or may simply reflect systematic morphological changes owing to parasite pathology." Here, again there appears to be a slightly positive affect on reproductive success due to parasitism in that the offspring from parasitized dams tended to be larger than offspring from non-parasitized dams and there was no effect on the parasitized female.

No effect on current reproduction was found in the laboratory rat (*Rattus rattus*) parasitized by the intestinal tapeworm *Hymenolepis diminuta* (Willis and Poulin, 1999). Rats were infected with eight cysticercoids (larval stage, which grew into adult worms in 3–5 weeks) and were then mated. Food pellets and water were available *at libitum*, but intakes were not measured. Litter size and mass did not differ between parasitized and nonparasitized mothers either at birth or at weaning (21 days). In addition, body mass change of parasitized and nonparasitized mothers were similar during lactation, both increasing in body mass. In this study, either scenario 1 or 9 could satisfy the findings.

Willis and Poulin (1999) concluded that "It would appear that parasite increases the relative value of the current litter and current levels of maternal investment, possibly because it reduces future reproductive success." It is possible that this holds true for the studies mentioned above. It would be interesting to determine the source of the added energy required for the current litter? However, this theory should be taken with caution as there was an increased litter production in parasitized wild-deprived house mice in four consecutive litters when compared to nonparasitized females, not only in the first litter (Kristan, 2004).

3 General conclusions

Studies on the effects of macroparasites on the energy budget of small reproducing mammalian hosts have not resulted in clear patterns. For example, responses have ranged from a reduction in reproductive success to no response to even an improvement in reproductive success. Furthermore, reproductive success in parasitized free-living and parasitized laboratory maintained small mammals appear to be quite different. It is evident that the impact of parasites on life-history traits in mammals is still poorly understood.

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