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ORIGINAL ARTICLE

Energetic trade-offs and feedbacks between behavior and metabolism influence correlations between pace-of-life attributes

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Abstract

Correlations between behavioral, physiological, and morphological traits linked to life history have been given the label "pace-of-life syndrome" (POLS), hypothesized to arise through variation in the resolution of a trade-off between present and future reproduction. However, other trade-offs over energy allocation may also have effects and influence the present-future trade-off. We analyzed an optimality model of basal metabolic rate (BMR) across variation in food availability and two types of mortality. The model contained three major features: (1) feedback between activity and energy acquisition, (2) links between BMR and the use of energy for other traits, and (3) allocation trade-offs between BMR and all other traits, between activity and defense, and between defense against activity-related risk and activity-independent risk. The model produced an intermediate optimal BMR that was usually highest at an intermediate level of food availability. Food availability and both types of mortality risk interacted to influence the exact value of optimal BMR. Trait correlations expected in the POLS existed under some environmental conditions, but these correlations flipped sign under different conditions and were not always strong. Our model reproduces trait correlations consistent with the POLS, but also generated a "sloppy" syndrome with considerable non-POLS-like variation. In addition, among-individual, non-adaptive variation in BMR produced adjustments of the other traits. These fit a best-of-a-bad job strategy, and the adjustments further weakened trait correlations. The results emphasize that variation in resources and mortality risk creates a diversity of correlation structures. This complexity means the POLS is likely to be a variable construct.

Significance statement

Many attributes important for reproduction and survival are associated. Such associations may arise through common physiological processes and correlated selection. We modeled metabolic rate within a system in which foraging behavior both depended on and mediated the acquisition of resources necessary for metabolism, while energy was allocated among multiple attributes. Variation in several environmental variables (food availability and two types of mortality risk) influenced basal metabolic rate, activity, and defenses against mortality risk. This variation affected the correlations between the traits in complex ways. When basal metabolic rate was non-optimal, evolution of the allocation of energy to other traits partially compensated, but this further eroded consistent trait correlations. Our results indicate that complexity in how energy is acquired and used can potentially disrupt trait correlations normally associated with the pace-of-life syndrome.

Keywords Life history · Syndrome · Activity · Energy allocation · Conflicting demands · Optimization

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Introduction

Many organisms exhibit correlated suites of characters, such as fast growth rates or rapid and early reproduction, higher activity, greater risk-taking, higher aggression, lower allocation to immune responses, and a higher basal metabolic rate (BMR), while others feature an opposite set of characters (Careau et al. 2009; Réale et al. 2010). Such correlations among characters may exist both within or between species and have been labeled the "pace-of-life syndrome" (POLS; Ricklefs and Wikelski 2002; Biro and Stamps 2010; Réale et al. 2010; Careau and Garland 2012). The POLS thus

characterizes suites of traits along a fast-slow continuum, and indeed, comparative studies in fish (Goodwin et al. 2006; Bjørkvoll et al. 2012), reptiles (Bauwens and Diaz-Uriarte 1997), birds (Sæther 1987; Sæther and Bakke 2000), mammals (Oli 2004; Gaillard et al. 2005; Bielby et al. 2007), and even plants (Adler et al. 2014; Salguero-Gómez et al. 2016) reveal correlated traits linked to life history. The idea of a POLS is compelling because it integrates aspects of physiology, behavior, reproduction, and performance in a suite of traits that may form the central explanatory axis for much of the diversity in living organisms.

The dominant hypothesis to explain the correlated nature of these suites of traits is that selection acting on components of a trade-off between current and future reproduction produces correlated selection on many traits, favoring combinations that perform best (e.g., Réale et al. 2010; Galliard et al. 2013; Royauté et al. 2015). Conditions favoring current reproduction thus favor "fast" life histories and associated traits, whereas conditions favoring long-term survival, maintaining high residual reproductive value, favor "slow" life histories (Ricklefs and Wikelski 2002; Biro and Stamps 2010; Réale et al. 2010; Mathot and Frankenhuis 2018, topical collection on Pace-of-life syndromes). An alternative, but not mutually exclusive hypothesis, is that because metabolism provides the energy needed to express multiple kinds of traits, strong positive correlations might exist with metabolism (Careau et al. 2008, 2010, 2011; Krams et al. 2013; Turbill et al. 2013; Shearer and Pruitt 2014; Mathot et al. 2015, but see Le Galliard et al. 2013; Mathot et al. 2013; Gifford et al. 2014; Royauté et al. 2015). For instance, circumstances favoring a higher metabolic rate would produce more energy for a variety of traits such as activity, immune function, and reproduction (Burton et al. 2011; Martel et al. 2014).

Although the idea of a syndrome linking life history, behavior, and physiology captures what we generally know about variation among species in the array of attributes commonly comprising the POLS, two recent reviews and several empirical studies suggest that the POLS idea over-simplifies what may be a more loosely linked constellation of traits. Réale et al. (2010) noted multiple contrary examples in which a predicted component of the POLS did not vary as expected, in both intra- and inter-specific studies. For example, risk-taking in the face of predation was not associated with most life history characters in a comparative study of birds (Blumstein 2006) and in a separate study was negatively associated with BMR (Møller 2009). Závorka et al. (2015) did not find support for the POLS hypothesis in brown trout (*Salmo trutta*). Instead, they posited that food availability played a key role in determining the relationship between growth rate and activity level, and cited other empirical work that supports this claim (Biro et al. 2004, 2006; Adriaenssens and Johnsson 2011; Höjesjö et al. 2011). Equivocal support such as this suggests that either the idea of POLS is flawed in some way or that other complexities of the traits involved are not adequately incorporated into the idea (Montiglio et al. 2018, topical collection on Pace-of-life syndromes).

Careau and Garland (2012) presented several ideas for why imperfect or "sloppy" syndromes might be expected. Although metabolic rate should positively correlate with all other activities that require energy (the "performance model"), it also depends on finite resources that must be acquired. Given trade-offs over how to acquire food and what to do with the energy produced (the "allocation model"; Careau et al. 2008), we expect antagonistic relationships between some attributes predicted to be positively correlated by the pace-of-life hypothesis. An intriguing possibility is that combinations of processes that fit either the performance or the allocation models (Careau and Garland 2012) may influence the suite of trait correlations that have been labeled as POLS.

To date, relatively little theory has been generated to explore how the present-future trade-off is associated with the POLS (Mathot and Frankenhuis 2018, topical collection on Pace-of-life syndromes) and none has explored how underlying trade-offs dealing with energy allocation might influence the POLS. The classic analysis of van Noordwijk and de Jong (1986) investigated the correlation between two life history traits and showed that its sign depended on the among-individual variation in resource acquisition (a condition of the environment) and allocation; however, their approach did not incorporate metabolism. Some more recent work included metabolism in models of behavior (e.g., Wolf and McNamara 2012; Houston and McNamara 2014). Wolf and McNamara (2012) found that including metabolism in a model of aggression led to stronger between-trait correlations.

Two recent models directly address energy metabolism and its effect on behavior. Houston (2010) derived the conditions affecting optimal resting metabolic rate (RMR) when a forager experiences predation risk while foraging. Einum (2014) modeled optimal RMR and explored how it tracked with variation in food availability. Both models contained two central constraints supported by empirical findings. First, behavior and metabolism were involved in a feedback relationship; energy could not be obtained without activity, so energy must be expended to acquire more energy. Second, they both included a correlation between RMR and activity or metabolism during activity, a mathematical version of the performance model (Careau et al. 2008), also called the "increased-intake model" (Careau and Garland 2012). By this mechanism, higher levels of activity demand a higher RMR, despite the fact that RMR is measured when there is no activity. The underlying reason proposed for this is that the systems functioning when the organism is active demand maintenance. For example, the performance of a cardiovascular system capable of allowing more strenuous activity for longer periods of time may require more energy while at rest. Similarly, a higher food intake needed to support higher activity demands more

capacity for digestion, by way of larger alimentary tract organs, such as the gut, intestines, and liver, which have high metabolic costs and contribute significantly to BMR (Konarzewski and Diamond 1995; Nespolo et al. 2002; Konarzewski and Książek 2013). The models thus captured the essential feedbacks that occur between metabolism and activity.

Houston's (2010) and Einum's (2014) models, despite some structural differences, produced several intriguing results. First, optimal RMR was neither zero nor very large, but intermediate, reflecting the trade-off between the benefit of increased energy intake with increased activity versus the cost of increased energy demand due to RMR. Einum (2014) further found that optimal RMR varied across food availabilities and was highest at an intermediate food availability, presumably because the activity necessary to acquire the more abundant food required less RMR in support, since it was easier to acquire. This contrasted with a previous hypothesis, which posited that individuals with high RMR should be at an advantage when environmental conditions are favorable (e.g., food availability is high, Biro and Stamps 2010; Burton et al. 2011). It also provided a compelling ecological hypothesis for variation in RMR, both among individuals and among species, documented in numerous taxa (Gillooly et al. 2001).

Both Houston (2010) and Einum (2014) necessarily simplified the potential selection acting on metabolism. Several well-known complexities concerning energy allocation might affect the ways selection acts on metabolic rate and could impact our understanding of variation in attributes linked to POLS. For example, the energy provided by metabolism is used for a variety of fitness-enhancing traits besides foraging activity, as recognized by Careau et al. (2008). Houston (2010) incorporated the well-known fact that foraging activity exposes the organisms to mortality risk via predators (e.g., Sih 1987; Lima and Dill 1990; Werner and Anholt 1993; Skelly 1994) or parasites (Barber and Dingemanse 2010; Wengström et al. 2016), but the effect of this was to shift activity budgets from being active to non-active with corresponding effects on RMR. However, energy expenditure and the mechanisms for its acquisition are subject to an expanded array of trade-offs concerning either the allocation of energy or opposing fitness effects of how it is acquired, including effects on survival as well as reproduction and/or growth. We thus explore how multiple trade-offs involving energy allocation influence some of the attributes linked to the POLS.

We build on Houston's (2010) and Einum's (2014) basic approach and develop a model of optimal metabolic rate that places metabolism at the heart of a set of potentially correlated traits, but with feedbacks and allocation decisions creating some necessary positive and negative relationships among them. These allocations affect traits that impact total fitness, which is a product of survival and reproduction, and so we explore the role of energy allocation underlying the trade-off between present and future reproduction. However, we focus on complexities in the selection acting within a life stage or across a lifetime but with constant schedules of reproduction or mortality to keep the model tractable. Also, to better align our terminology with that of metabolic physiologists (e.g., McNab 1997; Speakman et al. 2004; White and Kearney 2013), we model BMR. BMR is distinguished from RMR by being the metabolism that occurs when an endotherm is thermally neutral, at rest, non-reproductive, and post-absorptive (not digesting any food) in order to better represent a minimum, mandatory metabolic cost of maintenance.

We asked four questions of our model:

1. Does optimizing multiple allocation decisions regarding the energy obtained from metabolism affect the optimal BMR?
2. How does selection act on BMR in environments that vary in both food availability and risk of mortality?
3. If food supply and multiple sources of mortality matter, how might trait correlations that typically comprise the POLS change with environmental variation in resources and extrinsic mortality risk?
4. How might non-adaptive variation in BMR, either due to selection for a different optimum in the past or from among-individual variation, affect the other traits involved in energy allocation?

Answering these questions provides new insight into processes affecting both the evolution of metabolic rate and within- and among-species variation in correlated suites of traits linked to POLS.

Methods

The Model

The core of our model is an energy budget for a hypothetical organism (Fig. 1). The organism takes in food, from which energy is obtained at a particular assimilation rate, S, then partitions that energy among various functions. Basal metabolic rate, B, as discussed above, is an obligate cost that the organism must pay to maintain essential anatomical structures and physiological systems, so the energy consumed through BMR is deducted from the assimilation rate first. What remains is the net energy gain rate, N, of the organism, which is allocated among activity, A, defense, D, and growth/reproduction, G. To maintain tractability and avoid non-essential complexity, we do not distinguish in this model between growth and reproduction, but subsume them both into the productivity term, G. These relationships can be expressed as

$$N = S - B \quad (1)$$

and

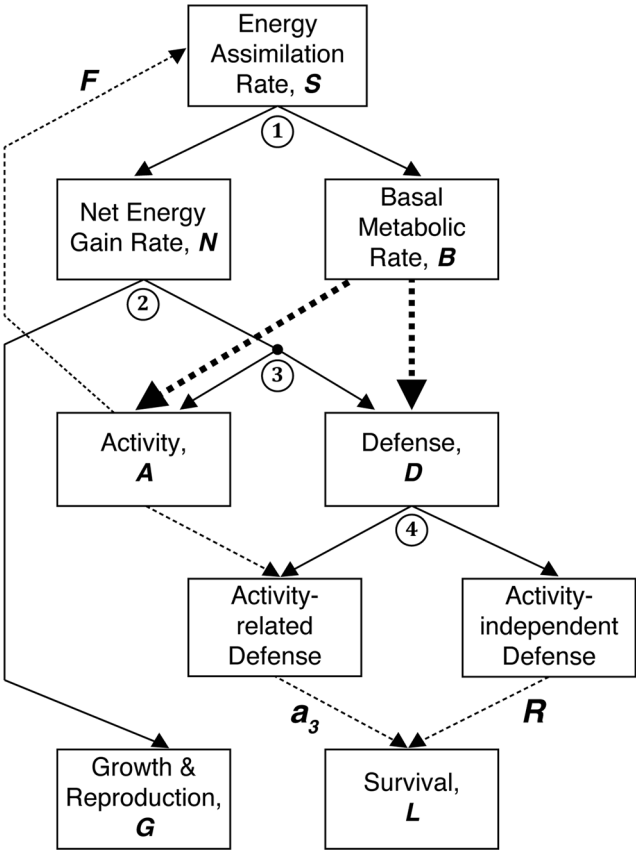

Fig. 1 Conceptual framework of the model as an extension of Fig. 1 in Biro and Stamps (2010). Solid arrows indicate energy flows, and dashed arrows show quantitative causal relationships, with equation numbers referring to the equations in the text. The bolded, dotted lines indicate the fundamental constraint that basal metabolic rate imposes on maximum activity and defense. The four key trade-offs over the allocation of energy are indicated by numbers; each proposes that a unit of energy can be used for one or the other task shown in the downstream box. Equations described in the text define key relationships; Eq. 3 defines the assimilation rate (S), Eq. 4 the constraint of BMR (B) on activity (A), Eq. 5 the constraint of BMR on defense (D), and Eqs. 6 and 7 define the effects on survival (L). Optimal basal metabolic rate and all downstream allocation decisions combine to affect fitness (the product of excess energy going to growth/reproduction (G) and survival (L), Eq. 8), and the model solves for the combination of trait values that maximizes fitness in a given environmental context

$$G = N - A - D. \tag{2}$$

Activity is the "general level of physical activity of an individual in terms of muscular movement leading to locomotion" (Careau and Garland 2012). In this model, activity is the rate at which an organism expends energy by moving through its environment. We assume that our organism feeds opportunistically as it moves, so that assimilation rate is a function of activity: more active individuals move through their environment more quickly and thus assimilate food more rapidly (Fig. 1). Assimilation rate also increases with increasing food availability in the environment, since in this case, an organism will encounter and consume more food items over the same movement distance.

We model the assimilation rate, S, of energy by the organism as the assimilation efficiency, ε (energy food^{-1}), multiplied by the feeding rate, expressed as a modified type II functional response function of food availability, F (food area^{-1}) (Holling 1959):

$$S = \frac{\varepsilon\theta AF}{1 + \gamma\theta AF}, \tag{3}$$

where γ is the handling time of food items (time food^{-1}) and θA is the attack rate, expressed as the product of the attack coefficient, θ (area energy^{-1}), and the activity level, A (energy time^{-1}). The assimilation rate changes non-linearly as a function of activity level and food availability (Fig. S1).

We assume, as did Houston (2010) and Einum (2014), that an organism's activity level is positively related to its BMR, following the performance model (Careau et al. 2008); we express activity level as a power function of BMR. This model conceptualizes BMR as the "idling cost" of a metabolic "engine:" a larger engine has higher idling costs but can also generate higher sustained energy output necessary to accommodate greater performance (Biro and Stamps 2010). Although there are other models concerning how BMR may relate to activity, such as the compensation model and the substitution model (Careau and Garland 2012), the performance model has received the most empirical support. For example, in a review of 31 studies, Careau and Garland (2012) found that about half reported a positive relationship between BMR and activity. Both Houston (2010) and Einum (2014) assumed that activity is linearly proportional to BMR. We relaxed this assumption in some versions of our model, allowing for non-linear (but positive) activity-BMR relationships using a power function (see below), although in most cases we retained the linear relationship.

In our model, "defense" is a broad category comprising both anti-predator (e.g., vigilance, camouflage, armor, use of refuge), immune defenses, and traits that may buffer against poor environmental conditions (e.g., insulation, water-saving mechanisms). These defenses consume energy, but they also reduce the organism's extrinsic mortality rate by mitigating the impacts of environmental risk factors, thereby increasing fitness. We assume that defense positively relates to BMR, using a power-function formulation as for activity. Mounting a defense requires some form of underlying biological machinery, which, even when not in use, incurs a maintenance cost contributing to BMR. We assume that the higher the maintenance cost, the more vigorous and effective the resulting defense. Support for this assumption

comes from studies demonstrating energetic costs associated with predator defense strategies (Villagra et al. 2002; Møller 2009; Harrison and Preisser 2016) and immune response (Ots et al. 2001; Derting and Compton 2003; Martin et al. 2003; Martin et al. 2017).

Allocation of energy to activity and defense increases with net energy intake, N, with diminishing returns, scaled by a power function of basal metabolic rate, B:

$$A = \alpha a_1 B^{b_1}\left(1-e^{-\beta_1 N}\right) \tag{4}$$

and

$$D = (1-\alpha) a_2 B^{b_2}\left(1-e^{-\beta_2 N}\right) \tag{5}$$

where a_1 and a_2 are the coefficients, and b_1 and b_2 are the exponents that determine the strength of the relationship between activity or defense, respectively, and BMR (Fig. S2) and capture the mathematical relationship of the performance model. The exponent β sets the rate at which the functions converge on their upper bounds as functions of net energy intake rate, N, and α is the proportion of the energy that flows to activity, with the proportion $1-\alpha$ going to defense. Thus, the magnitude of α expresses the balance of an energetic trade-off between activity and defense. Einum's (2014) proportional relationship between RMR and activity metabolism, q, is equal to $1/a_1$ and is a special case of Eq. 4, with $b_1 = 1$ and $\beta_1 N$ very large.

To account for the wide variety of mechanisms under the umbrella category of defense, we recognized two sub-categories: activity-related defense and activity-independent defense (Fig. 1). An animal's environment contains both: (a) exposure to mortality risk that scales with activity level (activity-related risk) and (b) mortality risk that is independent of activity level (activity-independent risk). For example, predation risk generally increases with activity, because greater movement speed increases encounter rates with and detectability by predators (e.g., Werner and Anholt 1993), and a higher density of predators would presumably mean a higher exposure to mortality risk due to activity. However, some sources of mortality (parasites obtained at roosts, predation while at rest, weather) may not arise from being active. We assume that organisms are capable of developing and directing defenses towards both activity-related and activity-independent risks, yet the specific adaptations may be suitable for only one type of risk. For example, vigilance against predators may be an activity-related defense mechanism (either because it is increased during active periods or because activity interferes with it), while a cold-tolerance mechanism such as thick fur or an enhanced immune system when pathogens are prevalent in the environment would be unrelated to activity. The trade-off between activity-related and activity-independent defenses is captured in the mortality rate equation. Here, mortality rate, μ, is comprised of three components (cf. Werner and Anholt 1993):

$$\mu = \mu_0 + a_3 A^{b_3} e^{-p_A (D/D_0)} + R e^{-(1-p_A)(D/D_0)} \tag{6}$$

The first term μ_0 represents background mortality independent of activity or deterrable environmental risks; we did not vary this term in the analyses presented here. The second term contains a coefficient a_3 that translates activity and activity-related defense, $p_A D$, into a component of mortality rate. This parameter defines how activity exposes the organism to mortality risks in the environment, shaped by factors like the density of predators or the availability of cover that alter mortality risk as the organism becomes more active. We therefore treat a_3 as a key environmental variable in our analysis. Equation 6 also contains an exponent b_3 that produces non-linear effects of activity on mortality risk. In the default, this exponent is set to a value of 1, making realized activity-related risk proportional to activity. The third term in Eq. 6 contains a parameter, R, indicating the magnitude of environmental mortality risk that the organism experiences independent of activity. The parameter R is another key environmental attribute that we vary in different versions of the model. The p_A contained in the exponential portion of both second and third terms denotes the proportion of energy allocated to ameliorating activity-related mortality risk and so represents an additional allocation trade-off. $p_A D$ is the level of activity-related defense. The term $1-p_A$ is the proportion of defense allocated to responding to activity-independent mortality risk. Hence, $(1-p_A)D$ is the level of activity-independent defense.

To examine how BMR is optimized in any given environment, we assess fitness as a function of all trait values. We do this by first calculating survival, L:

$$L = e^{-\mu t_g}, \tag{7}$$

where t_g represents the organism's generation time, which we hold constant for all investigations. We then express fitness, w, as the multiplicative product of productivity and survival:

$$w = GL. \tag{8}$$

Therefore, fitness is proportional to productivity, G ($=N-A-D$ in Eq. 2) (see also Einum 2014).

The standard or default parameter values we used for most runs of the model are listed in Table 1. We note here that Eq. 8 captures an emergent trade-off between survival and productivity.

Optimal basal metabolic rate

We defined the optimal magnitude of a variable as the value that maximized fitness. Using our default parameter set (Table 1), we conducted three nested, numerical hill-climbs

Table 1 Default parameter values and descriptions

Parameter	Description (units, if any)	Default value
F	Food availability (food area^{-1})	5
ε	Assimilation coefficient (energy food^{-1})	10
γ	Food handling time (time food^{-1})	1
θ	Attack coefficient (area energy^{-1})	1
a_1	Coefficient for the BMR-activity power function	1
b_1	Exponent for the BMR-activity power function	1
β_1	Activity energetic exponent (time energy^{-1})	0.5
β_2	Defense energetic exponent (time energy^{-1})	0.5
D_0	Standard defense level (energy time^{-1})	0.5
a_2	Coefficient for the BMR-defense power function	1
b_2	Exponent for the BMR-defense power function	1
a_3	Exposure to environmental risk due to activity (time^{-1})	1
b_3	Activity-related risk exponent	1
R	Activity-independent environmental risk (time^{-1})	3
t_g	Generation time (time)	1
μ_0	Background mortality (time^{-1})	0.2

in MATLAB to find the three-way optimum of basal metabolic rate, B, allocation to activity versus defense, α, and allocation to activity-related versus activity-independent defense, p_A. These values represent an organism's best strategy with regard to allocating energy among five main characters (BMR, activity, activity-related defense, activity-independent defense, and productivity). Our model applies to any level of phenotypic variance (within individuals, among individuals, or among populations or species). Because it is an optimality model, the level at which it could act depends on the level at which there is environmental variation. Thus, the outcome of the model could represent an optimal genotype, in which the individual is fixed for these values (because the population only experiences one combination of environments). Scaling up to among-species variation follows the same logic. Alternatively, the model could be interpreted as producing points on an optimal reaction norm, in which the organism can adjust to different environments because all individuals in a population are experiencing an array of environmental conditions. Regardless, we found this optimization for ten levels of food availability, F, and of each of the two types of risk, a_3 and R, to determine how these characteristics of an organism's environment influence its optimal BMR. In each case, we extracted fitness to show how it varies across a range of BMR values. The MATLAB program used to generate these and the other results presented here is available on request from the authors.

Ecological effects on syndrome structure

We assessed the consequences of the modeled relationships for the set of phenotypic traits commonly linked with the POLS (BMR, activity, activity-related defense, activity-independent defense, and productivity). To do this, we ran the model to generate the optimal allocations of energy across variation in each of three environmental conditions, the coefficient of exposure to mortality risk due to activity, a_3, mortality risk that is independent of activity, R, and food availability, F. A syndrome would be indicated by strong correlations among the five traits that persist across differences in the environment, and here we examine variation in environment occurring along three axes. To do this, we estimated correlations by generating variation in one environmental axis (e.g., food availability) and then calculated the correlations for all ten pairs of the five optimal trait values that were generated across that environment. We then assessed how those correlations changed along a second environmental axis (while holding the third environmental parameter at the default value) for all six combinations of the three environments. In order to produce non-zero trait values that would skew correlations in non-relevant ways, we examined the parameter space of $F > 1.02$ (the minimum value that produced non-zero traits, Fig. 2) and a_3 and $R \geq 1$ (the minimum values that produced non-zero traits, Fig. 3a, b). To assess how these correlations changed across environmental space, we had to use the same minimum F value (1.02) to avoid trait values of zero, but could use a minimum risk of zero for both types of risk (a_3 and R). These slices of three-dimensional environmental space provide a view of the optimal trait combinations that should be expressed by individuals in a population experiencing variation in all three axes, or species that are distributed across variation in all three axes. Because the model generates the suite of optimal trait values, the results can be relevant to variation occurring within individuals, among individuals within a population, or among populations or higher taxa, depending on the scale of environmental variation.

Constraints on BMR, pleiotropic effects, and best-of-a-bad job

One application of the POLS hypothesis has been to assess variation within a population (Niemelä et al. 2013; Løvlie et al. 2014; Rádai et al. 2017). Because all traits are mechanistically linked to BMR, we wondered how within-population variation in BMR might then influence these traits and the correlations between them. We used our model to impose variation in BMR in a single environmental circumstance (defined by the defaults in Table 1); that is, BMR was constrained to one of a range

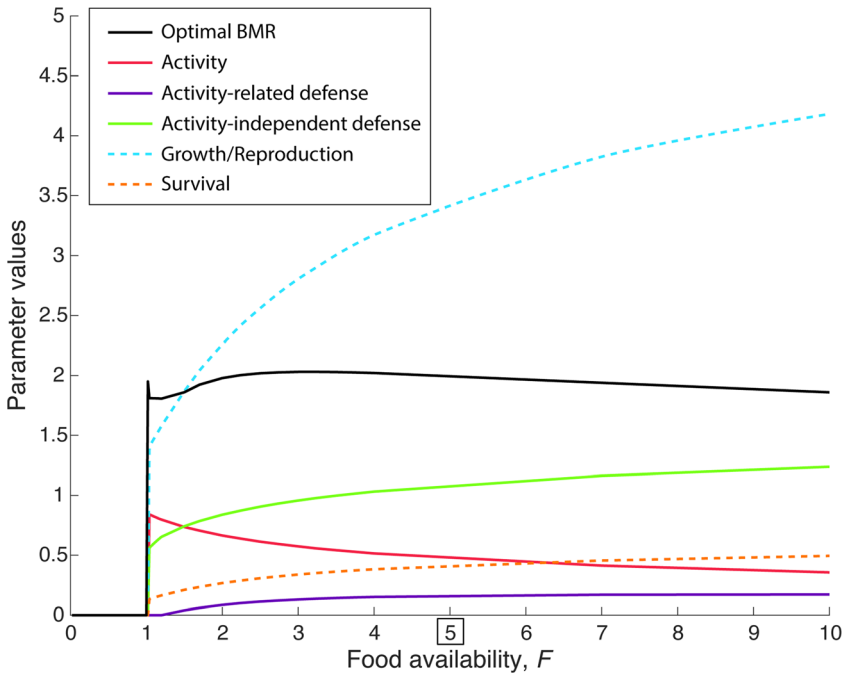

Fig. 2 Plot of optimal trait values (solid lines) for BMR (*B*, black), activity (*A*, red), activity-related defense ($p_A D$, purple), and activity-independent defense ($(1 - p_A)D$, green) and the two fitness components (dashed lines), growth/reproduction (*G*, light blue) and survival (*L*, orange) with respect to food availability (*F*). The model was run under default conditions (Table 1) for all other parameters except for food availability. The box around $F = 5$ highlights the default condition

of values, which included the optimal BMR. Others have assessed reasons for this variation (McNab 2009; Konarzewski and Książek 2013; White and Kearney 2013; McNab 2015); here we focus on the consequences if it is present. For each constrained BMR, we assessed the relative fitness of four versions of the model. The first case simulated the most extreme circumstance where all allocation decisions were fixed at optima associated with the optimal BMR and did not vary across the range of selected BMR values (e.g., they were non-optimal with respect to BMR except at optimal BMR). This would, for example, simulate the case of genetic variation that had major pleiotropic effects on the whole system. In the second case, we constrained only the allocation between the two types of defense (p_A) and allowed the allocation between activity and overall defense (a) to optimize (partial pleiotropy). In the third case, we allowed the two types of defense to optimize while constraining the allocation between activity and defense. The final case was the full model, in which we allowed both trade-offs to optimize, but conditional on the chosen value of BMR. We assessed the fitness achieved across an array of predetermined BMR values.

To explore the consequences of such constraints on POLS, we used the model that produced the highest fitness and calculated the other trait values. We assessed these across an array of BMR values, simulating a population of individuals with differing BMR and potentially other traits as well.

Data availability There are no data associated with this publication to be archived.

Results

Optimal basal metabolic rate

Across all food levels, an intermediate BMR was optimal, and this optimal BMR was maximal at an intermediate food level (Fig. 2) regardless of the parameters of the power function relating BMR to activity. This is a similar result to Einum (2014), but over a broader range of conditions. Our results also suggest that BMR is highly sensitive to food availability. The other trait values were also sensitive to food availability, though they increased or decreased steadily across the viable range of food (Fig. 2). We found that when food availability was below 1, the model produced trait values of zero and therefore we considered only $F > 1.02$ as viable parameter space.

BMR also varied with respect to variation in both environmental influences on mortality risk (Fig. 3a, b). When food availability was held constant at the default, intermediate level ($F = 5$), increases in mortality risk in the environment (regardless of type) increased the optimal BMR with diminishing slope (Fig. 3a, b). Activity decreased with both types of risk (Fig. 3a, b). As expected, activity-independent defense decreased with a_3 (Fig. 3a), but increased with R (Fig. 3b), whereas activity-related defense did the opposite. Increasing either type of mortality risk decreased productivity.

Food and both mortality risk parameters interacted to influence optimal BMR (Fig. 4a, b). BMR continued to show an intermediate optimum with food availability across most levels of both types of mortality risk (but see below), and BMR increased with both parameters of mortality risk

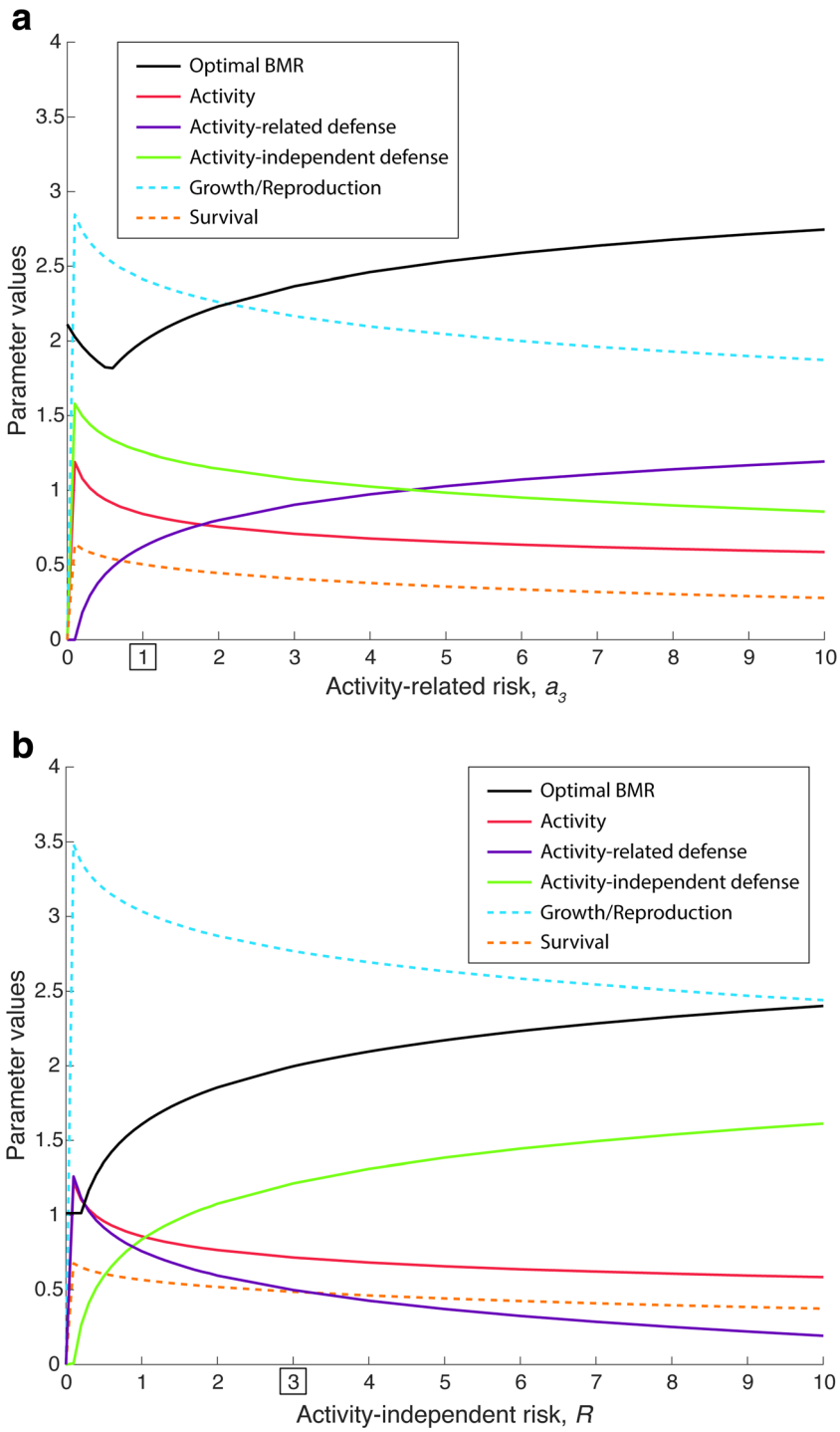

Fig. 3 Plot of optimal trait values (solid lines) for BMR (B, black), activity (A, red), activity-related defense (p_AD, purple), and activity-independent defense ($(1 - p_A)D$, green) and the two fitness components (dashed lines), growth or reproduction (G, light blue) and survival (L, orange) as a function of the magnitude of **a)** the coefficient of activity-related risk (a_3) and **b)** activity-independent risk (R). The model used the default values of all other parameters (Table 1). The boxes around $a_3 = 1$ and $R = 3$ highlight the default conditions

regardless of food availability. However, the two mortality risk parameters modulated the impact of food availability on BMR in different ways. In the case of mortality risk incurred when active (Fig. 4a), the model produced higher BMR as risk increased, but the pattern of optimal BMR over different food availabilities changed dramatically from the case of no activity-related risk to some risk (dashed compared with solid lines in Fig. 4a). The curves at higher mortality risk also have slightly different shapes, with the peak in BMR occurring at higher food availability as activity-related risk increases, implying that the slope of the curve of BMR over food changes with increasing risk.

A more complex response was produced when both food availability and activity-independent risk were varied but activity-related risk was held constant (Fig. 4b). At lower levels of activity-independent risk, the optimal BMR across variation in food availability exhibited a pattern similar to that seen in Figs. 2 and 4a, with an intermediate maximum optimal BMR.

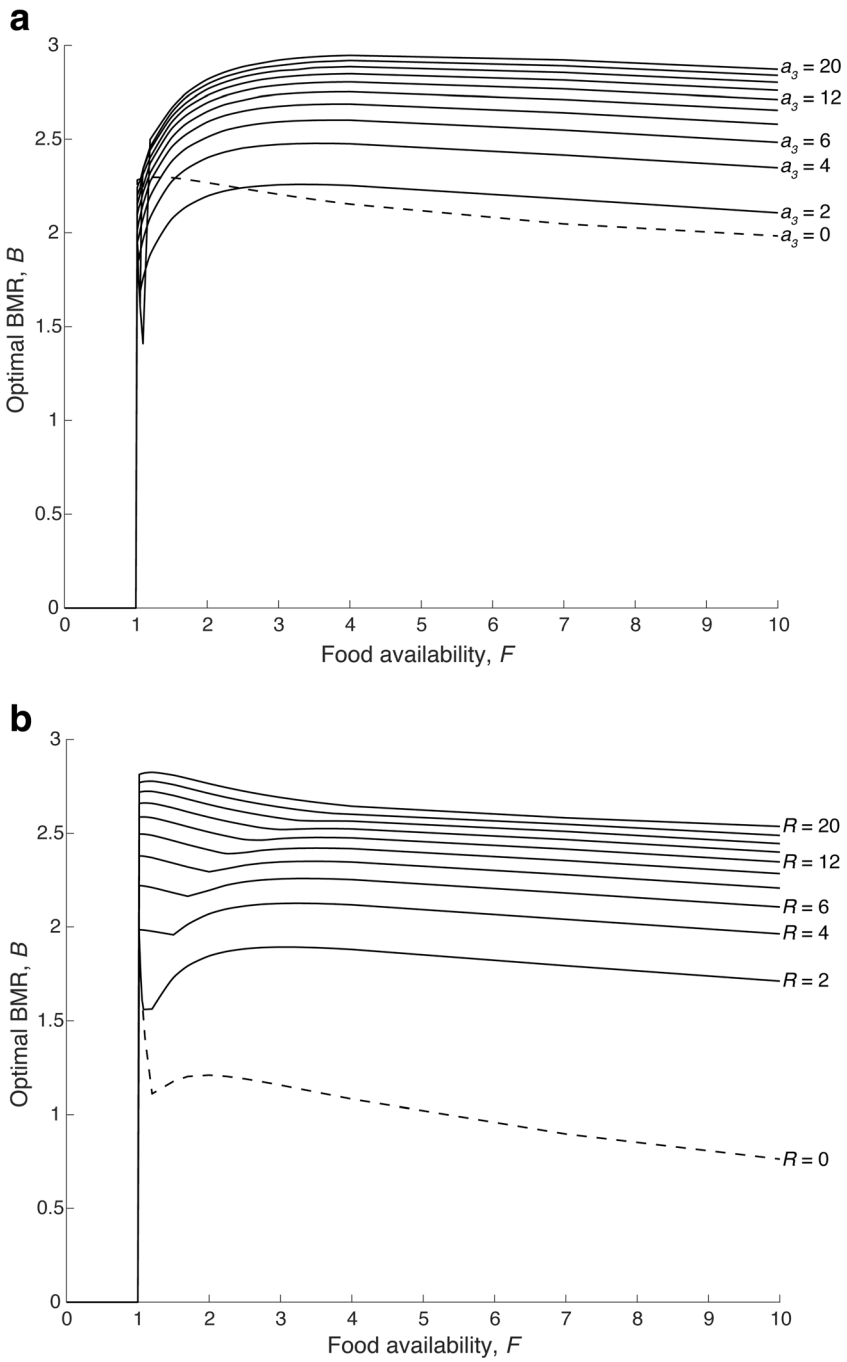

Fig. 4 Optimal BMR curves across levels of food availability (F) for differing levels of **a)** activity-related risk (a_3) and **b)** activity-independent risk (R). Note the slight shifts in slope of the lines indicate an interaction between food and activity-related risk on BMR in **a**. The interaction between food and activity-independent risk is much stronger as indicated in **b** with a shift towards the highest optimal BMR occuring at low food and dramatic shifts in the shape of the curves as risk increases

However, at higher levels of activity-independent risk, the maximum optimal BMR occurs at very low food availability, nearly at the level that first produces positive values for BMR.

Ecological effects on syndrome structure

Our model produced a sloppy syndrome, with the magnitudes and directions of correlations strongly influenced by the environment. The correlations between trait values of BMR, activity, the two types of defense, and productivity were strong when measured across some environments, but not others (Fig. 5, Table S1, Figs S3A, B). For example, we obtained strong positive correlations between optimal BMR and activity (B, A), optimal BMR and activity-related defense (B, p_AD), activity and activity-related defense (A, p_AD), and productivity and activity-independent defense (G, $(1-p_A)\,D$) when the correlations were measured across the level of exposure to activity-related mortality risk, a_3 (Table S1). Trait correlations were also strong and generally consistent when measured across levels of activity-independent mortality risk, R, but several, including between BMR, activity, or productivity and both types of

defense were of opposite sign than occurred across a_3 (Table S1). Interestingly, correlations across either type of risk were strongly positive or negative at most levels of food availability, but when food dropped to low levels, these correlations collapsed to near 0 (Table S1). This is because when F becomes low, the lack of energy produces rapid changes in some traits, which can quickly change the correlations between traits.

The dynamics of these correlations emerged most clearly when they were measured across variation in food and plotted against either type of risk (Fig. 5a, b). Most of the correlations were closer to 0, and both types of mortality risk modulated them, in some cases quite substantially. The only correlation in these conditions that showed stability was that between activity and activity-independent defense, which was always strongly negative. We note that the correlation between BMR and activity was highly variable. When assessed across activity-related risk (a_3), the correlation started positive and steadily decreased, eventually becoming negative at high levels of mortality risk (Fig. 5a).

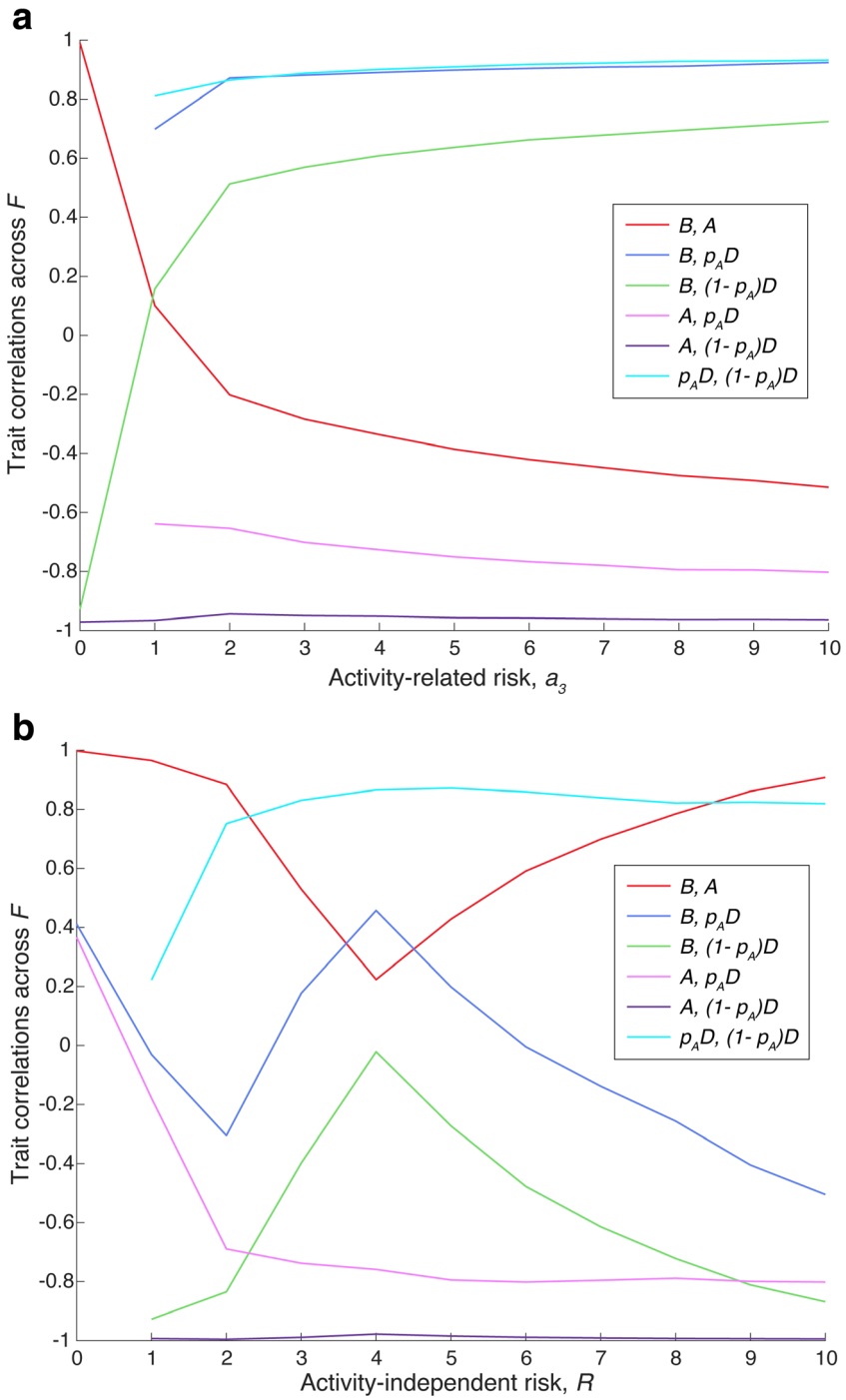

Fig. 5 Plots of six bivariate trait correlation coefficients for four traits (BMR (B), activity (A), activity-related defense (p_AD), and activity-independent defense ($1 - pA)D$), calculated across the optimized values obtained at points along the range of food (F) levels. These correlation values were then obtained and are plotted here for different levels of **a)** activity-related risk (a_3) and **b)** activity-independent risk (R). For each graph, there are three curves that start at a risk level of 1 because the corresponding defense trait has a zero value while the risk is also zero, thus producing inappropriate correlation coefficients

Across activity-independent mortality risk (R), the correlation was much higher, declined at low levels of mortality risk, but then began increasing again at higher levels of R (Fig. 5b). Some of the other trait correlations also either decreased or increased with R only to switch direction in the middle of the range. Finally, the correlation between BMR and productivity was weak and changed across both types of mortality risk (Figs S3A, B).

Constraints on BMR, pleiotropic effects, and best-of-a-bad job

We asked what happens to fitness in the model if we constrained BMR to be non-optimal and allow both, one, or none of the other allocation decisions to be optimized. When the results for the four combinations of optimizing or not optimizing each of the two trade-offs across the range of BMRs are superimposed, the contributions of each trade-off to mitigating non-optimal BMR are clear (Fig. 6a). Constraining BMR to be non-optimal reduces fitness. However, compared to the case when both trade-offs are fixed (maximum pleiotropy, black line in Fig. 6a), allowing both trade-offs to optimize given the fixed BMR substantially increases fitness (no pleiotropy, blue line in Fig. 6a). Thus, optimizing the allocation trade-offs can compensate in part for a non-optimal BMR (best of a bad job, Dawkins 1976). Intriguingly, the two trade-offs do not contribute equally, at least under the default conditions we used. Allowing the allocation between the two types of defense to optimize (and fixing the allocation between activity and defense, red line in Fig. 6a) is nearly as good as optimizing both trade-offs, at least over a range of non-optimal BMRs that is close to the optimum. Fixing the allocation between types of defense and allowing the allocation between activity and defense to optimize (green line in Fig. 6a) are little better than the extreme case where nothing is optimized.

We also examined how the other traits adjusted to non-optimal BMR using the version of the model with no pleiotropy (Fig. 6b). The pattern of adjustment produces a correlation structure that only partially resembles the POLS. Three of the traits do not change consistently in one direction over the range of BMRs that we analyzed (Fig. 6b). Productivity, G, initially increases but then decreases. Activity-related defense is minimized at 0 for low values of BMR and then rises only to tail off at larger values of BMR. Similarly, activity-independent defense also increases, but then decreases, at high values of BMR. Only activity increases as BMR increases, although it does so non-linearly. If this variation occurred within a population, it would cause weaker correlations than might be expected and create some correlations that are not predicted by the POLS (e.g., a negative correlation between productivity and all other traits including BMR).

Discussion

The POLS describes the case when multiple attributes of organisms co-vary (Réale et al. 2010), typically with metabolic rate and reproductive rate as core attributes (e.g., Careau et al. 2008). Both Houston (2010) and Einum (2014) modeled optimal metabolic rate and explored how it would evolve under certain conditions. We modified and extended their approach to assess the hypothesis that metabolism exerts a central influence on energy-dependent characters hypothesized to be part of the POLS (i.e., BMR, activity, defenses, and growth/reproduction), and hence might drive correlation structures among POLS traits. We incorporated a non-energetic cost of activity (exposure to mortality risk of, for example, predation) favoring activity-related defenses, and a cost of activity-independent mortality risk that selected for other kinds of defense (e.g., insulation or immunity). The model thus contained a set of allocation decisions regarding the energy acquired through metabolism. Similarly to Houston (2010) and Einum (2014), we modeled selection acting as if there were no life stage differences in mortality and/or productivity. Under some conditions, our model produces correlations characteristic of the POLS. However, we examined model solutions over a complex environment in which food availability and the level of each type of mortality risk could vary. These extensions reveal that metabolic rate evolves in some initially non-intuitive ways in response to complex environments, and there is considerably more complexity to the associations among some POLS attributes than expected.

Our model confirms several previous results. Although we incorporated a different equation for linking BMR with the ability to achieve high levels of activity or defense (the increased intake model, sensu Nilsson 2002; Careau and Garland 2012, or the performance model, sensu Careau et al. 2008), we found that the trade-off between the benefits of BMR for increased trait expression and the energetic costs produced an intermediate optimum for BMR. The magnitude of the linkage between BMR and performance (the parameters a_1 and b_1 for activity, and a_2 and b_2 for defense) has the expected effects on the magnitude of optimal BMR—with less linkage, selection then favors a lower BMR.

Empirically, the performance model has received widespread support (Daan et al. 1990; Careau et al. 2008), but exceptions abound (e.g., Ricklefs et al. 1996; Mathot and Dingemanse 2015). Our results draw attention to two aspects of these empirical results. First, correlations between particular traits such as organ size and BMR may be misleading, because we might expect variation in the link parameters (e.g., a_1 or b_1) to exist among different components of the underlying machinery driving elevated metabolic rates. For example, larger digestive organs (e.g., Daan et al. 1990; Selman et al. 2001; Song and Wang 2006; Konarzewski and Książek 2013) are not the only support mechanisms for

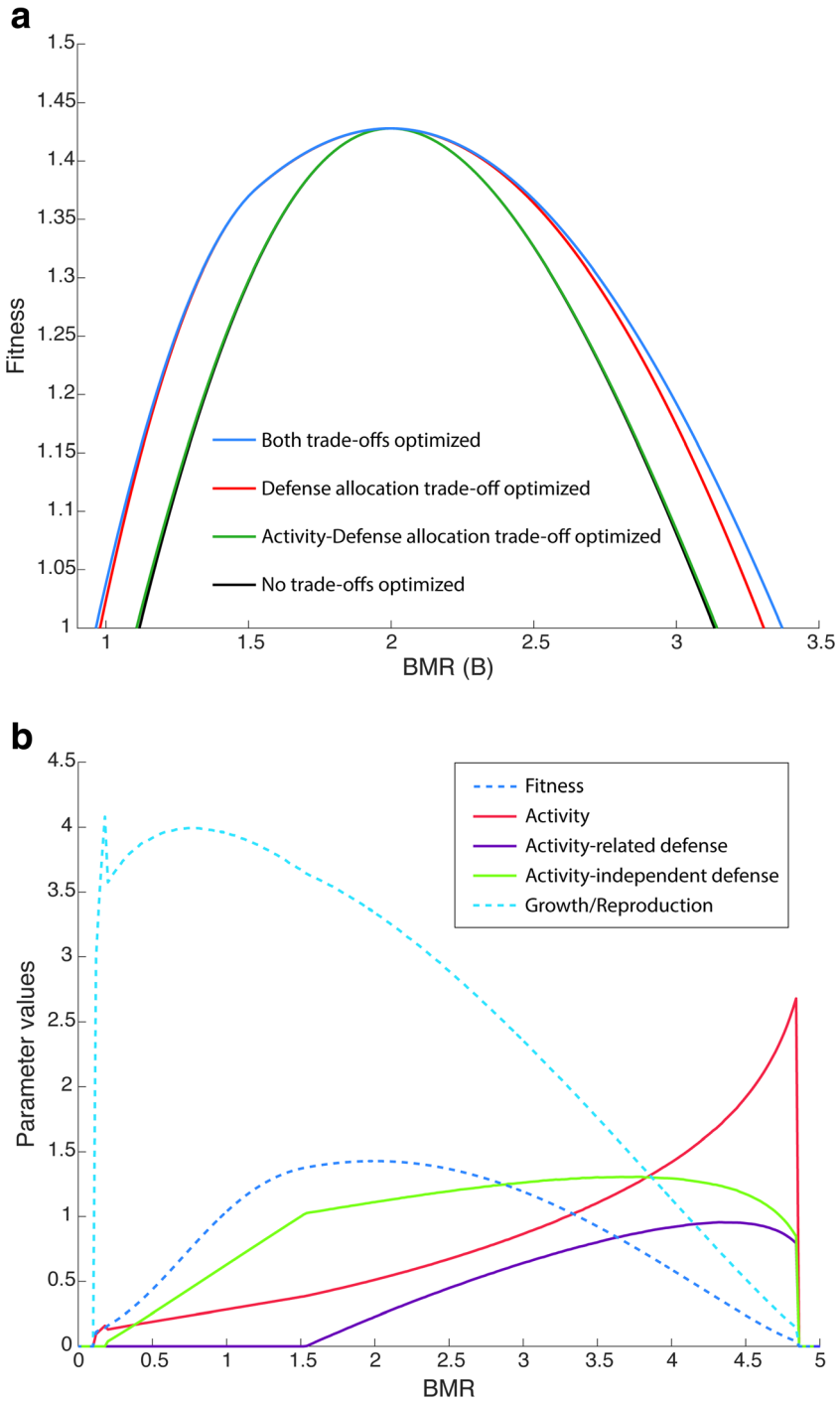

Fig. 6 Analysis of four constrained versions of the full model. **a)** Fitness achieved when BMR (B) was constrained to the value on the x-axis. The black line indicates fitness when we fixed the trade-offs between activity and defense and between both types of defense at the values for the optimal BMR (e.g., at $B \approx 2.1$), i.e., no trade-offs were optimized. The green line tracks fitness when we constrained only the trade-off between types of defense to the optimal value when B was optimized but we allowed the activity-defense trade-off to optimize. The red line indicates fitness when we allowed the trade-off between types of defense to optimize but fixed the activity-defense trade-off. Finally, the blue line indicates fitness when we allowed both trade-offs to optimize. All models were run under the set of default conditions (Table 1). **b)** Optimal trait values obtained from the model constrained only by the value of B (e.g., blue line in Fig. 6a) over the range of B. This simulates genetic variation in B among individuals in a population with a single optimal B. Trait correlations with B would switch sign passing from left to right of the optimal B, and correlations with G would also switch signs across some of the range of B, producing a sloppy POLS within a population

acquiring new energy. Foraging requires muscles for movement and sensory systems for detecting prey. Each of these components could have different link parameters. Our model focuses on the summed linkage between the energy cost of the underlying support mechanisms and maximal metabolism rather than any particular component. Second, our model combines the performance and allocation models (Careau et al. 2008; Mathot and Dingemanse 2015), and all of our results depend on this combined view of how energy is acquired and allocated. Some form of this combination is likely to be relevant to real organisms. If so, understanding isolated components of this system, especially across certain types of environmental variation, may be quite misleading.

Optimal basal metabolic rate

Our model also confirms Einum's (2014) finding that food availability affects optimal BMR non-monotonically, such that optimal BMR is at a maximum at intermediate food levels in most conditions. Einum (2014) suggested that this intermediate

is a consequence of activity having the benefit of increasing food intake rate while also increasing energy expenditure, so that when food becomes sufficiently abundant, acquiring energy is easy and both activity and BMR decline. Our model produces this same result, apparently for the same reasons, over most (but not all) values of the other two environments (the two levels of mortality risk exposure). A final point about the intermediate maximum with food availability is that it appears to drive substantial complexity in the correlational structure of POLS attributes. We discuss both the interaction effects and the impact on correlations in more detail below.

Our expansions of previous models to include allocation to defense revealed several novel results. First, our incorporation of two types of mortality, activity-related and activity-independent, produces some unexpected effects. Optimal BMR increases with exposure to either type of mortality, largely because attributes that reduce mortality (defenses) cost energy, require higher levels of active metabolism, and hence higher BMR in support. However, optimal BMR increased over most of the range of mortality risks we modeled (Figs. 3 and 4), in contrast to the pattern obtained over increasing food availability. The type of mortality risk affected the rate of increase but not the fact that BMR always increased. While Houston (2010) included a mortality risk of activity, this influenced only how much time an organism spent being active, as he did not explore allocation of energy more generally to defense. Allocation of energy to defenses against various types of risks has received some attention (e.g., Møller 2009; Versteegh et al. 2012; Mathot et al. 2015) and clearly could have an important impact on the suite of attributes involved in POLS. In environments with higher mortality risk, an individual will benefit from greater allocation of energy to defense; defense requires structures or systems (e.g., immunity) that must be maintained even when defense is not employed, and so this necessitates higher BMR. Our model thus predicts that individuals or species vulnerable to more sources of mortality will likely have higher BMR independent of the life history impact of mortality if they also have a means to defend against such mortality risks.

Empirical support for this prediction is equivocal. For example, at the among-species level, Møller (2009) found that bird species with higher BMR exhibited greater wariness towards predators, as measured by longer flight initiation distances. He concluded that increased BMR was necessary for sufficient alertness to potential predator attacks and facilitates the flight response. However, in two different among-species comparative studies, BMR was negatively associated with immune measures in 63 species of European birds (Pap et al. 2015) and in 12 species of tropical birds (Tieleman et al. 2005). Assuming other factors were not varying across these organisms, these results fit the context-dependent outcome of our model for correlations between BMR and defenses (Table S1).

Investigations of the within-species (among-individual) level have yielded similarly ambiguous trends. Mathot et al. (2015) found that, for free-living great tits (*Parus major*), the relationship between BMR and risk-taking behavior was context dependent. They define risk-taking behaviors as those that "increase resource acquisition at the expense of an increased risk of mortality." Therefore, risk-taking behaviors can be viewed as the opposite of defenses that, in our context, would consume resources (energy) and, by definition, decrease mortality. Under one scenario, a disturbance created by a human presence, there was a negative relationship between BMR and a measure of risk-taking behavior, which is essentially in line with our assumption of the relationship between BMR and defense. However, when presented with a predator model, the relationship reversed. A similar mixed result was found for western stuttertrilling crickets (*Gryllus integer*), for which two behaviors associated with risk-taking (boldness and exploration) showed opposite correlations with RMR (positive and negative, respectively; Krams et al. 2017). Versteegh et al. (2012) found that stonechats (*Saxicola torquata*) exhibited a significant, negative relationship between BMR and some immune measures, but not others. Of the immune measures that did not significantly correlate with BMR, trends in both the positive and negative direction were observed.

The correlational relationship between BMR and various defenses appears highly context dependent, which our model produces especially if food levels vary (Fig. 5a, b). These results depend in part on defenses requiring energy and exerting a cost of maintenance, captured mathematically in the model. We reaffirm the logical basis of this. To effectively mount a defense, anatomical and/or physiological systems are necessary, and larger systems that support higher levels of defense should necessitate a larger maintenance cost associated with higher BMR. The empirical evidence discussed here validates our findings that the direction and strength of correlations between BMR and different types of defense will depend on the specific ecological context in which the association is investigated (Table S1).

We also found that food availability and mortality risk interacted to affect optimal BMR. Empirically, food and mortality risk have interactive effects on growth rates (Arendt and Reznick 2005; Gale et al. 2013), metamorphosis (Richter-Boix et al. 2007), reproduction (Karels et al. 2000; Zanette et al. 2003), and behavior (Anholt and Werner 1995; Vehanen 2003; Krause and Liesenjohann 2012), and so a similar impact on metabolic rate would be expected. Intriguingly, we found that the interaction between activity-independent mortality risk and food was more complex than that between activity-related mortality risk and food. This could arise because activity-independent mortality risk does not invoke as many feedbacks as activity-related mortality risk. Activity is necessary to acquire food, yet if it exposes

the organism to danger, further increases in activity may have minimal net benefits, so when food is too low to mount other defenses, activity and hence BMR are reduced (Fig. 4a). Activity-independent mortality risks, on the other hand, always favor increasing activity so that energy for defense can be obtained. This is evident in Fig. 4b; when food is low and mortality risk is high, defense is strongly favored, driving up the optimal BMR.

Ecological effects on syndrome structure

The interactive effects of extrinsic mortality risk and food availability on optimal BMR and the interaction between activity and food availability in determining total energy budget both appear to complicate the idea that metabolism, as a core attribute, might produce trait correlations (Careau et al. 2008; Biro and Stamps 2010; Réale et al. 2010). We measured trait correlations in all possible environments. Across some slices of the environmental space defined by any two of the environmental parameters in our model, several key attributes are strongly correlated and the correlations are relatively stable (Table S1). In these combinations of environment, increasing BMR correlated strongly with all other traits (activity, activity-related defense, and activity-independent defense). While some of these correlations match predictions of the POLS (Réale et al. 2010), others do not. Furthermore, across some other combinations of environments, the correlations became weaker and fluctuated with other environmental factors. For example, when food availability varies, the correlation between BMR and activity was weak and modulated by both types of risk (Fig. 5), possibly because optimal BMR has an intermediate maximum across food availability (Fig. 2) such that activity level changes differently across food than does BMR. Intriguingly, both types of extrinsic mortality risk initially reduce the correlations between BMR and activity measured across F, perhaps due to the increased value of defense relative to activity as both mortality risk and food increase. Again, the fact that food and activity-independent mortality risk interact to affect optimal BMR (Fig. 4b) may influence correlations across food as activity-independent mortality risk is also changing. The correlation between BMR and activity across F begins to increase again at high levels of R, perhaps because across increases in food, BMR (Fig. 4b) and, in turn, activity decrease as the increase in available energy is shunted towards activity-independent defense.

The allocation trade-offs within the model also have dramatic effects on trait correlations. Consider the correlations between activity-independent defense (e.g., immunity) and both metabolism and activity. Many discussions of the POLS suggest that organisms with a faster life history (high metabolism, high activity) should have lower allocation to immunity, and indeed, some studies show the expected negative correlation (e.g., Pap et al. 2015). Our model indicates that under some conditions, the correlations with activity-independent defense indeed are strongly negative (Table S1). However, these correlations shift to being strongly positive when activity-independent mortality risk varies. Moreover, the correlation between metabolism and activity-independent defense is quite weak across different levels of food availability (Fig. 5b). Again, food availability has a hump-shaped effect on the optimal BMR, whereas the effect of food on activity-independent defense is consistently positive. Thus, values for BMR and for activity-independent defense change positively with each other over low food values and then shift to a negative correlation once food becomes more abundant. Low food also has big and unexpected effects on trait correlations across most of the three-dimensional environmental space we considered. This suggests that organisms living in harsh environments (i.e., when energy assimilation is difficult) may show very different trait correlations than organisms living in more benign environments. Our model thus suggests that the correlation structure among POLS attributes is likely to be quite sensitive to which aspects of the environment are varying. The variation in relationships that has emerged in the empirical literature (e.g., Tieleman et al. 2005; Careau et al. 2011; Niemelä et al. 2012, 2013; Versteegh et al. 2012; White et al. 2016) might be one predicted outcome from our model. Whether or not this variation is due to the specific factors we explored in the model needs to be tested.

Constraints on BMR, pleiotropic effects, and best-of-a-bad job

Because metabolism is so central to most other organismal processes, constraints on the evolution of metabolic rate have been proposed as important drivers of correlated traits (Brown et al. 2004; Careau et al. 2008). We explored how our model might behave when parts of it were constrained. Naturally, a BMR displaced from its optimum results in lower overall fitness, and for the parameter values we have used, fitness drops off quite rapidly. We compared models in which the other allocation decisions were allowed to evolve conditional on a suboptimal BMR or were also constrained to set values (in all cases we chose values that would have been optimal if BMR was optimal). Allowing the allocation trade-offs to optimize rescued some fitness, suggesting that constraints in one part of the system can be partially compensated for by other components. We note as well that while a constrained metabolic rate might limit the total energy flowing through the system, and so influence absolute trait values, if the mechanisms that drive these other allocation decisions are free to evolve, the relative trait values might not be constrained. Our model thus illustrates an important potential aspect of real organisms; those complex systems under selection may still be optimizing components even if portions of the system are non-optimal. This also produces a sloppy syndrome. Because trade-offs acting within individuals

may produce patterns that differ from processes acting among individuals, our model, because of its potential to capture effects at both levels, reminds us of the importance of measuring trait correlations at the appropriate level of variance (Dingemanse and Dochtermann 2013).

Some caveats

As with all models, we made some simplifying assumptions that are worth re-examining briefly. First, we assumed that any energy allocated to one task could not satisfy the need for energy for another task. That is, the trade-offs were absolute. This is probably extreme, as biological solutions to one problem could also ameliorate another problem. For example, dark, cryptic body coloration may reduce both activity-related mortality (e.g., predation) and activity-independent mortality risk (e.g., extreme cold) (Clusella Trullas et al. 2007). Traits that solve multiple problems might further complicate the correlation structure between traits that makes up the POLS.

A second caveat is that we used arbitrary values for the key parameters. Some of our results appear only in extreme parts of the range of values we chose to explore. It is not clear how common such conditions might be in the real world. While we are comfortable with the insights these parameter values provide, we suggest that specific application of the predictions of our model to real systems will require assessing parameter values and exploring empirically supported regions of the parameter space.

Finally, although the trade-offs explicitly embedded in this model produce a trade-off between productivity, G, and survival, L, we have not included any additional variation in life history, such as a terminal life stage or differential mortality or performance with age that would drive more complex selection. Given the lack of theory on the POLS (Mathot and Frankenhuis 2018, topical collection on Pace-of-life syndromes), it is not clear if we have omitted the main selective force verbally proposed for the POLS (Stearns 1989; Ricklefs and Wikelski 2002; Réale et al. 2010). Adding more complex patterns of mortality or reproductive success with age might be a next step for models like ours. However, as we have attempted to show here, attributes affecting the trade-off between present and future reproduction are also likely to be involved in trade-offs with each other. Our model illustrates that such nested trade-offs may drive the complexity of pace-of-life-like suites of correlated traits (see also Montiglio et al. 2018, topical collection on Pace-of-life syndromes, Royauté et al. 2018, topical collection on Pace-of-life syndromes).

Advice for empiricists

Our model makes several general predictions about some of the potential forces acting on trait correlations associated with the POLS. A key issue is the level (within-individual, among-individual, or among-population/among-species) at which environmental variation occurs. Our model does not specify a particular level, but its results could apply at any of them if mortality risks and food supply vary at that level. So an immediate goal for empiricists is to assess the level at which their target organism experiences environmental variation.

Second, trait correlations will be dramatically different if the range of food availabilities includes the intermediate maximal optimal BMR or not. We know very little about the scale of variation in BMR within a species, let alone within individuals. Thus, another major goal of empiricists would be to document both among-individual and within-individual variance in BMR with respect to environmental conditions, especially food abundance.

Finally, more information about types of mortality risk and the influence of activity on realized risk is likely to be important in parsing out why trait correlations are strong or weak in a given organism. We have lumped such risk into two categories and have assumed that traits that ameliorate one type of risk are useless for another. Empirical information documenting these assumptions and the scale of variation in risks of different types will be very useful for providing a more precise version of our model that can generate specific predictions.

In summary, our analysis of the role of metabolism in providing the energy to allocate among several fitness-enhancing tasks within a world that can vary along three environmental dimensions (e.g., food, predators, and pathogens) produces a much more nuanced understanding of both metabolic rate and the suite of potentially correlated characters referred to as the POLS. The combination of multiple trade-offs and a multivariate environment make for some complex patterns in trait combinations. We conclude that the POLS should not be viewed as an invariant construct arising from only one simplified type of selection. Instead, the combinations of traits that make up real organisms are almost certainly emerging from multiple factors interacting in diverse ways. Untangling these both empirically and theoretically remains a major challenge.

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Compliance with ethical standards

This research did not involve either humans or animals.

Conflict of interest The authors declare that they have no conflicts of interest.

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