Ecological Modelling 322 (2016) 48–53

Contents lists available at ScienceDirect

Ecological Modelling

journal homepage: www.elsevier.com/locate/ecolmodel

The metabolic economics of environmental adaptation

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ARTICLE INFO

Article history:
Received 4 September 2015
Received in revised form
25 November 2015
Accepted 27 November 2015

Keywords:
Biocognition
Data Rate Theorem
Emergence
Local extinction
Rate Distortion Theorem
Selection pressure

ABSTRACT

Applying biological versions of the Data Rate Theorem and the Arrhenius reaction rate relation, it becomes clear that the search-and-response feedback of developmental selection associated with phenotypic plasticity requires a significant rate of metabolic free energy. Too rapid change in environmental conditions, often coupled with decline in available sources of metabolic free energy, leads to highly punctuated local extinction events. The observed dynamic is likely to be that the animal seems to adapt to environmental alterations for a long time, but then, and quite suddenly, developmental selection fails, leading to local extirpation of the reproducing population. Conversely, mosaicking, by imposing selection demands associated with diversity in time, space, and mode – as in traditional and conservation agricultures – can create energy barriers limiting the evolution and spread of pest or pathogen populations.

1. Introduction

Snell-Rood (2012) defines developmental selection as a developmental process that involves the sampling of a range of phenotypes and feedback from the environment reinforcing high-performing phenotypes, particularly in the context of rapid environmental change. This perspective is consistent with, and much in the style and direction of, that of West-Eberhard (2003). While developmental selection has clear benefits in the context of rapidly changing environments, it carries a variety of costs, many of which express themselves as high metabolic demands across the life course of an organism.

As Snell-Rood (2012) puts the matter, while developmental selection may increase performance of individuals in the local environment, the process of sampling phenotypes and receiving environmental feedback comes with costs of time, energy, and risk of predation. Developmental selection requires energy and material in order to physically express a range of different phenotypes. In the case of behavioral plasticity, there is a material cost in the form of more neurons and more neural connections required to sample a range of behaviors. This is significant because neural tissue is some of the most metabolically expensive tissue in the body. Across other components of organismal development, exploration should result in energy costs associated with construction and atrophying diverse phenotypes. The costs of developmental selection should result in major changes in allocation of energy, resulting in changes to life history strategies.

Snell-Rood (2012) goes on to identify possible subtleties in these processes. For example, if the population shifts into a novel constant environment, the costs of developmental selection will select for loss of plasticity, or genetic assimilation. If environmental variation remains, there may be selection to reduce environmental variation (and subsequently plasticity), for instance through habitat or resource selection. In other words, animals may niche-construct their environment in ways that make it less variable. Niche construction is usually a separate and massive sink for metabolic free energy.

Here, we first adapt a control theory perspective to the sampling-and-feedback mechanisms inherent to these constructs, in particular invoking the Rate Distortion Theorem and the homology between information and free energy so elegantly expressed by Bennett (1988), as summarized by Feynman (2000). It becomes possible to explore minimal levels of metabolic free energy required for stability in developmental selection using a biological version of the recently developed Data Rate Theorem that, after a half-century of work, formally links information and control theories (Nair et al., 2007). Environmental demands beyond such limits may express themselves in punctuated extinction events. Conversely, systematic reduction in metabolic demand – one form of ecological smoothing – may express itself in punctuated emergence, for example blooms of pests or pathogens (e.g., Wallace et al., 2014; Wallace and Wallace, 2014).

A second line of argument adapts the Arrhenius reaction rate relation: the rate of metabolic free energy supply is taken as a

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http://dx.doi.org/10.1016/j.ecolmodel.2015.11.017
0304-3800/

temperature analog in driving the biological cognition associated with developmental selection and phenotypic plasticity. Too slow an adaptation rate, of course, would lead to extirpation of local populations under rapidly changing environmental burdens.

Biological regulation, like selective processes in development, can be viewed as involving the transmission of control signals to physiological systems at a variety of scales and levels of organization (Wallace, 2014; Wallace and Wallace, 2010). Gene expression, in the context of environmental and developmental signals, is very much a cognitive process in the sense of Atlan and Cohen (1998) (Wallace, 2012, 2014; Wallace and Wallace, 2014). That is, given a fixed 'instrument' of the organism's limited set of genes, regulatory systems must, at each developmental branch point (West-Eberhard, 2003) chose to 'play' a single possible outcome out of a much larger set of possible outcomes. Such choice requires reduction of uncertainty, in a particular formal sense, and this implies existence of an information source 'dual' to the cognitive process. Again, see Wallace (2012, 2014) and Wallace and Wallace (2008, 2009, 2010) for more complete explication, elaboration and application of arguments regarding the cognitive nature of gene expression.

Several obvious questions arise:

(1) How well is the control signal obeyed?
(2) What is the rate of metabolic free energy (RMFE) necessary to impose an adequate level of control?
(3) What is the RMFE needed to impose a particular rate of response to incoming signals?

Answering these questions requires some formal development.

2. A first approach

The Rate Distortion Theorem, described more fully in the Mathematical Appendix, states that, for a communication or control channel, there is a function, $R(D)$, that is the minimum necessary rate of information transmission that ensures communication does not exceed an average distortion D, by some appropriate measure. Thus $R(D)$ defines a minimum necessary channel capacity. Cover and Thomas (2006) provide details. The rate distortion function has been calculated for a number of systems, using Lagrange multiplier or Khun–Tucker optimization methods.

Cover and Thomas (2006, Lemma 13.4.1) show that $R(D)$ is necessarily a decreasing convex function of D: $R(D)$ is always a reverse J-shaped curve. Convexity is an exceedingly powerful mathematical condition, and permits deep inference. We will use the Gaussian channel as an easily calculated example, but the arguments are quite general.

For the standard Gaussian channel having white noise with zero mean and variance σ^2, using the squared distortion measure (Cover and Thomas, 2006),

$$R(D) = \frac{1}{2}\log\left[\frac{\sigma^2}{D}\right], \quad 0 \le D \le \sigma^2$$
$$R(D) = 0, \quad D > \sigma^2 \tag{1}$$

Feynman (2000) argues that there is a deep homology between information source uncertainty and free energy density. Following Bennett's (1988) arguments, he constructs a simple ideal machine that can turn the information of a message to useful work – a surprisingly elementary result. Thus information should be viewed as another kind of free energy, and the construction and transmission of information within living things consumes metabolic free energy, with necessarily massive losses as a consequence of the second law of thermodynamics. If there are limits on available metabolic free energy, there will necessarily be limits on the ability of living things to process information, and, in particular, for essential regulatory mechanisms to maintain or change the living state. $R(D)$ can be interpreted as a free energy rate measure.

Let metabolic free energy be available at a rate M. Let $R(D)$ be the Rate Distortion Function describing the relation between a critical regulatory system intent and the real effect on the regulated system. To reiterate, this is a channel capacity index. We assume, in first approximation, that the rate of metabolic free energy needed to sustain a biological control channel having a Rate Distortion Function $R(D)$ is

$$M \approx \kappa_1 R(D) + \kappa_2 \tag{2}$$

where the κ_1 may be quite large as a consequence of Second Law losses in the translation of metabolic energy into information energy.

The Mathematical Appendix uses a classic Black–Scholes cost analysis to derive this result as the outcome of an exactly solvable approximate model, with the serious limitations that implies (e.g., Pielou, 1977, pp. 107–110).

Van den Broeck et al. (1994, 1997), Horsthemeke and Lefever (2006), and many others, have noted that the relation of phase transition to driving parameters in physical systems can be obtained by using the rich stability criteria of stochastic differential equations. We apply similar arguments, deriving a 'biological' form of the important Data Rate Theorem relating control and information theories that is described in more detail below.

The motivation derives from the observation that a Gaussian channel with white noise variance σ^2 and zero mean has the Rate Distortion Function described by Eq. (1). Define a 'Rate Distortion entropy' as the Legendre transform

$$S_R = R(D) - \frac{DdR(D)}{dD} = \frac{1}{2}\log\left[\frac{\sigma^2}{D}\right] + \frac{1}{2} \tag{3}$$

Then the simplest 'nonequilibrium Onsager equation' describing system dynamics (de Groot and Mazur, 1984) is

$$\frac{dD}{dt} = \frac{-\mu dS_R}{dD} = \frac{\mu}{2D} \tag{4}$$

Here, t is the time and μ is a diffusion coefficient. This has as the simplest solution $D(t) = \sqrt{\mu t}$, precisely the classic result for the ordinary diffusion equation. Such correspondence reduction allows argument upward in both scale and complexity. That is, regulation does not involve a diffusive drift of some average distortion. Quite the contrary.

Taking M again as the rate of available metabolic free energy, a plausible model – in the presence of white system noise having variance β^2 in addition to the environmental channel noise variance σ^2 – is the stochastic differential equation

$$dD_t = \left(\frac{\mu}{2D_t} - F(M)\right)dt + \frac{\beta^2}{2}D_t dW_t \tag{5}$$

dW_t represents white noise and $F(M) \ge 0$ is a monotonically increasing function that will be explicitly derived in subsequent calculations.

Eq. (5) has the nonequilibrium steady state (nss) expectation

$$D_{nss} = \frac{\mu}{2F(M)} \tag{6}$$

where D_{nss} is the nonequilibrium steady state value of the average distortion.

Next, applying the Ito chain rule to Eq. (5) (Protter, 1990; Khasminskii, 2012), it becomes possible to calculate the variance in the distortion as $E(D_t^2) - (E(D_t))^2$.