Chapter 19

PARASITES

Ryan F. Hechinger, Kevin D. Lafferty, and Armand M. Kuris

SUMMARY

1 The metabolic theory of ecology (MTE) can inform parasitology and vice versa. The host–parasite interaction is fundamentally metabolic. Parasites obtain all their energy and materials from hosts. This and other aspects of parasitism consequently impact a range of processes from the performance of individual parasites and hosts, to the organization of food webs in ecosystems. Because parasites comprise a massive component of diversity, considering them can help to test, refine, and expand MTE.

2 Parasites can differ from free-living species in basic attributes of energy metabolism. This might involve maximizing metabolic power instead of efficiency, something not generally realized.

3 Parasite life-history traits seem to scale with parasite body size. The scaling exponents appear to be similar to those characterizing free-living species. Host and parasite allometric scaling can be used to parameterize parasite population models. Measures of fitness can be extracted from these models to examine how parasite body size and consumer strategy might evolve in relation to host body size.

4 Maximum parasite abundance within hosts might scale with parasite body size to the −3/4 power while total parasite biomass within hosts scales with host body size. However, a thorough MTE framework has not been developed for parasite abundance within hosts.

5 The abundance of parasitic and free-living species in three ecosystems follows the same universal scaling relationship. But this is only apparent after accounting for the flow of energy among trophic levels. The universal −3/4 scaling of abundance implies the invariant production of biomass with body size across all life forms, regardless of functional group: ectotherm or endotherm, vertebrate or invertebrate, free-living or parasitic.

6 There are many, promising avenues for future research, both empirical and theoretical.

19.1 INTRODUCTION

Metabolic ecology uses individual metabolism as the foundation for a conceptual framework of ecology. This is natural, because we can consider metabolism to be the transformation and use of energy and materials by individual organisms, and much of ecology can be expressed as the flow of energy and materials within organisms and between them and their environment (see Brown, Sibly, and Kodric-Brown, Introduction). The metabolic theory of ecology (MTE) (Brown et al. 2004; Brown and Sibly, Chapter 2) puts metabolic

Metabolic Ecology: A Scaling Approach, First Edition. Edited by Richard M. Sibly, James H. Brown, Astrid Kodric-Brown.
© 2012 John Wiley & Sons, Ltd. Published 2012 by John Wiley & Sons, Ltd.

ecology to practice by capitalizing on the empirically documented scaling relationships of individual metabolism with body size and temperature. This enables prediction of broader-scale ecological patterns and processes with the use of few, easily measured variables, in particular, body size and temperature.

Parasitism is a consumer lifestyle and the term applies to all infectious agents (parasites), from viruses to multicellular animal and plant parasites (Kuris and Lafferty 2000; Lafferty and Kuris 2002). We can easily consider parasitism from a metabolic perspective. A parasite feeds on a single individual – the host – for the duration of its life (or life stage, for complex life cycle parasites), while a predator feeds on multiple prey. The durable nature of the parasitic association highlights the possibility of intense and intertwined metabolic interactions between parasites and their hosts above that expected between predatory consumers and their prey. There are several distinctive parasitic consumer strategies. These basic consumer strategies (which are taxon neutral) have different absolute and relative body sizes of parasites and hosts (Fig. 19.1A). The overriding importance of body size for these consumer strategies emphasizes the promise of a metabolic scaling approach to understanding the ecology and evolution of parasitism.

This chapter covers several areas that are relevant to MTE, parasites, and infectious disease. We first expand on the merits of incorporating parasites into MTE. We follow with a discussion of parasite energy metabolism, which is different from that of most free-living species. We then investigate several areas where MTE interfaces with parasitology by covering relevant research and reanalyzing data, spanning topics ranging from individual- to ecosystem-level patterns and processes. We conclude by summarizing and suggesting avenues for future research.

19.2 WHY CONSIDER PARASITES AND INFECTIOUS DISEASE?

19.2.1 MTE should shed light on parasitology

Parasites impact host individuals to varying degrees and can affect populations (e.g., Lafferty 1993; Hudson et al. 1998) and communities (e.g., Dobson and Hudson 1986; Mouritsen and Poulin 2005; Wood et al. 2007). Most aspects of parasite–host interactions are fundamentally metabolic or can be easily considered from a metabolic perspective. Like any consumer, parasites extract energy from their resources. For instance, 21% of an infected isopod's biomass production goes to the growth of a larval acanthocephalan parasite (Lettini and Sukhdeo 2010). Parasites also drain energy from hosts beyond what they consume. Hosts resist parasites in several ways (Rigby et al. 2002) and repair damage caused by parasites. These activities must incur metabolic costs. For example, juvenile chipmunks respond to botfly parasitism by increasing their resting metabolic rate by 8% per fly and they can have up to 6 botflies; the metabolic effects persist after the parasites have left and result in reduced body mass of adult chipmunks (Careau et al. 2010). Also, juvenile damselfish previously infested with a single gnathiid isopod micropredator had a 35% higher oxygen consumption rate than uninfected fish (Grutter et al. 2011). Metabolism also underlies other aspects of parasitism. For example, parasite manipulation of host behavior to alter predation rates (reviewed in Moore 2002) not only involves metabolic processes of the parasite and host, but also involves altering the flow of materials and energy in food webs. Thus, metabolic ecology and MTE can inform the ecology and evolution of parasites and infectious disease at multiple scales. Research covered in this chapter shows that metabolic scaling relationships for hosts and parasites can illuminate optimal parasite consumer strategies, optimal body sizes, and the scaling of abundance of all life forms in ecosystems.

19.2.2 Most species are parasitic and they change the game

Generalizations about life should pertain to most of life. From this standpoint, the increasing attention given parasites by ecological research is warranted because parasites likely comprise over half of species diversity (Price 1980; de Meeûs and Renaud 2002; Dobson et al. 2008). Considering parasites tests the universality of empirical generalizations and theoretical assumptions of research focused on free-living species.

Not only do parasites comprise a major component of diversity, but parasites differ on average from free-living species in ways that directly bear on MTE. For instance, as detailed in section 19.3.1, many parasites primarily rely on anaerobic energy metabolism. This is different than the aerobic metabolism that characterizes most free-living species. Parasites also differ from

free-living species in basic ecological attributes directly relevant to MTE. For example, MTE research has been used to help understand the flow of energy among trophic levels in ecological networks of free-living species (e.g., Brown and Gillooly 2003; Jennings and Mackinson 2003; Reuman et al. 2008; Petchey and Dunne, Chapter 8). Much of this work assumes consumer–resource body-size ratios to be constant and larger than 1, or the existence of a positive relationship between trophic level and body size. Although these assumptions may apply to some predator–prey interactions, parasites clearly violate those assumptions (Fig. 19.1). Parasites also greatly distort the relationship between trophic level and body size (section 19.5). As we will see, these typical differences between parasites and free-living species highlight the utility of using parasites not only to test MTE, but also to refine it in a way that enhances its performance for all life forms.

19.3 PARASITE INDIVIDUALS

19.3.1 The scaling of metabolic rate

MTE relies on understanding how metabolic rate scales with body or cell size (Brown et al. 2004; Brown, Sibly, and Kodric-Brown, Introduction). Metabolic rates influence ecological dynamics and patterns. As described in Chapter 2, equation 2.3, whole-organism metabolism, I, scales allometrically with body size, M, and exponentially with temperature as

$$I = I_0 \, M^{\alpha} \, e^{-E/kT} \tag{19.1}$$

In equation 19.1, I_0 is a normalization constant that varies for organisms of different physiological types (e.g., plants, endothermic vertebrates, ectothermic vertebrates, invertebrates) and α is the scaling exponent. Across a wide range of multicellular organisms, α has an average value of ~3/4 (e.g., Kleiber 1932; Hemmingsen 1960; Peters 1983). Recent work indicates that metabolic rates in bacteria scale with an exponent larger than 1 and in protists scale with an exponent ~1 (Makarieva et al. 2008; DeLong et al. 2010). Later in the chapter, we will use 3/4 scaling for host metabolic rates, as this applies to most host organisms studied and helps keep clear the identity of exponents. The $e^{-E/kT}$ term in the equation is a formulation of the Arrhenius equation, which can capture the influence of temperature on metabolic rate (Gillooly et al. 2001; Brown and Sibly, Chapter 2).[1]

Logging both sides of equation 19.1, so that $\log I = \log I_0 + \alpha \log M$, gives a linear form that makes data easier to visualize and analyze. Anti-logging the intercept provides the normalization constant and the slope provides the scaling exponent, both of which can be tested by linear regression.

Metabolic scaling studies of parasites have suggested that parasite metabolism scales allometrically, similar to free-living species. For several parasite groups, intraspecific whole-organism oxygen consumption scales with fractional exponents of body size, consistent with observations from free-living species (see Fig. 19.2 for examples). Plotting oxygen consumption against body size across four parasitic nematode species gives a scaling exponent of 0.62 (von Brand 1960). Figure 19.2D indicates that parasitic protists scale with the same exponent as free-living protists, but with a lower normalization constant. Similarly, in his classic comparative work, Hemmingsen (1960) included one parasite, the pig nematode, *Ascaris suum* (as *A. lumbricoides*). This parasite's metabolic scaling fit with the slope characterizing free-living species, with a below-average normalization constant.

However, there may be a major problem with the above scaling studies. They rely on metabolic rates approximated by quantifying oxygen consumption, with the assumption that the organisms rely on standard aerobic mitochondrial respiration.[2] But many parasites, including most of the species in the above scaling evaluation, do not rely primarily on aerobic energy metabolism. Most parasitic worms and protists do not completely oxidize fuel to carbon dioxide, even when in well-oxygenated environments such as the vertebrate bloodstream (von Brand 1973; Barrett 1981, 1984; Saz 1981; Kohler 1985; Bryant et al.

[1] In this equation, E is the activation energy for enzymatic reactions (~ 0.63 eV on average for aerobic respiration), k is Boltzmann's constant (8.62×10^{-5} eV K^{-1}), and T is the average operating temperature in kelvin (Gillooly et al. 2001; Brown et al. 2004). The units cancel out, leaving it as a dimensionless modifier.

[2] Aerobic respiration involves the tricarboxylic acid cycle, which generates high energy electrons ("reducing equivalents") by completely oxidizing carbon to CO_2, followed by ATP generation using the electron transport chain and the electrochemical proton gradient. Aerobic respiration occurs in mitochondria for eukaryotic organisms.

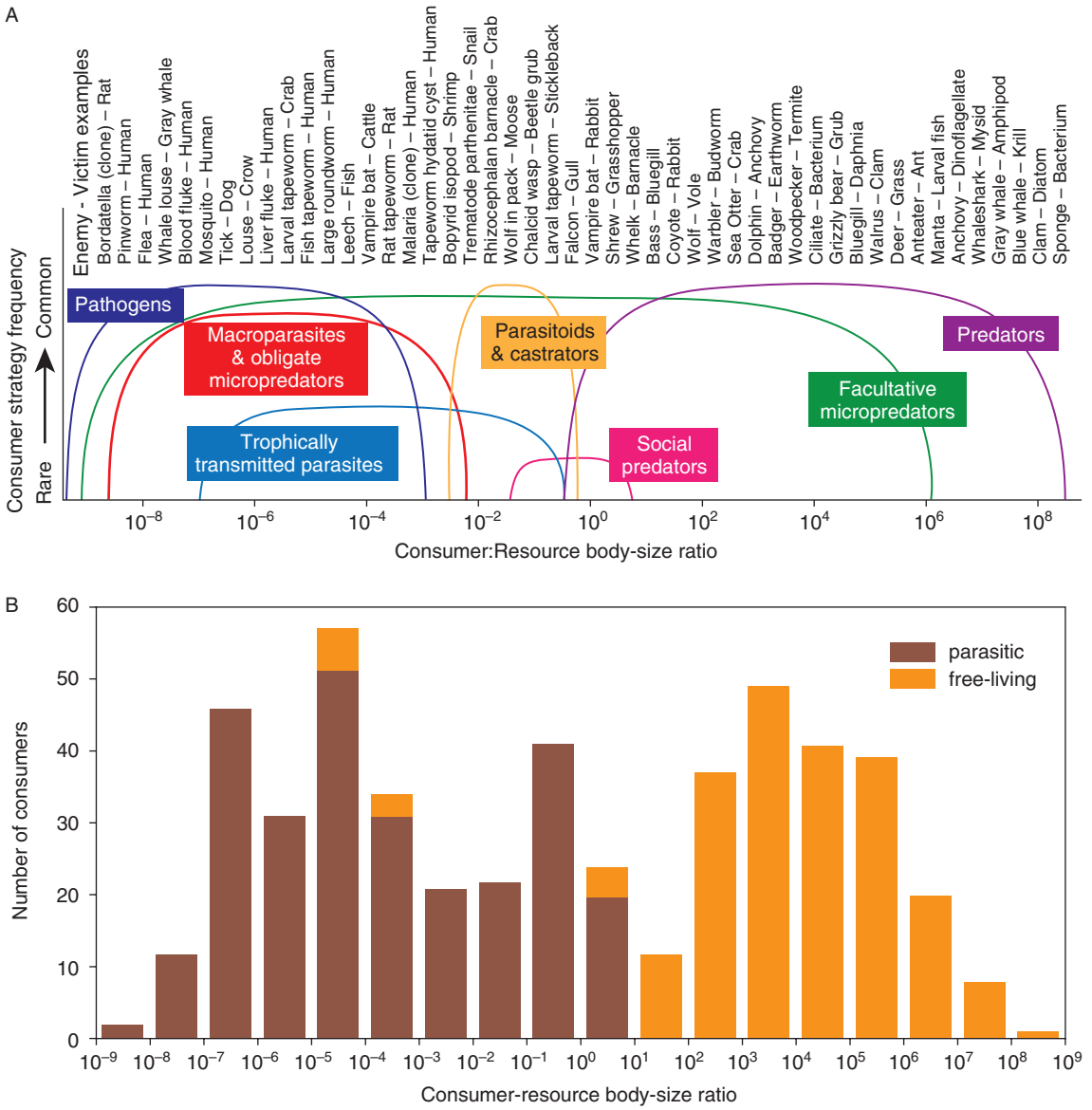

Figure 19.1 Consumer–resource body-size ratios for parasitic and free-living consumers. (A) Conceptual diagram representing the diversity of consumer strategies in nature. The *x*-axis represents consumer–resource body-size ratios on a log scale. The *y*-axis represents the authors' impression of the relative diversity of consumers employing different strategies. Examples of the various interactions are indicated above. Note that "trophically transmitted parasites" also includes trophically transmitted pathogens and castrators, which explains the large size ratios attained. Figure modified from Lafferty and Kuris (2002). (B) Actual frequency distributions of logged consumer–resource body-size ratios from three estuarine food webs. Figure pools the data presented for three estuaries by Hechinger et al. (2011). Values less than 0 are for consumers that are smaller than their resources. Note that body-size ratios vary among both parasitic and free-living species, and when parasites (and micropredators) are considered, body-size ratios are not restricted to being larger than 1. The log consumer–resource body-size ratio is slightly larger than 0 for several parasitic castrator trematode species, because these parasites grow to be 10–40% the mass of their snail hosts (Hechinger et al. 2009), tend to infect larger hosts (Sousa 1983), and consequently can have a slightly larger average size than the average host.

1989).[3] The main source of ATP for parasites is often glycolysis, or slight variants, none of which require oxygen. Even parasites that require oxygen still maintain relatively high rates of glycolysis. A lack of complete oxidation can also characterize parasites of invertebrates, where amino acid catabolism and aerobic respiration can be more important.

The lack of aerobic respiration reflects inefficient energy metabolism in terms of ATP extracted per unit resource. But parasites may gain by the strategy they adopt. Due to an inverse relationship between rate and efficiency, maximum metabolic power (ATP per unit time) is coupled to suboptimal efficiencies (e.g., Angulo-Brown et al. 1995; Waddell et al. 1997). Many readers have experienced this trade-off while driving cars varying in horsepower and fuel efficiency. Easy access to fuel can favor metabolic power gained by glycolysis over the efficiency of aerobic respiration (e.g., see Pfeiffer et al. 2001; Aledo and del Valle 2004; Vazquez et al. 2010).[4] Abundant resources and power maximization can explain the high glycolytic rates of some free-living microbes and fast-growing mammalian cells, including cancer tumor cells.[5] Parasites exist in conditions of abundant resources, potentially explaining why they are both productive and "inefficient" (Barrett 1984).

All this means that empirical scaling relationships based on oxygen consumption underestimate the normalization constants for parasite metabolic rates. This is because parasites generate more ATP than predicted from their oxygen consumption – for the majority of endoparasitic worms potentially about an order of magnitude more (Barrett 1991). Further, if parasites tend to operate under a different metabolic optimization regime than related free-living species, then they may also possess different scaling exponents (similar to how different metabolic constraints may explain the different scaling exponents characterizing bacteria, protists, and animals; DeLong et al. 2010). If parasites scale with different exponents, using standard scaling relationships will lead to inaccurate predictions about how parasite metabolic rates scale with parasite body size. What does this mean about including parasites in MTE analyses? Until high-quality investigations on the scaling of parasite metabolic rates are available, a sensible approach may be to use standard empirical scaling relationships with the expectation that they underestimate parasite metabolic rates relative to free-living species. We should also consider that parasite scaling exponents may differ from free-living species, tempering precise comparisons of parasites of different body sizes until better data and analyses are available.

19.3.2 Parasites and temperature

Just as for free-living species, temperature influences biological and ecological rates for parasites (reviewed in von Brand 1973, 1979). For instance, malaria develops faster in warmer mosquitoes and warmer mosquitoes bite more frequently. But warmer mosquitoes also die faster, and the net result is that there is an optimum temperature for malaria transmission (Anderson and May 1991; Rogers and Randolph 2000). MTE addresses temperature via its influence on metabolic rates (equation 19.1; Brown et al. 2004; Brown, Sibly, and Kodric-Brown, Introduction). Temperature correction in MTE is often carried out in comparative tests of ectothermic organisms from different environments. It also helps explain the faster biological rates characterizing endotherms. With parasites, we have the same concerns, but with fascinating differences. Parasites are ectothermic with body temperatures matching their hosts. Parasites of ectotherms therefore operate at near-ambient environmental temperature. Parasites living in the body of endothermic hosts have a consistently warm environment without paying the high costs of endothermy. Parasites living on the surface of endotherms will approximate the surface temperature of the host, which is often somewhere in-between ambient envi-

[3]This pertains to life stages that are parasitic (established on or in a host). Free-living stages of parasites typically use full aerobic respiration. Migrating parasitic larval stages are often somewhere in-between free-living stages and established parasitic stages. Further, almost all the metabolic research pertains to endoparasites; little is known about how ectoparasites metabolize.

[4]Biochemists have only recently appreciated that glycolysis may be thermodynamically optimized for power (e.g., Angulo-Brown et al. 1995; Waddell et al. 1997). We appreciate the power of glycolysis when we sprint and it provides over 100 times more ATP per second than aerobic respiration (Voet and Voet 1995).

[5]The increase in glycolysis characterizing many cancer cells is known as the "Warburg effect." This is also why positron emission tomography (PET) detects cancer cells. The radioactive tracer is a glucose analog, which is disproportionally taken up and trapped in cancer cells.

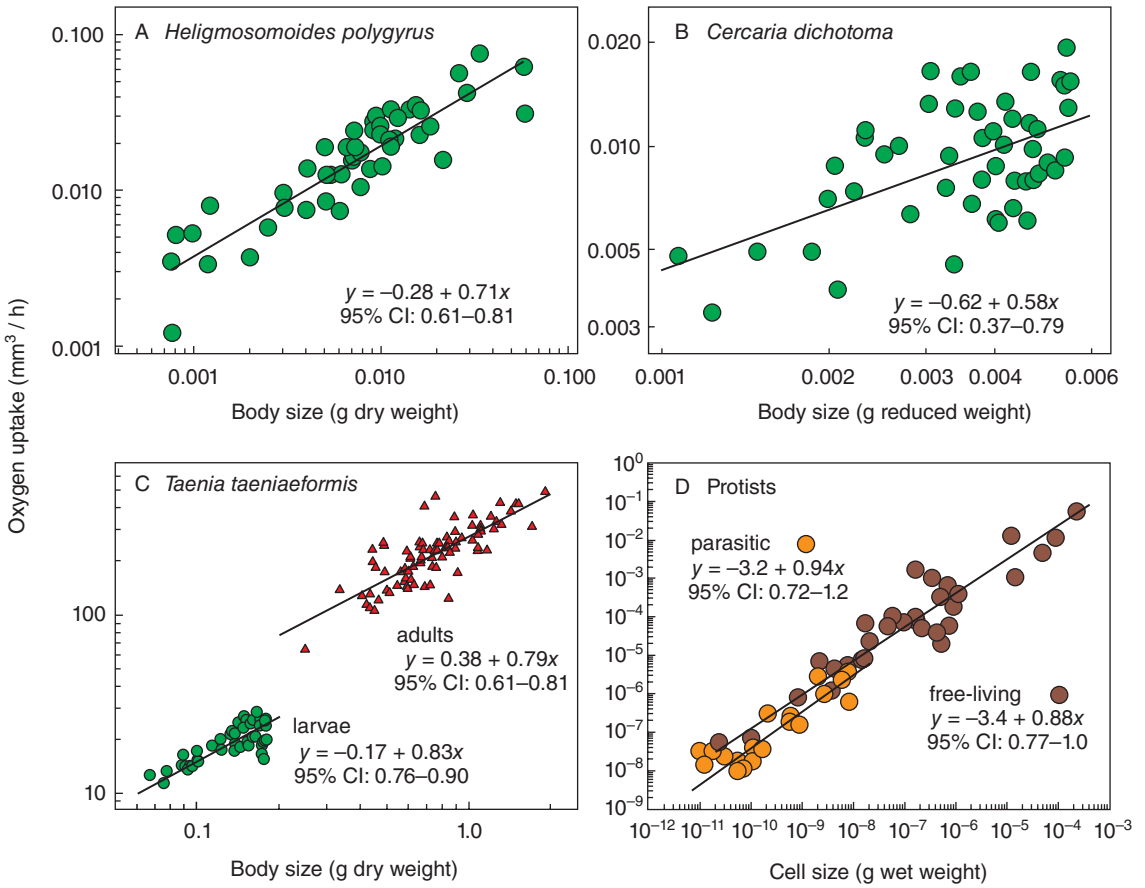

Figure 19.2 Scaling of "standard" or "endogenous" respiration rates (oxygen consumption) versus body size for parasites. (A) An intestinal nematode of mice, *Heligmosomoides polygyrus.* We reanalyzed data from Bryant (1974) Fig. 3a,b, pooling genders. (B) Individual parthenitae (sporocysts) of the trematode *Cercaria dichotoma*. Data from Pascoe et al. (1968). Note, data possesses extra scatter because worms were starved for varying amounts of time (1–19 days). For day 1 worms ($n = 10$), Pascoe estimated an exponent of 0.70 ± 0.03 95% CI. (C) Larval stages (cysticerci) from rat livers and adult stages from cat intestines of a tapeworm, *Taenia taeniaeformis.* Data from von Brand and Alling (1962) Figs 1 and 3. (D) Interspecific scaling of oxygen uptake versus cell size for heterotrophic free-living ($n = 32$) and parasitic protists ($n = 20$). Data from DeLong et al. (2010) (who used data from Makarieva et al. 2008, which itself was mostly compiled from a report by Vladimirova and Zotin 1985). We converted data to O_2 uptake, and used a general linear model on log-log data to test the significance of the different normalization constants for parasites and free-living species ($F_{1,49} = 10.1$, $p = 0.0025$), but then fit separate RMA regressions to each group following DeLong et al.'s analysis. We obtained all other estimates using OLS on log-log data.

ronmental temperature and internal host body temperature. An interesting twist arises for the many parasites with complex life cycles. Such parasites encounter several temperature regimes during their free-living stages and stages parasitic of endothermic and ectothermic hosts. These temperature differences should be appropriately accounted for when applying MTE to parasites. Additionally, the influence of these temperature differences on metabolism likely has interesting ecological and evolutionary ramifications (e.g., differences in rates of growth, interactions, and evolution).

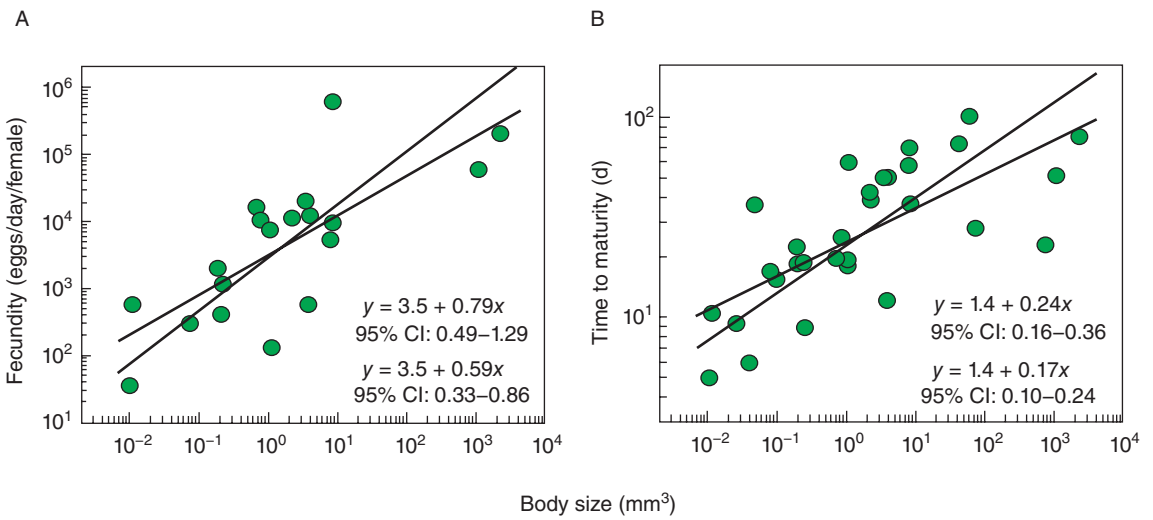

Figure 19.3 The allometric scaling of life-history traits for nematode macroparasite genera of mammal intestines using data from Skorping et al. 1991. (A) Fecundity (eggs/day/female) versus body size. We took data from Skorping et al.'s Fig. 2 fecundity vs. pre-patency plot, matched pre-patency data with data from their Fig. 1, and fit lines. Our OLS estimate matches well with the value that they reported in text. (B) Time to maturity (time post-infection to egg release) versus female body size. We took data from Fig. 1 of Skorping et al. (1991), flipped axes, and fitted lines. In both panels, the upper statistics and steeper lines are from RMA regression, whereas lower statistics and shallower lines are from OLS. 95% CIs are for exponents.

19.3.3 The scaling of life-history traits with parasite body size

Body size affects metabolic rates, which, in turn, set the pace of life histories (Brown et al. 2004; Sibly, Chapter 5). For instance, fecundity and maturation time scale with body size in free-living species (reviewed in Peters 1983; Calder 1984; Schmidt-Nielsen 1984). A few studies indicate that parasite life-history traits vary with body size as they do for free-living species. However, most of these analyses were not performed to examine the allometric scaling of life history, making it difficult to estimate scaling exponents and normalization constants. For instance, Loker (1983) compared life-history relationships among schistosome trematodes. But these blood flukes span less than an order of magnitude in size, limiting our ability to discriminate allometric from isometric scaling. Studies spanning a broader size range have tended to only use phylogenetically independent contrasts, report correlation coefficients instead of slopes and intercepts, and use genus- or family-level data. Additionally, several of these studies used length, or length × width, as a measure of body size for groups that vary considerably in body shape – copepods (Poulin (1995), trematodes (Poulin (1997), nematodes Morand (1996), and platyhelminths (Trouvé et al. 1998). Hence, although these studies suggest that parasite life histories scale roughly similarly to free-living species, their analyses and data make it difficult to estimate scaling exponents and coefficients.

However, two studies have reported life-history data appropriate for evaluating parasite allometric scaling relationships. Skorping et al. (1991) analyzed several life-history relationships for nematode macroparasites of mammals. They reported that fecundity allometrically scales with an exponent of 0.55 (95% CI: 0.35–0.75). The confidence limits barely reach the predicted exponent of 0.75. However, the slope was estimated using OLS.[6] This might underestimate the slope because error in body-size estimates could be close to the error in fecundity (because the data were means for nematode genera). We found that an RMA slope indicates an exponent of 0.79 (Fig. 19.3A). We were

[6]OLS refers to "ordinary least-squares" regression and RMA refers to "reduced major axis" regression (see White, Xiao, Isaac, and Sibly, Chapter 1). RMA provides steeper slope estimates than OLS, and is appropriate when error in estimates of the response (dependent) and predictor (independent) variables are of similar magnitude. OLS provides appropriate slope estimates when error in the response variable is at least three times larger than error in the predictor.

also able to use their data to show that time to maturity may scale as $M^{1/4}$, which is predicted by the allometric exponent for biological times (Fig. 19.3B). Also studying nematode intestinal parasites of mammals, Morand and Poulin (2002) reported $M^{0.76}$ scaling of daily fecundity versus body volume based on reanalyzing data from Morand (1996). These analyses suggest scaling exponents for nematode worms consistent with MTE predictions concerning the timing of life history, but additional studies on a broader range of parasites are needed to assess the generality of these findings for parasites.

19.3.4 The scaling of optimal parasite body size

Measures of parasite fitness are often derived from parasite population models that include both host and parasite dynamics. The scaling relationships between body size and various vital rates and demographic patterns permit us to express model parameters as allometric functions of host and/or parasite body size. Expressing model parameters as allometric functions of body size can enable exploration of parasite evolution, including optimal parasite body size.

Morand and Poulin (2002) explored how optimal parasite body size might vary with host body size for single-host life cycle nematode parasites of mammals. They applied allometric scaling to both hosts and parasites in a macroparasite–host population model. They solved for the body size that maximizes a measure of fitness, R_0, which is the parasite net reproductive rate when the parasite invades an uninfected host population. The equation for R_0 involves several parameters that scale with either parasite body size (parasite fecundity and mortality) or host body size (host mortality and density). They predicted that optimal nematode body length would scale with host mass to the 0.38 power and that optimal nematode body volume would scale with host body mass to the 0.94. Both predictions were supported by estimates derived from data on pinworm nematode parasites in their mammalian hosts: nematode length scaled with host mass to the 0.33 power (95% CI: 0.28–0.39) and nematode volume scaled to the 0.80 power (0.63–1.04). Inputting into their model the body sizes of North American mammals, they generated a predicted distribution of nematode parasite body sizes. The predicted mean was indistinguishable from the observed mean, and the observed and predicted distribution of lengths was grossly similar. These results imply that the effects of metabolic scaling on host and/or parasite demographic processes affect the evolution of parasite body size.

Kuris and Lafferty (2000) also used allometric scaling to model how R_0 responds to different host and consumer body sizes, comparing a variety of predatory and parasitic consumer strategies. Their modeling indicated that a predator strategy is most profitable on small-bodied resource species, while a parasitic strategy is better when eating large-bodied resources. This outcome was explained by comparing resource lifespan with consumer lifespan. If resource lifespan is short, it does not pay to form a durable feeding relationship and a consumer should immediately consume the entire resource. Relatively large and long-lived resources, however, can be "milked" over time, favoring a parasitic strategy. Parasitoids are able to infect hosts close in size to themselves because they eventually kill the host as part of their development. Parasitic castrators are able to infect hosts that are only a little larger than themselves because they primarily take energy that does not impair the host lifespan. However, the most profitable hosts for castrators tend to be absolutely small because such hosts devote relatively more resources to reproduction. More can be done with such models, including testing with empirical data on consumer–resource size distributions in nature (Fig. 19.1).

19.4 PARASITES IN HOSTS

MTE predicts population abundance by using the allometric scaling of whole-organism metabolism (Brown et al. 2004). If organisms of different body sizes have, on average, equal access to resources, steady-state abundance, N, should decline with body size. This is because a given amount of resources supports fewer large-bodied individuals than small-bodied individuals. Individual resource requirements parallel whole-body metabolic rates, which scale with M^{α} (see equation 19.1). Hence, holding resource supply constant, abundance will scale with body size as $N \propto M^{-\alpha}$. Recall that temperature effects can also be factored into the relationship (see equation 19.1).

Researchers studying free-living species often measure species abundance as the density of individuals per unit volume or area of habitat. However,

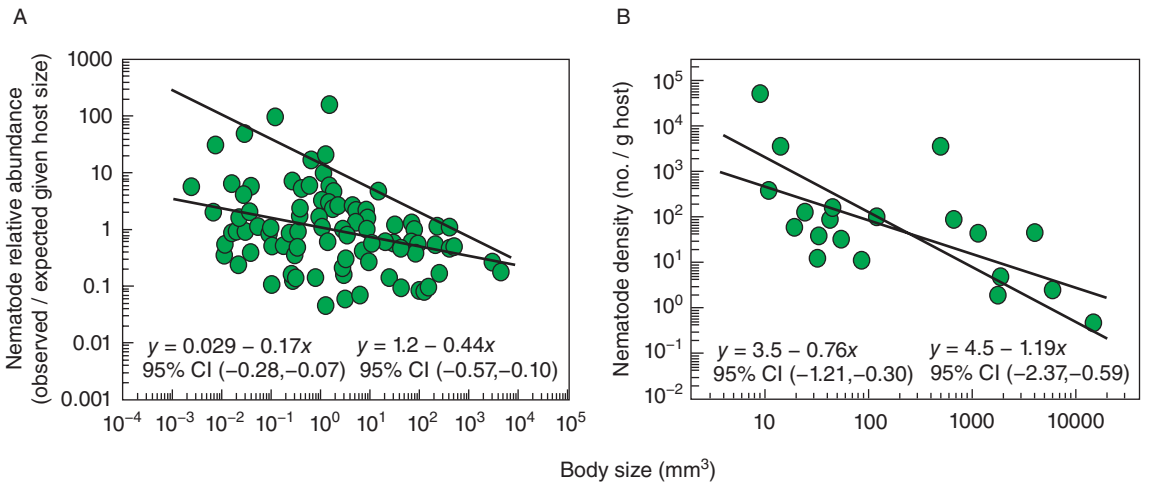

Figure 19.4 Parasitic nematode abundance within infected hosts versus parasite body size. (A) Relative abundance versus female body size for nematode parasites of mammals (data from Fig. 1 of Arneberg et al. 1998). Dependent data represent anti-logged residuals of log mean parasite numbers in infected hosts versus host body size. Arneberg et al. reported an OLS slope of –0.19 (95% CI: –0.31 to –0.07) which agrees well with our OLS estimate (left statistics). We also fitted a 95th percentile quantile regression to approximate the upper bound (right statistics). (B) Mean number of worms per gram of host tissue for nematode parasites of mammalian guts (data from declining part of the abundance distribution in Fig. 2b of Morand and Poulin 2002). Morand and Poulin used RMA to provide a slope of –1.19, which matches with our estimate (right statistics). We found that OLS, which could be more appropriate, provides a slope of –0.76 (left statistics). There is insufficient data for quantile regression to be stable over the 75th percentile, so we did not use it to estimate the upper bound of the data.

ecological parasitologists have traditionally used the natural unit of a single host body as the unit for parasite density (but see section 19.5). A group of conspecific parasites within an individual host is termed an *infrapopulation*, as distinct from the population of parasites in the entire host population or ecosystem (Bush et al. 1997). The suite of parasite species within individual hosts is called an *infracommunity*. Can we apply MTE to parasite infrapopulations and infracommunities?

19.4.1 Parasite infrapopulation abundance vs. parasite body size

Two studies examined the interspecific scaling of infrapopulation abundance. Does parasite abundance decline with $M^{-3/4}$ as it does for free-living animals?[7] Arneberg et al. (1998) examined data for nematode species parasitizing mammals. They used the mean number of parasites per infected host as the raw data for the dependent variable. They showed that OLS slopes were appropriate, because abundance error was over 11 times greater than body-size error. In a variety of analyses – including those using observed abundance data, abundance residuals corrected for host body size, and phylogenetic contrasts – they documented allometric slopes shallower than –3/4, ranging from –0.12 to –0.21. They also estimated a slope of –0.46 (95% CI: –0.70 to –0.23) for the uppermost boundary of the abundance data. This slope matches our estimation of an upper-bound slope for their global data (Fig. 19.4A). MTE predictions are more likely to apply to upper-bound abundance data because upper bounds are more likely to represent infrapopulations experiencing resource limitation. Morand and Poulin (2002) also examined nematodes of mammals,

[7]The few other comparative studies relating parasite abundance to parasite body size (e.g., Poulin 1999) cannot be used to evaluate hypotheses about MTE because they were performed for other purposes and do not provide the appropriate data (e.g., actual measures of abundance and body mass).

restricting their data to those that live in host intestines. They measured infrapopulation density as the mean infrapopulation abundance divided by host mass. In the analyses using the *maximum* mean infrapopulation density observed for each nematode species among host populations, Morand and Poulin detected a mode where nematode densities peaked at small but not the smallest body sizes.[8] They estimated an RMA slope of −1.19 for the declining part of the distribution (Fig. 19.4B). In analyzing their data, we found that OLS, which could be more appropriate given that density error likely swamps body-size error, provides a slope of −0.76 (Fig. 19.4B).

Although all these analyses document the negative scaling of abundance with parasite body size, findings using maximum mean infrapopulation densities from the second study might be more consistent with MTE predictions, as both RMA and OLS exponent confidence limits overlap −3/4.

19.4.2 Parasite infracommunity biomass vs. host body size

George-Nascimento et al. (2004) and Poulin and George-Nascimento (2007) examined the scaling, with host body size, of the total metazoan parasite assemblage standing-stock biomass (or biovolume) within a wide range of vertebrate host species.[9] In a variety of analyses (using raw data or residuals, with or without temperature correction) on their global dataset, the authors detected that mean total parasite biomass scaled with slopes ranging from 0.54 (95% CI: 0.36–0.71) to 0.82 (0.64–1.0).[10] However, analyses on the highest-quality subset of their data (ecto- and endoparasites of fishes) indicated that infracommunity biomass scaled isometrically with host mass. This was particularly evident when they incorporated temperature effects and used maximum observed parasite infracommunity biomass: exponent 0.97 (0.80–1.14) (Poulin and George-Nascimento 2007). Isometric scaling is consistent with volumetric space limitations, or with parasites metabolizing at the same rates as host tissues.[11] This research did not factor in parasite body size, which would be important if parasite metabolic rates scale with parasite body size as we expect from the discussion in section 19.3.2. If parasite metabolism scales with parasite body size, and if host metabolism imposes a ceiling on parasite abundance, a particular host would support a smaller biomass of small-bodied parasites than of large-bodied parasites, because smaller parasites require more energy per unit mass. It would be worthwhile to construct and test an MTE framework for within-host total parasite abundance, biomass, and energy flux that explicitly factors in parasite species' body size.

19.5 PARASITES AND FREE-LIVING SPECIES IN ECOSYSTEMS

The above sections consider parasite abundance within hosts. But parasite abundance can also be calculated using the standard spatial units used in ecology. Doing so permits a direct comparison of the abundances of parasites and coexisting free-living species.

The MTE equation for steady-state abundance of multicellular organisms with equal access to resources

[8]This parallels findings observed for some free-living animals that may reflect an optimal body size for the group (reviewed in Brown 1995). As described in section 19.3.4, Morand and Poulin (2002) explored this issue using macroparasite population models.

[9]Also note Muñoz and Cribb (2005), who presented data on total metazoan parasite biomass in 14 individual conspecific fish. They reported a positive correlation for log total parasite biomass and host mass. We estimated allometric exponents of 0.44 (95% CI: 0.21–0.67) using OLS and 0.72 (0.40–1.28) using RMA. However, host size is confounded by host age, and we cannot distinguish longer cumulative exposure time or body size as being the factor driving the positive association.

[10]They also observed the mathematically required reciprocal relationships obtained by dividing parasite biomass by host mass.

[11]The theoretical framework developed in the pioneering papers of George-Nascimento et al. (2004) and Poulin and George-Nascimento (2007) assumes that parasites metabolize at the same rate as host tissues, but makes a faulty prediction that the summed standing-stock parasite biomass will then scale with host whole-body metabolic rate, $M_H^{3/4}$. If parasite tissue metabolized at the same rate as host tissue, we could expect isometric scaling, as is found in the scaling of many host tissues and organs with host body size (Calder 1984). This can be further understood by considering that the amount of tissue supported by a watt scales with $M^{1/4}$ (the inverse of mass-specific metabolic rate). Consequently the amount of any particular host tissue – or parasite tissue metabolizing like host tissues – supported by host metabolic rate could scale as $M^{1/4}M^{3/4} = M^1$.

Note: The original publication erroneously has a negative before the 1/4 exponent here.

captures the decline in abundance with the increase in body size as

$$N = iM^{-3/4}\, e^{E/kT} \tag{19.2}$$

where i represents a normalization constant (Brown et al. 2004). Dividing both sides of the equation by the temperature-dependence term gives "temperature-corrected abundance," $N\, e^{-E/kT}$.

Hechinger et al. (2011) plot abundance versus body size for parasites alongside free-living species. They used data from three estuaries for metazoan parasites and a wide range of free-living species belonging to different physiological groups – invertebrates, ectothermic vertebrates (fishes), and endothermic vertebrates (birds). Plots in log-log space of observed or temperature-corrected abundances versus body size show that equation 19.2 does not predict the scaling exponents and normalization constants for either parasitic or free-living species (Fig. 19.5A). For instance, parasites are less abundant than expected and have slopes that are too shallow.

The authors hypothesized that discrepancies in the data could be explained by trophic transfer efficiency, which is the fractional transfer of energy among trophic levels (Lindeman 1942; E. P. Odum 1971). Some MTE research has factored trophic transfer efficiency into abundance scaling (e.g., Brown and Gillooly 2003; Jennings and Mackinson 2003; Reuman et al. 2008; see also Petchey and Dunne, Chapter 8), but most of this work assumes consumer–resource body-size ratios to be constant and larger than 1, or the existence of a positive relationship between trophic level and body size. These assumptions may apply to some predator–prey interactions. However, parasites (and many micropredators, like mosquitoes) violate the "big consumer" assumption by flipping the typical consumer–resource body-size ratio, and by altering the distribution of these ratios in empirical food webs (Fig. 19.1). Including parasites also changes the trophic level versus body-size relationship in empirical webs (Fig. 19.6), further indicating the need to incorporate into theory the flow of energy among trophic levels independently of body size.

Although rarely done (e.g., Brown et al. 2004; Meehan 2006; McGill 2008), it is possible to incorporate into scaling theory the effects of trophic level and trophic transfer efficiency in a way free of assumptions concerning body-size relationships. Noting that trophic transfer efficiency, ε, can be expressed as a fraction, Hechinger et al. predicted that abundance would exponentially decrease with increasing trophic level, L, as

$$N\, e^{-E/kT} = iM^{-3/4}\, \varepsilon^{L} \tag{19.3}$$

where the basal level is defined as $L = 0$.

This incorporation of trophic dynamics revealed uniform −3/4 ecosystem-wide scaling of abundance with body size across all parasitic and free-living species (Fig. 19.5B). This remarkable finding highlights the utility of using the ε^{L} term to provide broadly applicable scaling relationships. After accounting for the flow of energy among trophic levels, all species clustered around a single −3/4 line, regardless of taxonomic or functional group affiliation: endotherm or ectotherm, invertebrate or vertebrate, free-living or parasite.

Hechinger et al. (2011) also examined an implication of the observed uniform −3/4 scaling of abundance with body size – that of the invariant production of biomass with body size. Because a single regression line roughly describes the $M^{3/4}$ scaling of temperature-corrected individual biomass production, P_{ind}, for a wide range of multicellular species (Ernest et al. 2003), total population production, P_{tot}, is $P_{\text{ind}}\, N$, which scales as $M^{3/4}M^{-3/4} = M^{0}$. Data from the three estuaries suggest that any species within a specific trophic level can produce biomass at the same rate, whatever their body size or functional group (Fig. 19.5C; Hechinger et al. 2011). This "production equivalence" appears to be a more general rule than the often discussed "energetic equivalence" rule that only applies to metabolically similar organisms (e.g., see Damuth 1981, 1987; Nee et al. 1991; Ernest et al. 2003; Brown et al. 2004). Concerning the importance of parasites to ecosystems, production equivalency reveals the comparable ecological relevance of a parasitic species compared to any free-living species existing at the same trophic level.

19.6 CONCLUSIONS AND FUTURE DIRECTIONS

We have surveyed the literature directly relevant to MTE and parasites, including basic aspects of parasite metabolic scaling. Applying MTE to parasites can inform several aspects of ecological and evolutionary parasitology. Conversely, considering parasites provides a novel way to test, refine, and expand MTE. Our

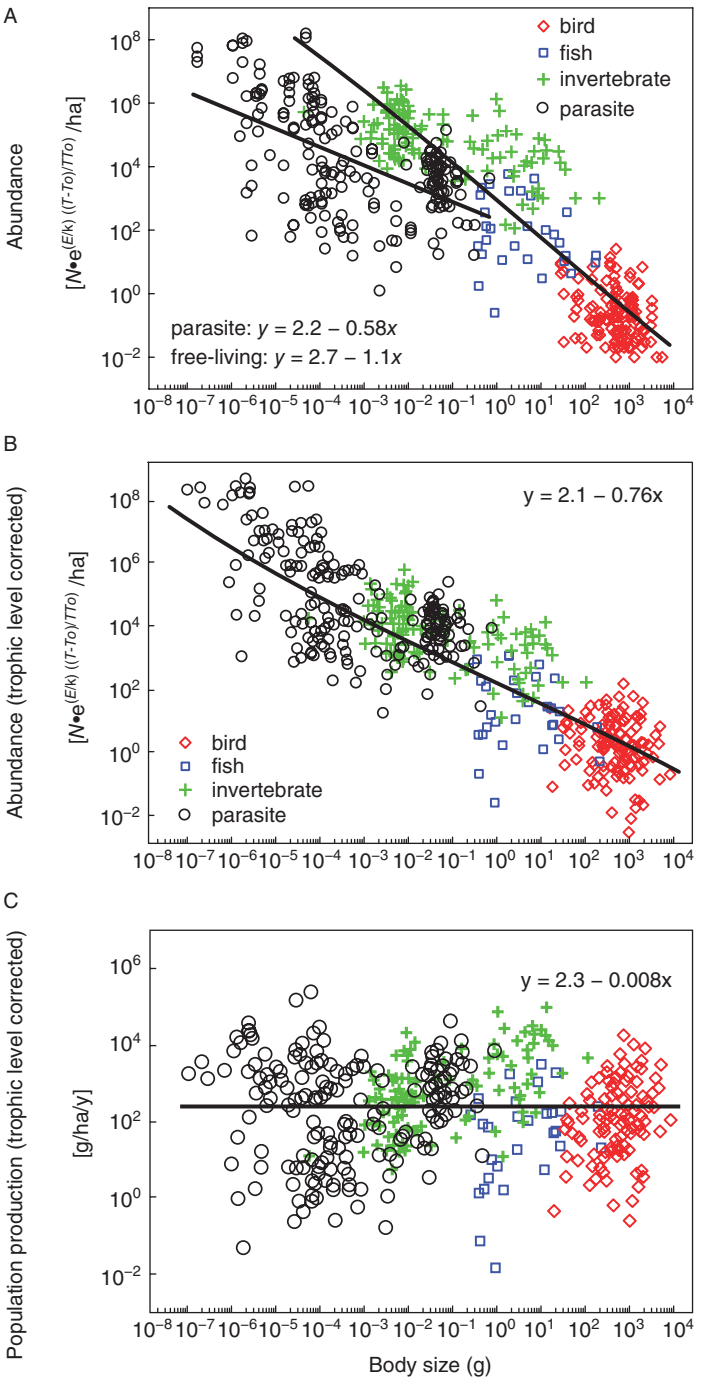

Figure 19.5 The scaling of abundance and production for parasitic and free-living species from three estuaries. (A) Temperature-corrected abundance versus body size for a wide range of parasitic and free-living species. A single line does not describe the relationship: parasites appear to scale less steeply than −3/4 and free-living species more steeply. (B) Temperature-corrected abundance decreases with body size to the −3/4 power when statistically holding trophic level constant. A single line now describes the various animal groups. (C) Total population production is invariant with respect to body size across all body sizes and animal groups when statistically holding trophic level constant. The data in (A–C) are from Hechinger et al. (2011), but pool the data from three estuaries. The estuary effect and interactions were not apparent and not significant in all cases (all $p > 0.73$). The temperature correction term in (A) and (B) used the Arrhenius equation expressed relative to the ambient estuarine temperatures.

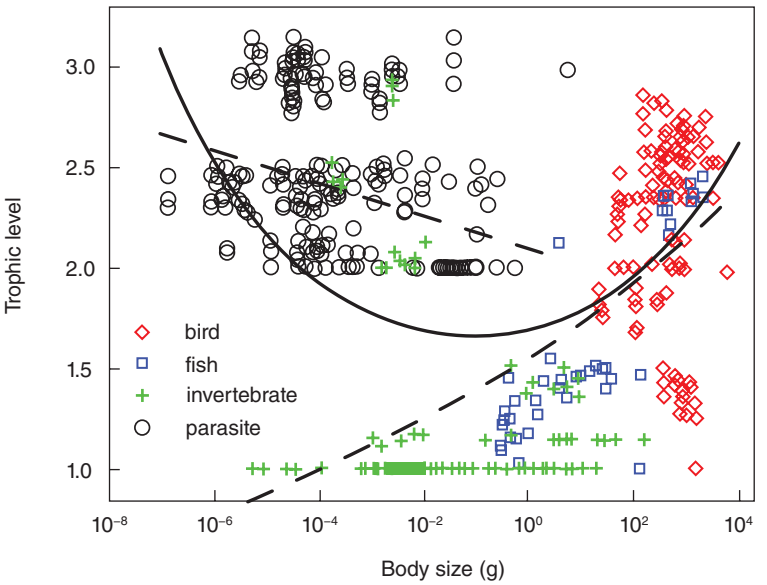

Figure 19.6 The relationship between trophic level and body size for the parasitic and free-living consumer species in three estuarine food webs (A–C). The dashed lines represent Poisson regressions fitted separately to parasites and combined free-living species (birds + fish + invertebrates) and the solid line represents the fit to the pooled data (with a quadratic term for body size). From Hechinger et al. (2011), using data pooled from three estuaries. The estuary effect was very weak and non-significant ($p = 0.60$).

review of the literature (we apologize for any oversights) and new analyses support these contentions. For instance, research that considered parasites simultaneously with free-living species showed how adding trophic dynamics into abundance versus body-size scaling increases the generality of MTE. This extension of MTE adds the flow of energy among trophic levels in a way that appears to perform well for all species. Continued application of MTE to parasites may help further generalize the theory.

There is much more to be done, both in terms of including parasitic species alongside free-living species in MTE and in applying an MTE framework to parasites and infectious disease. We need more, high-quality analyses of basic individual-based scaling relationships for parasites (metabolic rates and life-history traits). A sound understanding of parasite individual metabolic scaling will facilitate the use of MTE to estimate and predict the role of parasitism for hosts and ecosystems.

It would be worthwhile to construct a quantitative MTE framework based on parasite metabolic scaling to estimate and predict within-host parasite abundance, biomass, and energy flux. Such a framework could facilitate estimating the energetic impacts of parasites on hosts. This could help fill a hole in MTE, which currently does not account for the unobserved energetic burden of parasites on free-living species. Such an MTE framework could also provide a perspective on infectious disease previously unavailable to medical and veterinary science. Metabolic scaling may enable prediction of the impacts of infectious agents on hosts. For instance, although we did not cover it here, there is evidence that the allometric scaling of host biological times ($\sim M^{1/4}$) sets the pace of disease symptoms caused by microbial pathogens (Cable et al. 2007). Does disease caused by other sorts of parasites also scale with host biological times? Or does it primarily scale with parasite metabolic rates and population numbers? Expanding this work may help to predict the impacts of diseases when we are lacking data, including new infectious diseases of humans or livestock. Research can also begin to factor in the scaling of host immune responses, which may not simply scale with host body size to the 1/4 power (e.g., see Wiegel and Perelson 2004).

With appropriate extrapolation, an MTE framework for parasites could indicate direct energetic impacts of parasitism for entire populations, communities, and ecosystems. Section 19.3.4 showed that we can incorporate host and parasite allometric scaling into parasite–host population models to ask questions about parasite body-size optimization. However, expressing model parameters as allometric functions of body size also permits exploration of how other model outcomes scale with host or parasite body sizes. For instance, although not covered here, De Leo and Dobson (1996) showed that important rates of parasite transmission may scale with host body size (and see extensions in Bolzoni et al. 2008a, 2008b). One obvious benefit of incorporating MTE into parasite–host population models would be the inclusion of temperature effects. It would also be worthwhile to explore the interface between MTE and parasite diversity within hosts, something we did not investigate here. Finally, the research in section 19.5 shows the promise of MTE for the quantification and prediction of the role of parasites in entire communities and ecosystems. We hope that the scope and content of this chapter convey the promise and excitement of this area of research for ecology, parasitology, and medical and veterinary science.