How to get the most bang for your buck: the evolution and physiology of nutrition-dependent resource allocation strategies

Enoch Ng'oma[1], Anna M. Perinchery[1], and Elizabeth G. King[1]

[1]Division of Biological Sciences, University of Missouri, Columbia, MO 65211

Abstract

All organisms utilize resources to grow, survive, and reproduce. The supply of these resources varies widely across landscapes and time, imposing ultimate constraints on the maximal trait values for allocation-related traits. In this review, we address three key questions fundamental to our understanding of the evolution of allocation strategies and their underlying mechanisms. First, we ask: how diverse are flexible resource allocation strategies among different organisms? We find there are many, varied, examples of flexible strategies that depend on nutrition. However, this diversity is often ignored in some of the best-known cases of resource allocation shifts, such as the commonly observed pattern of lifespan extension under nutrient limitation. A greater appreciation of the wide variety of flexible allocation strategies leads directly to our second major question: what conditions select for different plastic allocation strategies? Here, we highlight the need for additional models that explicitly consider the evolution of phenotypically plastic allocation strategies and empirical tests of the predictions of those models in natural populations. Finally, we consider the question: what are the underlying mechanisms determining resource allocation strategies? Although evolutionary biologists assume differential allocation of resources is a major factor limiting trait evolution, few proximate mechanisms are known that specifically support the model. We argue that an integrated framework can reconcile evolutionary

models with proximate mechanisms that appear at first glance to be in conflict with these models. Overall, we encourage future studies to 1) mimic ecological conditions in which those patterns evolve, and 2) take advantage of the 'omic' opportunities to produce multi-level data and analytical models that effectively integrate across physiological and evolutionary theory.

Keywords: resource availability, resource allocation, phenotypic plasticity, evolutionary theory, proximate mechanisms, ecological context

1. The central importance of the interplay between resource acquisition and allocation

The amount of resources available to organisms, whether the source is sunlight, plant matter, or prey animals, is inherently variable over the landscape and across time. This variability presents a fundamental challenge to all organisms, from the smallest microorganisms to the largest plants and animals, all of which must coordinate the acquisition of resources from the environment with allocation of those resources among the many competing functions and structures that contribute to the organisms' fitness. When faced with variation in available resources, individuals could respond in one of two ways: (1) maintaining the same relative proportion allocated to each trait or (2) exhibiting phenotypic plasticity in resource allocation by altering the relative amount of resources allocated to one trait versus others. When the optimal allocation strategy changes with resource availability, selection will favour the evolution of a phenotypically plastic allocation strategy.

The inescapable link between the amount of resources available to an organism and subsequent allocation of those resources means it is critical to consider how

allocation strategies change across a range of resource availabilities. There are many examples of flexible strategies that depend on availability. For example, an adaptive shift in resource allocation is thought to underlie the commonly observed pattern of lifespan extension under dietary restriction (reviewed in [1–5]. Likewise, sexually selected traits often show strong condition dependence (i.e. dependence on acquisition), also thought to result from an adaptive shift in allocation (reviewed in [6,7]. Even the current obesity epidemic in modern human populations is often hypothesized to result from a mismatch between a selective environment favouring increased storage under high resources and the modern environment of constant high resource availability [8] (see [9] for a recent review). To understand this wide diversity in allocation strategies in the natural world, we must understand how different ecological conditions select for different strategies and what mechanistic changes underlie these strategies.

Understanding how and why this coordination of resource allocation with availability evolves has implications for nearly all areas of biology. Energetic costs to biological structures and functions (i.e. allocation trade-offs) are assumed to be universal and a major factor limiting trait evolution [10,11]. Typically, less attention is focused on the role of variation in the acquisition of resources, though it is no less important in determining trait values, and can obscure the detection of functional trade-offs. In a seminal paper, van Noordwijk and de Jong introduced the Y model - a mathematical model linking resource acquisition and resource allocation [12], which has been a central concept in the field of life history evolution. In the Y model, two traits draw from a single resource pool, with trait values determined by the proportion of resources allocated to each (Figure 1 [12]). One of the key strengths of this model is its

70 simplicity and generality; it can be applied to diverse questions such as why and how
71 organisms age, what limits crop yields in different environments, why some species
72 produce hundreds of offspring while others produce very few, and what constrains the
73 evolution of fitness. While the Y model provides a conceptual starting point to
74 understand the evolution of acquisition and allocation, in the Y model the underlying
75 mechanisms governing these processes are treated as a black box. Likewise, our
76 empirical knowledge of the genetic and physiological mechanisms underlying these
77 processes is still limited, due in large part to their vast complexity [13,14]. The allocation
78 of resources is thought to influence nearly all the major structures and functions of an
79 organism, is affected by an array of interacting physiological pathways, is variable
80 across the lifetime of the organism, and interacts with many different environmental
81 factors. To achieve a complete understanding of how resource allocation trade-offs
82 govern these processes, we must explicitly consider its interaction with resource
83 acquisition and integrate across genomics, physiology, and evolution.
84 As we advance our ability to collect “omic” data at multiple levels (genomics,
85 transcriptomics, proteomics, metabolomics, etc.) and in multiple environments,
86 achieving this integration is becoming increasingly feasible. A major challenge now is
87 developing new analytical methods to address multi-level, multi-environment data, and
88 pulling out emergent themes that will help us better understand the complex processes
89 underlying trade-offs and linking these with evolutionary models. We argue that
90 resource allocation is a natural focal point in this effort. This relatively straightforward
91 concept has the potential to integrate knowledge across fields and address key
92 questions facing the intersection between evolutionary and molecular biology.

In this review, our goals are to: 1) detail the diversity of resource allocation strategies in response to environmental fluctuations in resource availability, 2) review the evolutionary explanations for these strategies and highlight where new models are needed, and 3) evaluate current approaches and suggest strategies for understanding the genetic and physiological mechanisms underlying resource allocation strategies.

2. The diversity of phenotypically plastic resource allocation strategies in the natural world

In the wild, organisms vary widely between species and populations in how they respond to variation in resource acquisition, with a diverse array of examples of phenotypically plastic resource allocation strategies (Table S1). Variation in resource acquisition can result from variation in resource abundance in the environment, and/or from differences among individuals in their ability to acquire resources. By far the largest challenge in describing broad patterns of phenotypic plasticity in allocation strategies is to directly quantify resource acquisition and the amount of those resources allocated to different traits. In only a very few cases have resource acquisition and allocation been successfully estimated in terms of energy units (e.g. [15–18]). In the majority of studies, these patterns must instead be inferred indirectly from phenotypic patterns.

The problem of estimating acquisition can be avoided in part when acquisition can be experimentally manipulated via resource restriction. When resource levels are restricted, the expectation for resource-based trait values is that they will also decrease. When trait values increase instead or remain constant, it suggests increased allocation to that trait (Figure 1). A well-examined example of this type of pattern is the commonly

116 observed increase in lifespan (hypothesized to be due to increased allocation to somatic
117 maintenance) under food restriction coupled with reduced reproduction (reviewed in [1–
118 5]). The majority of the work on the response of lifespan to food restriction has been
119 focused on model organisms. While there are several examples in non-model species
120 that show a similar response (Table S1), not all species live longer on food restriction
121 [19], including some species of water striders [20], house flies [21], squirrel monkeys
122 [22], and rotifers [23,24]. Additionally, several species show a marked *increase* in
123 reproductive allocation under low resource conditions (flatworms [25], guppies [26],
124 rotifers [24]), demonstrating reproductive allocation does not always decrease under
125 food restriction. Another trade-off that is particularly well characterized in terms of
126 differential resource allocation is the trade-off between flight capability and reproduction
127 in several wing dimorphic insect species (reviewed in [27–30]). In these species, there
128 exist discrete flight capable (macropterous) and flightless (micropterous or apterous)
129 morphs. Wing morphology displays phenotypic plasticity in response to several
130 environmental variables including rearing density, a likely correlate with acquisition, with
131 different species displaying very different responses. In aphids and planthoppers,
132 induction of flight capable morphs increases in response to crowding and low nutrition
133 [31,32], while in crickets, group rearing and other stressors increase induction of
134 flightless morphs [33,34]. Both of these examples, the lifespan-reproduction trade-off
135 and the flight capability-reproduction trade-off, demonstrate the wide variation in
136 allocation patterns across different species.
137 Most experimental manipulations of acquisition simply consider a single “low”
138 and single “high” resource environment, and often the diet used is artificial and quite

139 different from the organism's natural diet. Recently, the community has begun to take a
140 “nutritional geometry” perspective, considering wider ranges of nutritional conditions,
141 both in terms of caloric content and individual diet components (i.e. protein,
142 carbohydrate, and lipid content), as well as a wider range of the timing of resource level
143 changes across an organism's lifetime [35–37]. These efforts provide a much more
144 complete picture of how an organism responds to diet, in that they distinguish between
145 allocation changes due to limitation in specific nutrient classes vs effects due to more
146 general caloric restriction [38,39]. However, this approach increases complexity, which
147 can make interpreting the results in an evolutionary context a challenge when patterns
148 are highly dynamic. To best place diet manipulations in an evolutionary context, we
149 need ecological studies that characterize typical diet sources, and the degree of natural
150 variation in resource availability experienced by populations in the wild. For many
151 populations, this goal will be a challenge.

152 Overall, a broad view of trait variation reveals many examples of variation in
153 plastic resource allocation in response to variation in acquisition (Table S1). Often,
154 patterns vary substantially among closely related species (e.g. [20,22,23,25]), among
155 populations of the same species (e.g. [20,26]), or between different inbred strains [40].
156 These examples argue against any hard and fast, universal resource allocation
157 strategies in response to variation in acquisition and lead to the key questions of why
158 and how environmental variation in resource availability leads to the evolution of
159 different resource allocation strategies. From the resource allocation strategies detailed
160 in Table S1, we can conclude two basic points: 1) phenotypic plasticity in resource
161 allocation is common, and 2) the pattern of plasticity varies widely among populations

162 and taxa. Beyond these points, it is difficult to draw any general conclusions given that
163 the dataset is biased (e.g. model organisms are overrepresented), and there are many
164 different methods for estimating resource allocation (see discussion of these methods
165 above), making it challenging to generalize across studies. Clearly, we require a better
166 understanding of the ecological conditions that would lead to the evolution of such
167 different populations.

168 **3. Why do phenotypically plastic resource allocation strategies evolve?**

169 There is a long and rich history of theoretical evolutionary models addressing both
170 optimal resource allocation patterns in different environmental conditions (i.e. life history
171 evolution models; e.g. [41–45]; see [10,46] for extensive reviews), and the evolution of
172 phenotypic plasticity [47–53]. However, there are few models that specifically focus on
173 the evolution of phenotypically plastic resource allocation in response to variation in
174 resource availability [54–56]. While this category might seem to be a special case, there
175 is reason to expect general models of phenotypic plasticity might not be fully applicable
176 to variation in resource availability. Resource availability places an ultimate constraint
177 on the maximal trait values for allocation-related traits, and in that way, it is
178 fundamentally different from other types of environmental conditions. The dependency
179 creates the somewhat paradoxical situation in which no plasticity in allocation will lead
180 to plasticity in trait values, as they will necessarily decrease with resource availability
181 (Figure 1). Thus, it is critical for theoretical models to explicitly consider variability in
182 resource availability when predicting how plastic allocation strategies will evolve.

183 One emergent property of models that do explicitly consider the interplay
184 between acquisition and allocation is that environmental predictability (i.e. whether

current resource availability is correlated with future availability) is a major determinant of the pattern of phenotypic plasticity that evolves [54–56]. In a model considering allocation to flight capability versus reproduction, King *et al* [54] showed completely opposite patterns of plasticity in allocation are expected to evolve in environments with predictable versus unpredictable patterns of resource availability. Fischer and co-workers [55,56] showed that, in response to short term resource availability fluctuations, populations should evolve to allocate toward somatic maintenance under low food conditions. However, this response is more complicated. If conditions are low enough to be indicative of low survival probability, allocation to survival is not favoured. Rather, a terminal investment strategy, investing heavily in reproduction at the expense of survival, is favoured.

One area where models of the evolution of condition-dependent (i.e. acquisition-dependent) resource allocation strategies is well developed is in the field of sexual signalling. In many cases, male advertisements to females are dependent on the condition of the male, producing so-called ‘honest’ signals (e.g. [57,58]; for reviews see [59–61]. This condition dependence can be continuous (e.g. call duration in male grey tree frogs [57]) or a discrete polymorphism (e.g. sexually dimorphic mandible growth in stag beetles [62]). There are several models considering how the benefits and costs of increased allocation toward a sexual signal change depending on an individual’s condition [59,61,63], with models predicting low condition individuals that allocate more toward sexual signals experience lower benefits and/or higher costs depending on the assumptions of the model (see [61]). These models are a subset of models considering allocation strategies in poor condition as a ‘best of a bad lot’ strategy [46]. In essence, it

208 does not pay to invest heavily in a sexual signal if one simply does not have enough
209 resources to produce a high-quality signal that will attract many mates.
210 The majority of evolutionary models focus solely on why, not how, allocation
211 patterns evolve, ignoring the underlying mechanisms. Often, this is a sensible strategy,
212 given that when mechanisms don't act as ultimate constraints, evolutionary endpoint will
213 remain the same, irrespective of the specifics of the mechanistic underpinning.
214 Nevertheless, evolutionary models that incorporate explicit mechanisms, can be highly
215 informative in explaining the mechanisms underlying evolutionary patterns. For
216 example, Mangel and Munch [64] integrated physiological parameters such as oxidative
217 damage associated with faster growth and resource allocation to damage repair in a
218 model predicting when compensatory growth (increased allocation to growth following a
219 period of food restriction) should evolve. Only by explicitly incorporating the
220 physiological mechanisms of damage and repair, were they able to simulate patterns of
221 compensatory growth that matched observations. Compensatory growth never arose
222 using a simple optimality framework, demonstrating that explicitly incorporating
223 physiology can fundamentally change the predictions of life history models in some
224 cases. We encourage the development of evolutionary models that integrate proximate
225 mechanisms as a way to expand our understanding of the evolution of resource
226 allocation strategies in multiple systems.

227 **4. Genetic and physiological mechanisms underlying phenotypic plasticity in**
228 **resource allocation**

229 It is clear organisms have evolved the ability to shift the allocation of resources in
230 response to their nutritional state in many different ways, but *how* do they accomplish

this change? What physiological changes accompany a shift in allocation strategy and what genes are involved? Not surprisingly, the greatest progress in the effort to uncover the mechanisms governing the coordination between acquisition and allocation comes from model organisms (e.g. yeast, worms, flies, and mice) that have been the focus of studies for decades. However, the relatively recent "omic" technologies available, and the decreasing cost of these technologies, make it increasingly feasible to gather data at multiple levels of the genotype to phenotype map in multiple environments for nearly any organism, opening up the possibility of moving beyond unnatural manipulations in model organisms and toward more ecologically relevant contexts.

a. Evolutionary endocrinology suggests key role of hormones in resource allocation

At first glance, resource acquisition and allocation might seem hopelessly complex, casting doubt on the prospect of uncovering the proximate mechanisms involved in the relatively subtle variation, at least when compared to mutants, in natural populations. However, an emergent theme from several systems, including many of the above detailed examples in model organisms, is the key role of hormone pathways as major determinants of resource allocation. These discoveries have spurred the expansion of the field of “evolutionary endocrinology” [65–68]. For instance, we have learned a great deal about the mechanisms governing allocation of resources in response to environmental changes from genetic screens and mutational analysis in model organisms (e.g. [68–70]). In this section, we review our current knowledge in model and non-model systems on how plasticity in nutrient allocation and hormonal

253 signalling impact reproduction-lifespan and reproduction-dispersal trade-offs. The
254 hormone pathways we discuss here include insulin, ecdysone, and juvenile hormone.

255 **i. Large effect mutations support the idea that major signalling pathways**
256 **modulate allocation**

257 Studies that have yielded the most insights have tended to focus on mutations of
258 large effect. Several studies have implicated leptin as a mediator of acquisition and
259 allocation of nutrients. In mammals, leptin together with AMPK (AMP-activated protein
260 kinase) control appetite thus regulating nutrient intake [71], and leptin also mediates the
261 energetic trade-off of reproduction with the immune system by acting as a proximate
262 endocrine indicator of the energy state to the immune system [72]. In *D. melanogaster*,
263 Upd2 (unpaired 2), a functional homolog of leptin [73], causes a nutrient dependent
264 effect on growth, mediating production of Dilps (*Drosophila* insulin-like peptides) in the
265 fed state, and subsequent secretion of insulin in response to dietary fat [73]. These
266 studies demonstrate a direct connection between nutrient limitation and allocation.

267 Both ecdysone and the insulin/insulin-like signalling pathway (IIS) have a role in
268 the plastic allocation of nutrients. Sequential perturbation of IIS and ecdysone signalling
269 in ovarian somatic cells of *D. melanogaster* on different diets showed that ecdysone
270 signalling regulated the rate of increase in ovary volume in general while IIS conferred
271 the same effect before larvae attained critical weight [74,75]. This nutrient-dependent
272 development of the ovary illustrates the role of hormonal signalling in plastic allocation
273 of nutrients. Perhaps one of the most significant contributions emerging from mutation
274 studies is that IIS signalling pathways are critical in the regulation of lifespan in many
275 species. In several model organisms (including fly, mice and worm), reduced IIS

276 phenocopies nutrient deprivation, resulting in longer-lived individuals (e.g. [76–78]). In
277 addition, a suppressed IIS or removal of the germ-line produces life extending effects by
278 activating the forkhead transcription factor (FOXO) which is conserved across *C.*
279 *elegans* (*daf*-16), *D. melanogaster* (dFOXO) and mammals (FOXO3a) [79,80].
280 At the whole-body level [71], AMPK regulates metabolic energy balance by
281 affecting feeding behaviour and circadian rhythms. When nutrient abundance is low, the
282 elevated AMP to ATP ratio activates AMPK, with subsequent gain in health span and
283 longevity in *D. melanogaster*. AMPK is a conserved modulator of lifespan in flies and
284 mammals linking energy sensing to longevity, and is emerging as a major mechanism
285 accounting for variation in longevity [71].

286 **ii. Lessons from studies with more ecological context**

287 Hormone pathways have also been implicated in nutrient allocation shifts in non-
288 model systems. Studies in flies and beetles have likewise suggested the IIS as a major
289 pathway involved in resource distribution. An exonic indel polymorphism in the Insulin-
290 like Receptor (*InR*) gene was identified as a functional direct candidate target of natural
291 selection in wild *D. melanogaster* [81,82]. In rhinoceros beetles, horn size is highly
292 sensitive to nutrition and to perturbations in the IIS than are other body structures [83].
293 The precise details about how nutrients are mobilized toward competing traits have
294 perhaps been best characterized in the wing dimorphic sand cricket, *Gryllus firmus* [84].
295 Juvenile hormone (JH) levels determine the morph, and trigger a whole host of
296 processes leading to differential allocation of actual resource components toward flight
297 capability versus reproduction. Flight capable morphs preferentially metabolize amino
298 acids and convert a larger proportion of fatty acids to triglycerides while flightless

299 morphs preferentially metabolize fatty acids and convert a larger proportion of amino
300 acids to ovarian protein [65,84]. Adult crickets on low food diets allocate proportionally
301 fewer resources toward flight capability [17,18], however, whether this diet-dependent
302 shift is also mediated through JH has not yet been established. Juvenile hormone
303 signalling is also involved in nutrition-based sex-specific mandible development via
304 *doublesex* gene in the staghorn beetle [62].

305 These studies support the hypothesis that the evolution of allocation patterns
306 ultimately results from the evolution of key endocrine pathways [66–68], potentially
307 providing a simple theme in complex web of traits at various levels. Thus, while there is
308 no denying acquisition and allocation of resources are highly complex processes, it is
309 clear that hormone pathways serve as major mediators in many cases.

310 **iii. Understanding the underlying genetics of natural variation**

311 Most of the above-described studies that identify key genes (except a few e.g.
312 [81,82]) rely on evidence from large effect mutations or major perturbations and they
313 have been very successful at identifying genes involved in the regulation of metabolism
314 and resource allocation and of the effects of large alterations to individual genes. Our
315 knowledge of the genetic basis of *natural* variation in metabolism and resource
316 allocation is severely lacking in comparison, a predicament that is shared by the
317 majority of complex traits [85–87]. The large effect genetic mutants identified via
318 classical genetic techniques are typically not segregating in natural populations, which
319 is not surprising given the central role of the pathways involved [88]. Additionally,
320 despite the fact that several large effect mutations have been found to influence lifespan
321 in *D. melanogaster* (see [3]), mapping studies and evolution experiments using natural

322 populations have not independently identified these same genes as important
323 contributors to natural genetic variation (e.g. [3,89–91]), with few exceptions [85,86].
324 These results are not due to a lack of genetic variation at these loci, given the
325 populations used are derived from wild populations and typically have high heritabilities
326 for most phenotypes, including gene expression levels of some of these same genes
327 [92]. There are several possible explanations for this large disconnect regarding genes
328 in these hormone pathways: 1) they do not contribute to natural genetic variation, 2)
329 their effects are subtle and thus difficult to detect, 3) their effects stem from *trans*
330 regulatory changes affecting gene expression [93]. Large effect mutant studies may
331 represent the extreme tail of effect size distribution in nature [14], or, in the case of
332 increase in longevity, different mechanisms altogether may induce altered nutrient
333 signalling pathways in captive populations due to absence of stressors [86,87,94].
334 One of the strongest messages to emerge from modern quantitative genetics is
335 that the genotype to phenotype map is more complex than some anticipated [95]. Within
336 this complexity, our goals should be to find the main roads and general patterns. Newer
337 mapping strategies, such as multi-parental populations, will help to assay multi-level
338 traits on the same set of lines and leverage what is known about hormone pathways to
339 reveal mechanistic bases of plastic resource allocation in natural organisms.

340 **b. Integrating genetic and physiological mechanisms into evolutionary**
341 **perspectives of resource allocation**

342 As with the above evolutionary models, traditionally, questions surrounding
343 proximate mechanisms have been considered separately from evolutionary questions,
344 with a more recent movement toward integration across sub-disciplines. In particular, a

345 major question surrounding hypothesized resource-based trade-offs is the degree to
346 which the proximate mechanism underlying trade-offs stems from functional resource
347 competition, or whether some other mechanism (e.g. hormone signalling), produces the
348 relationship between traits. Here, we argue that these proximate mechanisms are not in
349 conflict with the conceptual framework of the Y model.

350 **i. Challenge of a resource-based Y model**

351 The Y model of resource allocation, as a framework to explain proximate
352 mechanisms underlying life history trade-offs [12,96], has in recent years been criticized
353 by some as inadequate, leading some to seek revision of life history theory (see
354 exchanges in [97–101]). The challenge to a resource-cantered model is based on new
355 empirical data showing that 1) abrogation of reproduction does not always extend
356 lifespan, 2) some mutations that extend lifespan do not affect, or in fact, increase
357 fecundity, and 3) male and female organisms of several species respond differently to
358 interventions that increase lifespan. The most notable of these are studies in *C. elegans*
359 [102,103] and *D. melanogaster* [104,105] in which gonad ablation failed to increase
360 lifespan, while ablation of the germline only, doubled lifespan. Evidence suggests this
361 effect is mediated largely by the insulin/IGF-1 system, which is thought to integrate
362 molecular signals from the germ line and those from the somatic gonad to determine
363 lifespan, rather than direct redistribution of resources. This hormonal signalling
364 alternative has spurred a vigorous debate [99,100] whose reconciliation, in our view,
365 depends on the eventual and successful integration of proximate mechanisms of trade-
366 offs into evolutionary theory.

367 **ii. Is the new data really in conflict with the Y model?**

368 We have reviewed above, case studies that directly or indirectly offer support for a
369 resource model of life history evolution. Of particular note are studies demonstrating
370 preferential amino acid metabolism and allocation of fatty acids to either flight or
371 reproduction in winged vs wingless cricket morphs [84,106–110]. These works
372 represent compelling evidence for differential resource allocation associated with the
373 flight capability-reproduction trade-off. In addition, studies that quantified amino acid
374 metabolism *in vivo* confirmed the predictions of the Y model for this trade-off [84,107].
375 Studies that fail to find the trade-off or find a positive relationship may not logically
376 invalidate those that observe a negative correlation as multiple factors may be
377 responsible. Further, the bulk of known mechanisms have been described in non-
378 natural laboratory mutant organisms with limited or zero selection pressures
379 experienced in the wild [86,87,94]. Although, the evidence for the connection between
380 signalling and resource allocation is unclear, this absence of evidence should not be
381 treated as evidence of absence.

382 **iii. Opportunities for integration of fields**

383 Conceptual dichotomies where available empirical data do not sufficiently fit
384 standing theoretical principles are not new to biology. These apparent conflicts have
385 fuelled progress of the broad field and successfully led to the integration of once thought
386 disparate fields – Mendelian, molecular, and quantitative genetics in the last century
387 (see [111]). Instead of asking whether survival costs are best explained either by literal
388 resource competition or by resource-free signalling, it may be useful to explore how the
389 two integrate into the observed trade-off. This strategy can redirect attention to potential
390 connections between nutrients and signals and factors that affect that connection. There

391 is strong evidence that hormonal signalling is involved in nutrient sensing mechanisms
392 implicated in aging [112], and that these mechanisms are at the base of appetite
393 regulation and redistribution of nutrients [71]. It is thus possible to see how hormonal
394 signalling may regulate optimal allocation and account for evolution of diverse resource
395 allocation patterns. Thus, new data showing that signals regulate lifespan do not,
396 presently, preclude the evolutionary role of resource constraints, especially in natural
397 settings. It is completely fitting with evolutionary theory to expect organisms to use
398 specific cues to indicate environmental conditions such as food availability. Thus, when
399 results find that a single amino acid level can change how organisms allocate resources
400 [113], an evolutionary interpretation is that that amino acid is what is cueing the
401 organism about the environment, not that actual resource levels are not important to the
402 response.

403 We do not yet know whether one or more proximate explanations govern a given
404 trade-off. A possible scenario to emerge may negate the notion of a single proximate
405 explanation since there may be unique mechanisms in different species and/or
406 environmental settings. For example, in selected lines of *D. melanogaster*, offspring
407 ovariole number increased in response to maternal starvation [114]; in wild-living *D.*
408 *melanogaster* larval age and larval weight predicted survival in temperate and tropical
409 regions, respectively [115]; and, in redback spider dietary restriction extended lifespan
410 in mated, but imposed cost in unmated females [116]. These examples suggest multiple
411 mechanistic possibilities defining a given phenotypic trade-off in different species or
412 within species in different environments. Whether the trade-off is affected by diet,
413 temperature, or behaviour, molecular signalling could lead to changes in how resources

414 are allocated. It will benefit both fields if future studies take advantage of the 'omic'
415 technologies to step up cross-field approaches in the search for mechanisms governing
416 these traits in nature.

417 **5. Future Directions**

418 In this review, we have attempted to argue that a resource-based Y model is
419 uniquely favoured to facilitate integration of evolutionary life history theory with
420 proximate mechanisms underlying the near-ubiquitous trade-offs in life history traits. In
421 doing so we have brought to the fore two key areas where significant progress is
422 attainable, especially with the aid of 'omic' approaches: 1) performing studies in more
423 ecologically-relevant contexts, and 2) increasing the level of integration between fields.

424 A major gap in our understanding of life history trade-offs in general, and the
425 relationship between survival and reproduction in particular, is a general paucity of
426 studies focusing on the underlying mechanisms in natural species, and lack of
427 concordance between results of mutational studies in model species and those from
428 studies of natural variation in the few cases where these have been undertaken. Here,
429 we have attempted to show the wide variety of plastic resource allocation strategies in
430 response to environmental fluctuations in availability that exist among natural
431 populations and species. Understandably, many of the patterns so far uncovered have
432 been demonstrated using laboratory studies with explicit diet manipulations (at most,
433 three diet variations). We support this approach but, in addition, advocate for a broader,
434 more realistic consideration of experimental diets. In this direction, studies taking
435 'nutritional geometry' approaches discussed earlier have the potential to provide a
436 broader understanding of how organisms respond to changes in diet. In addition to

437 considerations of mere nutritional content, experimental diets should attempt to mimic
438 the natural diet of the organism as closely as possible, and the natural range of
439 availability in the field in order to ensure that results most reflect evolutionarily relevant
440 patterns that occur in nature.

441 Secondly, we have highlighted gaps in theoretical evolutionary models that
442 address both optimal resource allocation patterns, and the evolution of phenotypic
443 plasticity. To our knowledge, very few models specifically focus on evolution of
444 phenotypically plastic resource allocation in response to variation in resource
445 availability. We encourage the development of evolutionary models that integrate
446 proximate mechanisms as a way to expand our understanding of the evolution of
447 resource allocation strategies in multiple systems. In addition, testing the predictions of
448 models predicting the evolution of different resource allocation strategies should be a
449 major priority. Natural systems where patterns of availability differ among populations
450 and species, can also inform these questions. Alternatively, experimental evolution
451 approaches, where resource availability can be altered in a controlled way, and different
452 types of variability across time can be induced, are potentially a powerful way to test
453 these models. An experimental evolution approach could also allow for tracking change
454 across the genotype to phenotype map in an integrative way, tracking changes in
455 proximate mechanisms as evolution occurs.

456 Overall, viewing phenotypes within a framework of resource acquisition and
457 allocation allows for a natural integration of physiology, genetics, and evolution. Studies
458 that measure phenotypes at multiple levels (genomic, physiological, organismal levels)
459 and in multiple resource environments provide a potentially productive path forward.

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462 **Competing interests**

463 We have no competing interests.

464 **Author's contributions**

465 EGK and EN conceived the idea and wrote the manuscript; AMP and EN compiled

466 Table S1, EN, AMP and EGK provided editorial comments. All authors gave final

467 approval for publication.

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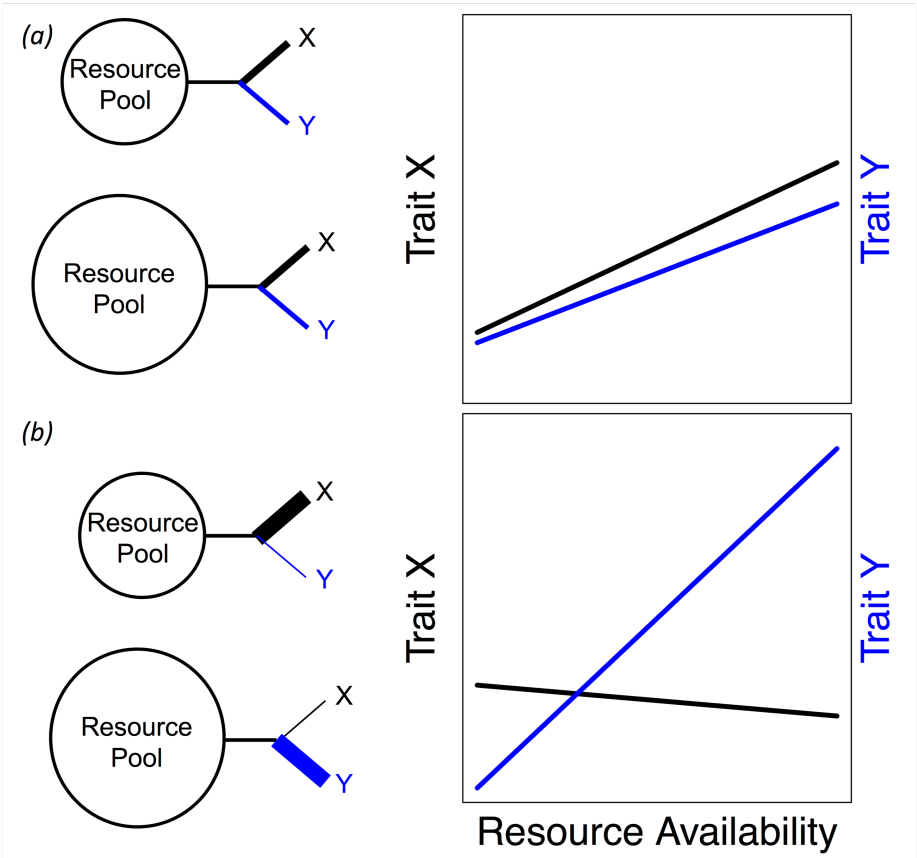

Figure 1: Expectations for trait values for two traits involved in a resource allocation trade-off when A) there is no phenotypic plasticity in allocation in response to resource availability, and B) there is phenotypic plasticity in allocation with increasing proportions allocated to trait Y as resource availability increases.

Table S1: Examples of the diversity of resource allocation strategies in some life history traits across the animal kingdom.

Trade-off	Taxa	Increased allocation with low resource availability	Selected examples
a) Trade-offs presented in the literature as two-trait cases			
Dispersal–reproduction	*Prokelisia marginata*	dispersal	[1]
	Gryllus rubens	flightlessness	[2]
	Gryllus firmus	flightlessness	[3]
	Gryllus firmus	adults to dispersal; juveniles to reproduction	[4,5]
Growth–reproduction*	*Daphnia magna*	growth	[6]
Current reproduction–future reproduction	*Synchaeta pectinata*	current reproduction^	[7]
Storage–reproduction	*Drosophila melanogaster*	storage	[8]
Survival–reproduction#	*Nothobranchius furzeri*	survival	[9]
	Trichoptera spp.	to storage in short lived species; no change in long lived species	[10]
	Elephas maximus	survival	[11]
	Poecilia reticulata	reproduction$	[12]
	Theraphosidae	survival	[13]
	Callosobruchus maculatus	survival	[14]
	Asobara tabida	survival	[15]
	Anastrepha ludens	survival	[16]
	Notiophilius buguttatu	survival	[17]
	D. melanogaster	survival	[18–22]
	Rhabditophora	reproduction	[23]
	10 rotifer species	most to survival	[24]
	Eupelmus vuilletti	depends on nutrient (lipid vs sugar)	[25]
	Romalea microptera	similar allocation to both traits	[26]
	Odocoileus virginianus	survival	[27]
	Larus michahellis	survival	[28]
	Saccopteryx bilineata	survival	[29]
	Gerris spp.	survival	[30]
	Diomedea exulans	survival (with terminal reproductive investment)	[31]
Survival–body size	*Macaca mulatta, Saimiri* sp.	M. mulatta to survival; Saimiri sp no effect	[32]
Starvation resistance–reproduction	*D. melanogaster*	starvation resistance	[18]
Egg mass–clutch size	*Microlophus delanonis*	to none	[33]

Trade-off	Taxa	Increased allocation with low resource availability	Selected examples
Total clutch mass–post-nesting condition	*Microlophus delanonis*	to none	[33]
Egg size–egg number	*Northobranchius furzeri*	egg size	[9]
Offspring quality–reproduction	examines 24 bird species on elevational gradient	quality in high elevation species	[34]
b) Trade-offs presented in the literature as multi-trait cases			
Body condition–progeny quality[‡]	*Paroedura picta*	body condition	[35]
Dispersal–survival–reproduction–body mass	*Speyeria mormonica*	survival	[36]
Dispersal–lifespan–reproduction	*Colias eurytheme, Speyeria mormonica*	dispersal, no effect on lifespan	[37]
Growth rate–development time–body size	*Scathophaga stercoraria*	faster growth rate and faster development time	[38]
Growth–reproduction– sprint speed	*Anolis sagrei*	survival	[39]

[#]Many of these refer to early reproductive effort; *current reproduction; ^ovary size reduction, not absolute resource allocation; [‡]body length, head length vs clutch size, egg size; [$]Study measures somatic investment which may benefit both lifespan and fecundity.

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