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Metabolic acceleration in animal ontogeny: An evolutionary perspective

S.A.L.M. Kooijman

Department of Theoretical Biology, VU University, Amsterdam, The Netherlands

A R T I C L E I N F O

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A B S T R A C T

Acceleration of metabolism is defined as a long-term increase of respiration, that is faster than the typical trajectory during the life cycle of an individual, from embryo to adult. The Dynamic Energy Budget (DEB) model is used to quantify the typical trajectory. All DEB parameters were estimated for over 300 animal species from most large phyla and all chordate classes. The goodness of fit is generally excellent, including embryo development (embryo weight, respiration, amount of yolk). Although many species match predictions for metabolic rates, particular taxa, including all those with larval development, but also ones with less clear larval stages, deviated and have a lower metabolic rate as embryo, compared to late juvenile and adult stages: they accelerate their metabolism during the life cycle. Five different types of acceleration are identified, examples are given, and methods are presented to recognise these different types. Associated life history traits are discussed in an evolutionary and ecological context. Arguments are presented for why accelerating species have an extra slow start of metabolism and why parental care evolved in endotherms.

1. Introduction

This paper is about metabolic acceleration in animals; a recently introduced concept (Augustine, 2012; Augustine et al., 2011, 2014, 2014; Kooijman, 2013b,c; Kooijman et al., 2011; Lika et al., 2014a,b; Mueller et al., 2012; Pecquerie, 2008; Pecquerie et al., 2009) now put in a broader perspective. Before I can discuss the acceleration of metabolism, we first need to consider what metabolic rate actually is and how it is quantified in the first place (without acceleration). Metabolic rate, i.e. the rate of chemical transformations in an organism, should be defined as entropy production, but is usually quantified in terms of heat production. The reason is that the entropy balance is hard to measure and requires a full energy balance (Sousa et al., 2006). In fact we need Dynamic Energy Budget (DEB) Theory to quantify entropy of living biomass (in combination with considerable effort), since it is the only theory that is available to specify mass and energy flows during the full ontogenic cycle. The result differs considerably from biochemical methods, which have little theoretical foundation in view of spatial effects and the effects of the process of life itself (Sousa et al., 2006). The existence of (anaerobic) endothermic bacteria, which consume heat rather than produce it, shows that quantification of metabolic rate in terms of heat production has already its limitations. Yet the measurement of heat production is not that easy at all, specially for small organisms. The popular method of indirect calorimetry (Lavoisier and Laplace, 1780) assumes that heat production is a weighted sum of dioxygen consumption and carbon dioxide and nitrogen waste production, which are much easier to measure than heat production. The DEB theory provides an explanation for this in the case of animals (Kooijman, 2010), but it also shows that we cannot quantify the energy balance, before quantifying the full mass balance. Yet, in practice, even the mass balance is not measured for animals for economic reasons. Such balances can only be found in the literature for (populations of) micro-organisms. Dioxygen consumption, i.e. respiration, is frequently taken as a proxy for heat production of animals that live in aerobic environments (Kooijman, 2013a). As long as the respiration quotient, i.e. the ratio of carbon dioxide production and dioxygen consumption does not vary too much, it can be understood within the context of the DEB Theory (Kooijman, 2010) that heat production is indeed about proportional to dioxygen consumption. This requirement has to be checked and the proportionality constant calibrated for each case (Walsberg and Hoffman, 2005), or indirect calorimetry should be used. In summary, metabolic rate is in practice quantified as the use of dioxygen for aerobic organisms, but this quantification has its limitations that we can understand on the basis of the DEB Theory, as explained in more detail in the study of Kooijman (2013a).

Metabolic rate increases over the life cycle of an organism. A freshly laid egg hardly respires, but respiration rate increases sharply during the embryonic stage, and after birth, here defined as the onset of feeding, it further increases during the juvenile and adult stages till the individual is fully grown, and then decreases somewhat at old age. Several factors are responsible for this increase in metabolic rate during ontogeny, increase in body size being one of them. Many papers since the 19th century have tried to explain why metabolic rate tends to increase with body mass to the power 3/4, and whether or not it is exactly this power law, or only approximately so; log–log plotting can easily give the misleading impression of accuracy. It is generally agreed that several processes contribute to metabolic rate, such as feeding and digestion overheads (known as specific dynamic action or heat increment

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of feeding), growth and reproduction overheads, maintenance and development. All these processes are functions of body size, which already demonstrate that some power law can only be a very crude first approximation at best. Feeding and temperature affect metabolic rate substantially, as does body size (both intra- and inter-specifically). The trajectory of metabolic rate over the life cycle of an individual and the inter-species variation are well understood quantitatively by the DEB theory (Kooijman, 1986, 2001). The DEB theory makes no (direct) assumption about the use of dioxygen and obtains it by closing the mass balance for chemical elements (carbon, hydrogen, oxygen and nitrogen). The implication is that the use of dioxygen can only be evaluated in combination with the production flux of water, carbon-dioxide and nitrogen-waste (e.g. ammonia). Appendix A presents a brief description of the metabolic trajectory in absence of acceleration.

This paper is about different types of metabolic acceleration, here defined as a deviation from the expected trajectory of metabolic rate during the life cycle of an individual by the standard DEB model. Although many species match predictions for metabolic rates by the standard DEB model, particular taxa, including all those with larval development, but also the ones with less clear larval stages, deviated and have a lower metabolic rate as embryos, compared to late juvenile and adult stages: they accelerate their metabolism during the life cycle. The standard DEB model is the simplest one in the DEB family of models, with one type of food, reserve and structure and isomorphy (i.e. no change in shape during growth). This paper discusses how the various types of acceleration can be recognised, and the coupling of acceleration with other traits and evolutionary patterns. During short boosts of activity, respiration increases sharply, falling back to the original level after activity is back to standard. This is not the type of acceleration that this paper aims to discuss. Other types of temporary elevation of metabolism are sported by migratory birds and by pregnant mammals. Although these types of metabolic elevation last longer already, it is still too temporary and too much coupled to specific activities for the present discussion. The paper focusses on much longer periods of increase of metabolic rate, faster than expected, and the rate also does not fall back later to lower levels and is not coupled to specific activities. The change is more permanent and combines a slow embryo development, with a faster late juvenile and adult development. What is here called type $\mathcal{M}$ acceleration was originally discovered in some fish species (Kooijman et al., 2011), but a systematic analysis of metabolic and life history data with the standard DEB model for a large collection of species from the various corners of animal kingdom, revealed that the phenomenon is much more general. Since metabolic acceleration is defined as a deviation from a typical metabolic trajectory, only some taxa have metabolic acceleration, most taxa don't have it. Some 380 species from a most larger phyla (of more than 500 species) and all 13 chordate classes have now been studied and we never found any metabolic retardation, i.e. metabolic trajectories that start faster than expected and later slow down.

The next sections give a description of five different types of acceleration. Appendix A presents a summarising table with these types of acceleration and a summary of diagnostic features. The first type concerns a change in allocation of resources, the other four an (extra) increase in assimilation (and feeding) during ontogeny. All examples with figures are from the add_my_pet collection (//www.bio.vu.nl/thb/deb/deblab/add_my_pet/Species.html). Each entry in the collection presents all data, parameter values, code that has been used to obtain these values from the data, goodness of fit quantifiers, a quantifier for the completeness of the data, and model predictions. Since the parameter estimation procedure uses all available data on the various aspects of energy budgets simultaneously, these entries can hardly be summarised in this paper.

2. Type *R* acceleration: change in allocation

An illustrative and remarkable form of acceleration has been observed in the Australian myobatrachid *Crinia georgiana*, compared to *Pseudophryne bibronii* (Mueller et al., 2012), see Fig. 1. *Crinia* sports developmental acceleration, *Pseudophryne* does not. Their maximum body weights are similar, but *Crinia* has larger eggs. They both have a free-swimming tadpole stage, but we also found type *R*-acceleration as well as no-acceleration in frog species with direct development.

When *Crinia* hatches, before it starts feeding, and the water table in their pond is low, it decreases allocation fraction κ steadily till birth. Between birth and metamorphosis κ remains constant at a low level, and is reset after metamorphosis. Acceleration type *R* stands for maturation. This has several coordinated effects: respiration is increased, growth is decreased and maturation is increased. In view of growth overheads, a decrease of growth might be expected to come with a decrease in respiration. But that does not happen: all reserve that embryos and juveniles allocate to the $(1 - \kappa)$ branch of mobilised reserve is spent on maturity maintenance and maturation, and eventually ends up as carbon dioxide, water and N-waste, which comes with the use of dioxygen.

This directly follows from the conservation law for chemical elements, given that reserve is a mixture of mainly carbohydrates, protein and lipids. The ecological significance is that they reach metamorphosis much earlier and smaller, allowing them to leave their pond before it dried out. It seems that the rate at which reserve is mobilised, which depends on the amounts of reserve and structure, is not changed. That is, the absolute mobilised flux steadily increases, and the one relative to structure steadily decreases. Mobilisation also does not change during the late embryo stage when type *R* acceleration occurs. So respiration increases, but mobilisation does not. Since size at metamorphosis is reduced substantially, size at puberty might also be reduced somewhat, as does allocation to reproduction directly after puberty. These effects are expected to disappear eventually, but data are lacking.

Acceleration of maturation is remarkable, because it demonstrates a trait-off between growth and maturation. I see the fact that the coordinated changes in respiration, growth and maturation are well captured as a strong support for the organisational position of maturation in the standard DEB model. Type R-acceleration is achieved by changing κ, while it typically remains constant during the full life cycle (Lika and Kooijman, 2011). The fact that it occurs in direct as well as indirectly developing frogs shows that the mode of development has nothing to do with the acceleration of maturation.

3. Acceleration of assimilation

The acceleration of maturation results from a shift in allocation, not from an increase in intake. Acceleration of metabolism, as discussed in this section, involves an increase in intake, stronger than being proportional to squared (structural) length. I discuss 4 different types in a particular sequence, involving more and more components of the energy budget.

3.1. Type X acceleration: change in food quality

A rather frequently occurring form of acceleration is an increase in feeding rate combined with shifts in food preference. The feeding capacity just increases with squared structural length, but the amount of food and/or the food quality increases faster for some period and remains higher. Acceleration type *X* stands for ‘food’, and differs from other acceleration types because the parameters of the individual remain constant. The individual as a dynamic system does not change, only the interaction with the environment (food).

Neonates need high-quality food to cover their relatively high growth needs, compared to later stages that mainly need to cover maintenance needs. Fed with the same type of food, neonates do less well and can sport retarded growth. Taxa like fish are born tiny and change in food preference to bigger prey while growing. Not only because they are bigger themselves, but they also can swim faster. This can sometimes lead to an extra increase in food intake.