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An ontogenetic perspective on the relationship between age and size at maturity

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Summary

1. Understanding the relationship between age and size at maturity is essential because these traits are pivotal determinants of an organism's fitness.

2. The relationship between age and size is commonly addressed using optimization and quantitative genetic approaches. Here we argue that the value of such studies is often limited by an insufficient consideration of organismal ontogeny.

3. On the basis of a simple conceptual framework of hierarchical resource allocation, we identify key aspects of ontogeny that prove critical to a fuller understanding of the relationship between age and size, and which, to date, have been insufficiently explored. In particular, these include intrinsic variation in growth rate within and among populations, and the physiological nature of the maturation process that co-ordinates growth and reproductive function in an organism.

4. We also provide some guidance to the empirical investigation of these aspects, anticipating that a wider theoretical, but especially empirical appreciation of ontogenetic detail will greatly increase the explanatory and predictive power of life-history studies.

Key-words: genetic correlation, growth rate, maturation, physiology, reaction norm

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Introduction

Age and size at maturity have a strong direct or indirect impact on an organism's fitness. These traits are therefore of central interest to life historians and ecologists alike (Peters 1983; Stearns 1992; Roff 2002), and figure among the most frequently studied organismal characters. The relationship between age and size at maturity is typically approached from two different angles. On the one hand, the relationship between the two traits is often addressed within the context of phenotypic plasticity using optimality approaches. The aim is to understand and predict how changes in environmental variables, such as temperature or food availability, alter the (optimal) relationship between age and size. Constraints on the independent adjustment of the two traits are usually neglected. Not surprisingly, optimality models have rarely proved successful in providing robust predictions of the reaction norms of age and size at maturity (Day & Rowe 2002).

On the other hand, quantitative geneticists focus on constraints on the joint evolution of age and size, commonly assuming a tight genetic association between the two characters within populations or species. Accordingly, benefits that accrue from a short developmental period should generally trade off against benefits that arise from prolonging development and thus growing larger. However, this assumption of an age and size trade-off, which suggests a relatively uniform intrinsic growth rate, is controversial. For instance, a recent review (Roff 2000) concluded that there is often considerable genetic variation in growth rates within populations. Further, the expression of genetic correlations is generally population- and environment-dependent (Sgro & Hoffmann 2004), complicating predictions of evolutionary change in age and size. Finally, we should be interested in the proximate mechanisms underlying trade-offs, about which simple correlations between age and size, as statistical abstractions, usually do not tell us much.

Obviously, optimization and quantitative genetic approaches fail to capture some detail in the relationship between age and size at maturity. It has long been recognized that this shortcoming is a consequence of the negligence of underlying ontogenetic (here used synonymously with developmental) processes within these research fields (Atchley 1984; Reznick 1990; Cheverud 1996; Schlichting & Pigliucci 1998; Johnson & Porter 2001; Wolf *et al.* 2001). Nevertheless, the integration of ontogeny in life-history studies has proceeded surprisingly slowly, and the empirical value of theoretical

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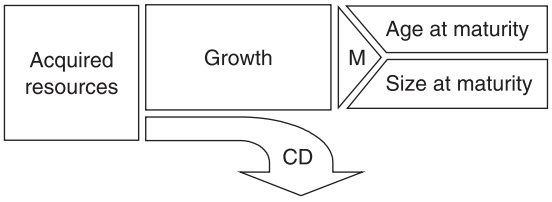

Fig. 1. Simplified hierarchical representation of ontogeny, with age and size at maturity as phenotypic end-points. The acquisition of resources forms the base of the hierarchy. At one or several superior level(s) (only one depicted), acquired resources are allocated to somatic growth on the one hand, and to a number of competing demands (CD) important to an organism's performance on the other hand. During maturation (M), the organism switches from growth to reproduction and the time–size phenotype becomes fixed. More details are given in the text.

attempts in this direction often remains elusive. The objective of our comment is to illustrate how a consideration of the way phenotypes are constructed during ontogeny can enhance our understanding of the relationship between age and size at maturity. Starting from a classical conceptual model of resource acquisition and allocation, we identify and discuss key components of ontogeny, which in conjunction essentially determine the relationship between age and size. We also provide some guidance to the empirical investigation of these determinants. Increased efforts along these lines promise to facilitate predictions of the joint evolution of age and size, and to help examine critically the adaptive value of their reaction norms.

AGE AND SIZE IN AN ONTOGENETIC CONTEXT

We envisage age and size at maturity as end-points of a resource-acquisition and -allocation cascade (Fig. 1) (van Noordwijk & de Jong 1986; Riska 1986; de Jong 1993). At the most basal level, an organism acquires limited resources during ontogeny. Resource acquisition depends on various factors, including food or nutrient availability in the environment, an individual's physiological capacity to extract nutrients from substrates, or individual foraging behaviour. At one or several superior level(s), defined allocation (or priority) rules (Zera & Harshman 2001) govern the division of acquired resources between growth and a number of competing demands (CD; Fig. 1). These competing resource sinks include diverse characters important to an organism's immediate or future performance, such as maintenance, resource storage, immune function or predator defence. During eventual maturation (M; Fig. 1), the transition from growth to reproduction produces an observable relationship between age and size at maturity. Note that, for the sake of generality, we define maturation very broadly: it subsumes all physiological regulatory processes involved in the transition from a non-reproductive growth phase to reproductive function. Maturation therefore encompasses ontogenetic phenomena as diverse as amphibian metamorphosis or the photoinduction of flowering in a plant. Despite the tremendous diversity and complexity of the processes underlying maturation, we argue that strong parallels across taxa justify this simplified view.

From the hierarchical representation of ontogeny shown in Fig. 1, it becomes evident that two specific aspects require particular, simultaneous consideration if we are to improve our understanding of patterns of correlation between age and size at maturity, and their reaction norms: (1) the factors that determine the amount of resources available to growth, and (2) the nature of the maturation process that co-ordinates growth and reproductive function. These issues are discussed in the following sections.

ACQUISITION AND ALLOCATION OF RESOURCES TO GROWTH

Acquisition directly determines the total amount of resources an organism can distribute among competing life functions, as emphasized by theoretical models of hierarchical resource acquisition and allocation (van Noordwijk & de Jong 1986; Riska 1986; de Laguerie *et al*. 1991; de Jong & van Noordwijk 1992; de Jong 1993; Worley, Houle & Barrett 2003). Applied to growth, this means that individuals that acquire resources more efficiently than others will be able to allocate more resources to both growth and competing resource demands. If there is little variation at the acquisition level, however, growth rate can still differ among individuals if they follow different physiological priority rules of resource allocation to competing life functions. In order to understand variation in growth rate between individuals, the sexes or populations, we thus need a thorough understanding of how environmental and genetic factors influence the resource cascade at different hierarchical levels.

An important and well studied issue in this context is variation in resource acquisition owing to environmental factors. A wealth of experimental studies have documented how food quantity or quality, the presence of competitors or predators, or abiotic constraints on activity affect an organism's ability to acquire resources and thereby modify growth rate. How the rules for allocation to competing resource demands can vary in response to environmental factors has, however, received little empirical attention so far (Zera & Harshman 2001).

More importantly, genetic (intrinsic) variation in growth performance within populations and species remains poorly understood and little explored, although it is frequently demonstrated or inferred (Arendt 1997; Roff 2000 and references therein). A fundamental explanation for the occurrence and maintenance of genetic variation in growth rate is the occurrence of trade-offs: a high growth rate might come at the expense of other functions critical to an organism's life-history.

At the resource-acquisition level, for example, fast growth due to high foraging effort has been demonstrated to entail increased predation risk in butterfly larvae (Gotthard 2000). At the allocation level, high growth rate has been shown to trade off with immune function under low food conditions in *Drosophila* (Kraaijeveld & Godfray 1997); with plant height (a competitively advantageous trait under low herbivore pressure) in a grass species (Hartvigsen & Naughton 1995); and with predator escape ability in a fish (Lankford, Billerbeck & Conover 2001). All these studies highlight that the optimal combination of resource allocation to growth and competing demands is a function of the environment. Considerable variation in growth rate within and among populations should therefore be maintained by spatio-temporal fluctuation in direct or indirect selection on growth rate (Gillespie & Turelli 1989; Reznick, Nunney & Tessier 2000).

As an additional explanation, standing genetic variation in growth rate might plausibly reflect mutation–selection balance (Charlesworth 1990; Houle 1991; Lynch & Walsh 1998): resource allocation, and especially acquisition traits, are probably encoded by a great number of loci, which mutations with mostly deleterious and pleiotropic effects can target. Such deleterious alleles that depress growth rate might sometimes become expressed only under environmental stress (Kause & Morin 2001), in novel environments (Service & Rose 1985), or as a consequence of inbreeding (Charlesworth 1990; Houle *et al*. 1994).

METHODOLOGICAL ISSUES IN THE STUDY OF GROWTH

Although rarely documented, growth trade-offs certainly play a key role in the maintenance of genetic variation in growth rate among individuals, the sexes and populations. They can be identified by the usual methods for detecting trade-offs (Reznick 1992; Zera & Harshman 2001; Roff 2002), including (1) the measurement of phenotypic correlations between growth rate and potential competing life functions in unmanipulated individuals; (2) the measurement of genetic correlations between growth rate and competing traits (e.g. using artificial selection; Kraaijeveld & Godfray 1997); or (3) by experimentally modifying growth rate (or competing characters) and measuring the response in the non-manipulated trait(s) (Lankford *et al*. 2001). Each of these empirical methods has its advantages and weaknesses, and a combination of different approaches is most informative. Likewise, studies of growth trade-offs should ideally be complemented by ecological investigations aimed at elucidating the fluctuating selection context that potentially maintains intrinsic variation in growth rate. It is also important to bear in mind that the trade-off between growth rate and competing life functions may become expressed only after some time, and that individual differences in resource acquisition might mask differences in allocation priority rules at higher levels in the cascade. Further, a deeper understanding of growth trade-offs will benefit from investigations of the underlying neuroendocrine processes. Significant advances in this direction are currently being made in some model systems (references in Zera & Harshman 2001). Finally, it would be important to address experimentally the importance of mutation–selection balance as an alternative to trade-offs in maintaining genetic variation in growth rate. This issue remains largely unexplored.

Another aspect that has received very little attention so far is the hierarchical level at which differences in growth rate arise (Worley *et al*. 2003). Do individuals or populations differ more frequently in foraging behaviour or in other characters related to resource acquisition, or does variation arise more often in physiological allocation rules at higher levels? Analyses of the genetic correlations between growth rate and multiple traits located at different levels in the acquisition-allocation cascade are most useful to address these questions. For instance, Kause *et al*. (1999) have demonstrated that genetic variation in growth rate in a moth population is likely to be attributable to individual differences in acquisition traits, such as relative consumption rate and conversion efficiency, while *Sceloporus* lizard populations have been shown to differ in their rules of resource allocation to growth *vs* reproduction (Niewiarowski 2001).

The methodological aspects discussed above raise the question of how growth rate is best quantified. While growth rate is intuitively understood by most people, and features as a variable in many life-history models (e.g. Abrams *et al*. 1996), its empirical measurement and analysis are not so straightforward. In the simplest case, which assumes a linear relationship between age and size, growth rate can be expressed and measured as body size (mass or length measures) accumulated per unit time. If this covers a short period (a few hours or days for an insect; Kingsolver, Ragland & Shlichta 2004), we obtain an estimate of the instantaneous growth rate at a given point in time or life stage at a fine resolution. Frequently, however, this approach is used to estimate overall growth rate, which then equals the slope of the straight line linking final adult body size and total development time (or age at maturity). This simple estimate of growth rate is then usually compared among individuals or groups (sexes, families, populations, species) using standard univariate ANOVA.

In reality, the relationship between adult size, S, and development time, t, in most organisms from insects (Teuschl, Reim & Blanckenhorn 2007), birds (Teather & Weatherhead 1994) and primates (Leigh 1992) to plants (Yin *et al*. 2003), is not linear, but is best described by an asymmetrical sigmoid function such as

$$S(t) = \frac{S_{\max}}{(1 + ve^{-k(t-t_m)})^{(1/v)}}, \qquad \text{(eqn 1)}$$

where t_m is the inflection point and $S_{\max}$ the asymptotic (final, maximal) adult size attained, and k and v are

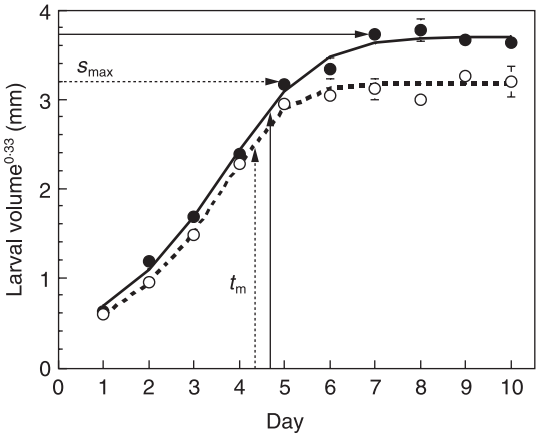

Fig. 2. Growth trajectories (mean cube root-transformed body volume ± SE per day; $N = 2$–14 per treatment combination and day) of yellow dung fly (*Scathophaga stercoraria*) larvae from laboratory lines selected for large (closed symbols, solid line) and small body size (open symbols, hatched line) at unlimited larval food (0 = day of egg laying), together with their estimated growth functions. Estimated parameters (see equation 1) for the large line: asymptotic size $S_{max} = 3{\cdot}71$, inflection point $t_m = 4{\cdot}51$, $k = 1{\cdot}18$, $v = 2{\cdot}46$; small line: $S_{max} = 3{\cdot}20$, $t_m = 4{\cdot}32$, $k = 1{\cdot}34$, $v = 2{\cdot}47$. Data from Teuschl *et al*. (2007).

two constants determining the curvature and degree of asymmetry of the function, respectively (an example is given in Fig. 2). The growth trajectory of an individual, or a population of individuals, is estimated by repeatedly measuring body size as individuals grow, and a sigmoid function is fitted to these data (Yin *et al*. 2003; Fitzmaurice, Laird & Ware 2004; Wellock, Emmans & Kyriazakis 2004).

To compare the growth function among individuals or groups, ANOVA can be performed using only the estimated, diagnostic function parameters (Teather & Weatherhead 1994). As an alternative, Teuschl *et al*. (2007) split the growth functions of populations of dung flies into two parts: the initial exponential growth phase and the terminal asymptotic phase. When the data are properly log- or cube root-transformed, the former phase is essentially linear (as is the flat, latter phase), so growth rate can be estimated by the slope of the initial exponential body-size increase (days 1–5 in Fig. 2), and adult body size by the asymptotic size (S_{max}, days 7–10). A method of analysing genetic variation in ontogenetic trajectories based on covariance functions that describe genetic variation in body size at any point along the growth curve was introduced by Kirkpatrick, Lofsvold & Bulmer (1990). Unfortunately, this method is technically demanding, so few people have used it and probably will in the future (but see Kingsolver *et al*. 2004 for an application). Other methods for analysing growth trajectories are available in the literature (e.g. Klingenberg 1996), or might arise.

Regardless of the specific growth function and analysis method employed, however, the critical point here is that the quantification of growth rate based on ontogenetic trajectories is generally much more informative than the use of the simple, linear size-by-time ratio. Although the latter approach can provide appropriate estimates of the instantaneous growth rate over short time spans of an organism's ontogeny (e.g. Fig. 3), growth trajectories not only capture growth rate alone, but also provide a simultaneous description of the relationship between growth rate, body size and development time throughout ontogeny. Hence the growth trajectory represents an excellent heuristic tool that permits at least a broad, phenomenological identification of ontogenetic events (e.g. the onset of maturation, see below) that translate to observed differences in age and size at maturity, as well as formulating testable hypotheses about the physiological processes underlying these events. Any mechanistic approach, such as that taken by Davidowitz and colleagues described below, will profit from a detailed analysis of growth trajectories to gain more insight into the simultaneous regulation of growth rate, age and size, and its evolution.

Another aspect of the study of growth rate concerns the dimension in which organism size is measured. As growth is the conversion of acquired resources to biomass, its quantification using (three-dimensional) mass is intuitively most appealing. For at least two additional reasons, mass rather than linear structures should generally be recorded. First, the proportion of resources allocated to specific tissues might change during ontogeny (allometric growth), potentially leading to biased estimates of growth rate when measuring the length of a single structure. Second, the measurement of body mass yields more precise growth trajectories in arthropods where mass gain is continuous, whereas external structures grow stepwise during moults.

MATURATION: THE CO-ORDINATION OF GROWTH AND REPRODUCTIVE FUNCTION

Correlations between, and reaction norms of, age and size at maturity do not result from variation in growth rate alone. Rather, they arise from the neurophysiological processes that co-ordinate growth and reproductive function (Fig. 1) (Bernardo 1993). This maturation process is traditionally studied within the realm of developmental biology and physiology, and very rarely considered in evolutionary work. Yet recent progress in a few model organisms underscores the key role of maturation determinants in life-history evolution. For the sake of illustration, we have briefly summarized salient findings from experimental work on the hawk moth *Manduca sexta*. In this organism, the physiological regulation of development time and body size, as well as its evolutionary implications, are most thoroughly explored. We then identify generalities in maturation pattern across diverse taxa, and discuss how the underlying processes can be addressed experimentally.

As in most organisms, growth in *M. sexta* from one larval stage to the next is exponential. This insect thus accumulates roughly 90% of adult body mass during the last (fifth) larval stage preceding metamorphosis

(D'Amico, Davidowitz & Nijhout 2001). As no further somatic growth occurs after metamorphosis, this developmental transition essentially determines development time and adult size. (Note that in this insect, time required for pupal development and gamete production decouples larval development time and age at maturity to some degree. Nevertheless, we can reasonably assume that the two traits usually correlate well.) Within the last larval stage, an individual grows roughly linearly and at some point exceeds a weight threshold termed the critical weight (CW; Fig. 3). In *M. sexta*, CW is defined as the minimal weight that allows an individual to complete metamorphosis and pupation in a normal time span without further feeding and growth (Nijhout & Williams 1974; D'Amico *et al*. 2001). Larvae that are starved before they have attained CW will either show delayed metamorphosis, or will fail to metamorphose altogether. The attainment of CW triggers an irreversible endocrine cascade that eventually culminates in ecdysteroid hormone secretion (ES; Fig. 3) and subsequent initiation of metamorphosis (Nijhout 1994). Between the attainment of CW and ES, a phase termed the interval to cessation of growth (ICG; Fig. 3), an individual continues to grow (although this is not required for metamorphosis) and may almost double its body mass.

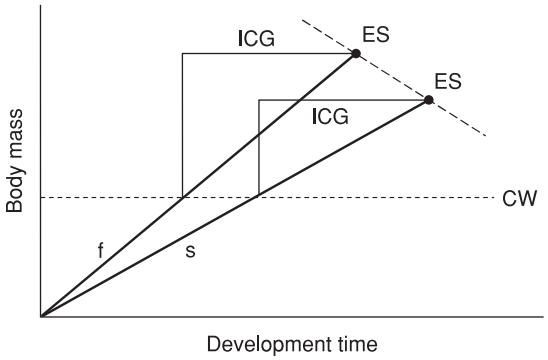

Fig. 3. Physiological determinants of development time and adult size in *Manduca sexta*. The thick lines f and s represent (linear) growth trajectories of a fast-growing and a slow-growing individual in the last (fifth) larval stage. At some point these growing juveniles attain a critical weight threshold (CW) where the secretion of juvenile hormone from the corpora allata glands is turned off. During the subsequent interval to cessation of growth (ICG), the insects continue to feed and grow while juvenile hormone is cleared from the blood. As soon as all juvenile hormone is eliminated, the larvae become competent to secrete prothoracicotropic hormone (triggered by a photoperiodic cue not illustrated), which in turn stimulates ecdysteroid secretion (ES) that causes the animals to stop feeding and prepare for metamorphosis. ES thus coincides with the fixation of the development time and adult size phenotype. The fast-growing individual (f) reaches CW at an earlier age than s, and f grows to a greater final size during ICG. In interaction with this type of ontogenetic regulation, environmental or intrinsic variation in growth rate thus results in negative covariance between development time and size among individuals within a population (dashed line). In contrast, given growth rate is relatively constant, variation in CW or ICG produces positive time–size covariance (not illustrated).

Knowledge of the environmental sensitivity of, and the amount of standing genetic variation in, these different regulatory determinants permits powerful predictions of plasticity and evolution in development time and adult size. For instance, Davidowitz & Nijhout (2004) dissected the physiological basis of the thermal reaction norm of age and size in *M. sexta*. They found that the value of CW is stable across rearing temperatures, whereas the duration of ICG decreases dramatically as temperature increases. At a high rearing temperature, which also accelerates growth rate, individuals thus reached CW earlier than animals reared at lower temperatures, and they were able to grow less during the shorter ICG. These findings explained the observed concurrent reduction in age and size at maturity with increasing environmental temperature, a general pattern in ectotherms (van der Have & de Jong 1996; Atkinson & Sibly 1997).

Furthermore, Davidowitz, Roff & Nijhout (2005) explored the response of the physiological determinants in *M. sexta* to simultaneous selection on development time and adult size. They showed that the determinant(s) that experience synergistic selection are likely to govern the evolutionary response of age and size. For example, simultaneous selection for large adult size and short development time results in a physiological conflict, and hence antagonistic selection, in both CW and ICG. An increase in the value of these components increases size, but also prolongs development time pleiotropically, and we might therefore expect relative stasis in CW and ICG. The evolution of a higher growth rate would, however, alter age and size synergistically. Overall, the experimental work on *M. sexta* demonstrates how the interplay of different ontogenetic processes can facilitate or constrain life-history evolution, and how it causes specific reaction norms in age and size at maturity. The complexity of the underlying physiology, however, cautions against simplistic adaptationist interpretations of phenotypic plasticity in these traits.

But how general are insights gained from *M. sexta* ontogeny? Do the most critical determinants of development time and size in this model system – the size threshold and the irreversible commitment to maturation after the threshold is attained – occur in other organisms? Indeed, the physiological model outlined above has general validity in insects (Nijhout 2003 and references therein) and, for example, explains negative age–size correlations in response to environmental or genetic variation in growth rate within populations (Wall & Begon 1987; Klingenberg & Spence 1997; Berner & Blanckenhorn 2006). The *M. sexta* model is also fully consistent with observations from the few crustaceans examined so far (Twombly 1996; Ebert 1997). Further, size thresholds have been shown to trigger metamorphosis in amphibians (Leips & Travis 1994; Beck 1997; Morey & Reznick 2000). Here, thyroid hormone appears to play a role analogous to ecdysteroids in insects (Denver 1997; Davey 2004). Like *M. sexta*, amphibians

reach a stage at which they become committed to metamorphosis: beyond this size threshold, development becomes relatively insensitive to environmental factors or variation in growth rate (Hensley 1993; Rose 2004), and similar findings have been reported from fish (Sohn & Crews 1977; Reznick 1990). Maturation size thresholds have also been documented for birds (Eitan & Soller 1996) and mammals (Bauer 1987; Brown-Douglas *et al*. 2004). In plants, photoperiodic and other cues are important determinants of reproductive timing. Nevertheless, a threshold weight must usually be surpassed before flowering can be induced (Lacey 1986; Wesselingh & Klinkhamer 1996; Wesselingh *et al*. 1997).

Taken together, these studies make clear that most multicellular organisms must acquire a specific amount of resources, which we observe as size or weight thresholds, before they can enter reproductive life. Moreover, the physiological processes that underlie maturation are often irreversibly triggered by the threshold itself and are relatively insensitive to environmental factors, although environmental stimuli may represent additional, essential determinants of maturation. A better knowledge of the physiological processes governing maturation, and how these processes respond to environmental factors and interact with growth rate, is therefore essential to developing a deeper understanding of the relationship between age and size at maturity, as exemplified in *M. sexta*.

METHODOLOGICAL ISSUES IN THE STUDY OF MATURATION

The ultimate goal is to understand the maturation process (and ontogeny in general) from the level of gene expression to physiology and phenotype. This, however, is a formidable endeavour requiring the collaboration of researchers across biological disciplines that rarely interface. Only in a few model organisms are molecular genetic and neuroendocrine details, as well as their links, beginning to be unravelled (e.g. *Arabidopsis*, Ungerer *et al*. 2002; *Drosophila*, Stern 2003; King-Jones *et al*. 2005). For instance, the physiological basis of size thresholds in animals (the way overall body size is assessed) remains poorly understood (Denver 1997; Nijhout 2003). However, exploring the phenomenological correlates (or symptoms) of the physiological processes underlying maturation is a fruitful and easily feasible first step towards a fuller understanding of ontogeny and the relationship between age and size.

As mentioned above, some clues to key ontogenetic processes may often be obtained by analysing growth trajectories. Figure 2, for example, indicates that growth rate did not respond to artificial selection on dung-fly body size. Instead, we might hypothesize that size divergence is attributable to changes in CW, ICG, or both (Fig. 3). A classic experimental strategy to examine the occurrence and estimate the value of critical size or weight thresholds is to manipulate the amount of resources available to growing individuals, and to track subsequent ontogeny. The specific protocol will depend on the organism and the operational definition of the threshold. In *M. sexta*, for instance, CW has been estimated by assigning individuals in their last larval stage to different weight classes (Nijhout & Williams 1974; D'Amico *et al*. 2001). Within each weight class, half the individuals were allowed to feed normally whereas the other half were starved, and individual time to ES was recorded. The CW was then determined by performing a pairwise *t*-test between the starved and non-starved group within each initial weight class; a significant difference in the time to ES indicated that the starved insects had not yet attained CW (which delays ES). In similar vein, Wesselingh *et al*. (1997) manipulated the length of the first vegetative growth period available to a biennial plant and determined individual fresh weights before exposure to winter conditions. Logistic regression revealed that the probability of reproducing during the following growing season was dependent on the attainment of a threshold weight before winter. Note that in non-clonal organisms, these approaches typically allow maturation symptoms to be examined at the family or population level only (but cf. Davidowitz, D'Amico & Nijhout 2003). Once critical weight is known, the occurrence of a switch to fixed development (commitment) can be addressed by exposing postcritical individuals to different food levels. If there is commitment to maturation, the period from the attainment of the weight threshold to reproduction will be constant across food treatments, and hence independent of growth rate (Hensley 1993).

Exploratory investigations such as those outlined above make it possible to address more specific hypotheses regarding the sensitivity of specific maturation determinants to environmental factors (de Jong, Goosen-de Roo & Klinkhamer 1998), including maternal effects such as embryonic size (Ebert 1997). Also, artificial selection experiments (Wesselingh & Klinkhamer 1996; D'Amico *et al*. 2001) or comparisons of the sexes or conspecific natural populations (Berner & Blanckenhorn 2006) allow to study the evolution of maturation determinants. To characterize directly the endocrine cascades involved in maturation, tools from experimental physiology need to be employed, such as the correlation of ontogenetic events with specific hormone levels, the artificial manipulation of hormone levels, or the removal of secretory organs. Exciting insights can also be expected from a dissection of the genetic architecture of maturation determinants using mapping of quantitative trait loci. Although this should now be feasible in at least some model systems, it has, to our knowledge, been undertaken only in *Arabidopsis* (Ungerer *et al*. 2002).

Conclusions

We have made the case that an explicit consideration of ontogenetic detail allows a deeper understanding of

the relationship between age and size at maturity, and has the potential to unify conceptually phenotypic and quantitative genetic approaches. Issues that deserve particular attention include the causes of intrinsic variation in growth rate; the physiological determinants of the maturation process; the amount of standing genetic variation in these determinants and how they respond to environmental factors; and finally, how the interaction of growth rate and maturation determinants shapes phenotypic and genetic correlations and reaction norms of age and size. The study of these aspects will benefit from comparisons of growth trajectories among individuals, sexes, populations and species, and across environments, from a phenomenological examination of maturation determinants, and ultimately from increasing collaboration among geneticists, physiologist and evolutionists.

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