12 Group Transformation
Life History Trade-offs, Division of Labor, and Evolutionary Transitions in Individuality

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12.1 INTRODUCTION

Division of labor is an old concept. One can find the basic idea in the Republic of Plato:

> Things are produced more plentifully and easily and of better quality when one man does one thing which is natural to him and does it in the right way, and leaves other things.

Ever since Plato, numerous theorists have proposed variations on this theme with different degrees of sophistication. In *The Wealth of Nations*, Adam Smith (1776) tells us that 10 men in a pin factory can produce approximately 48,000 pins in a single day, whereas he estimated that they would only produce less than 20 each individually or even none if 10 untrained men were performing all the 18 necessary steps to produce a pin on their own. This is because each man is *specialized* in one or two steps of the pin-producing process and, thus, performs the steps more efficiently without the need to switch between tasks than a man performing all the steps sequentially. Without this difference in efficiency and task-switching, there would be no advantage for a man to become a specialist because it only suffices that a single one of the 18 "types" of men is unavailable for no pins to be produced at all. However, in conditions where an individual can be confident in finding other men with each of the 17 other specializations (or with the ability to switch from one to another), it becomes advantageous to specialize in one of the steps for producing pins. This example illustrates the point that a division of labor entails "trade-offs." First, for dividing labor to pay off, an individual performing all the steps must be unable to produce an outcome with the same efficiency and at the same time as a specialist. Second, in situations where the men's interactions are limited or the number of men is too low, becoming a specialist must lead to a worse outcome than being a generalist. The idea of division of labor, like several other concepts in economics, has made its way to biological theory. Biological entities at all levels of organization exhibit division of labor, resulting in various degrees of specialization. However, in contrast to economic theory, division of labor is posited in evolutionary theory as an outcome of natural selection rather than rational decision.

One fundamental trade-off faced by all biological entities is the investment in maintenance (e.g., escaping predators, foraging, repairing damage) and reproduction (e.g., investing in gametes, finding a mate). It represents a particular kind of division—namely, *reproductive* division of labor. Multicellular organisms present intriguing examples of reproductive division of labor and a high degree of cellular differentiation. A widely accepted theory even suggests that germ-soma specialization has been key in the evolutionary transition from cellular groups to multicellular individuals (Buss, 1987; Simpson, 2012). Such transitions, where multiple preexisting entities form a new level of organization, are examples of *major evolutionary transitions* (Maynard Smith & Szathmáry, 1995) or *evolutionary transitions in individuality* (ETIs) (Buss, 1987; Michod, 2000). This chapter focuses on a theoretical model addressing the role of a trade-off between life history traits in selecting for a

reproductive division of labor during the transition from unicellular to multicellular organisms (Michod, 2005; 2007; Michod et al., 2006; Michod & Herron, 2006), hereafter referred to as the life history trade-off model for the emergence of division of labor, or "life history model" (*LHM*) (Chapter 3).

The *LHM* has been inspired by the volvocine green algae, a taxonomic group where contemporary species range from unicellular over simple multicellular to fully differentiated (Kirk, 1998). These phototrophic eukaryotes use flagella to remain in the photic zone of freshwater environments where photosynthesis is possible. The best studied unicellular representative of this group is *Chlamydomonas reinhardtii*, which can be observed to possess its two flagella only for parts of its life cycle, during the growth phase. For cell division (i.e., reproduction), the flagella must be absorbed as cells face the functional constraint of simultaneous cell division and flagellation. This constraint necessitates a fundamental trade-off between swimming and cell division (Koufopanou, 1994).

The constraint that simultaneously bears upon viability (i.e., swimming) and reproduction has been "solved" in the closely related multicellular species *Volvox carteri*, where these incompatible functions are segregated into two different cell types. Its spherical colonies move around in the water column due to approximately 2,000 cells that look very similar to the cells of *C. reinhardtii* in that they each possess two flagella. Crucially, these cells never lose their flagella and cannot divide—they are the irreversibly differentiated soma. Reproduction is carried out by a few germ cells, called gonidia, which do not possess flagella but do possess the ability to divide. In contrast to the uncellular *C. reinhardtii*, for which these two functions are separated temporally during its life cycle, the multicellular *V. carteri* displays a spatial rather than temporal separation of somatic and reproductive functions with two cell types, which is characteristic of a reproductive division of labor.

This example of the origin of the division of labor in the volvocine green algae illustrated here is not unique. In fact, the need to accommodate two incompatible processes is also thought to drive the origin of the reproductive division of labor in other multicellular groups—for example, in metazoans, the incompatibility between cell division and flagellation (King, 2004) and, in cyanobacteria, the incompatibility between fixation of atmospheric N_2 and photosynthesis (Rossetti et al., 2010; Hammerschmidt et al., 2021).

The *LHM* relies on five key assumptions concerning the relationship between fitness and life history traits (i.e., viability and fecundity) of cells and collectives: (1) fitness is the product of viability and fecundity; (2) collective traits are linear functions (sum or average) of their cell counterparts; (3) there is a trade-off between a cell's viability and its fecundity; (4) cell traits are optimal in the sense that they display the traits that ensure the highest contribution to collective fitness; and (5) the viability–fecundity trade-off is convex for large collectives due to the initial cost of reproduction. Assumptions 1–2 are summarized in Figure 12.1, Assumptions 3–4 in Figure 12.3, and Assumption 5 in Figure 12.4. The definitions of the symbols used are presented in Table 12.1; the assumptions are summarized in Table 12.2.

In this chapter, we pursue two aims. First, we provide a step-by-step guide to these assumptions for the reader to build an intuitive understanding of the *LHM*. In doing so, we highlight some strengths and limits of the model and provide directions

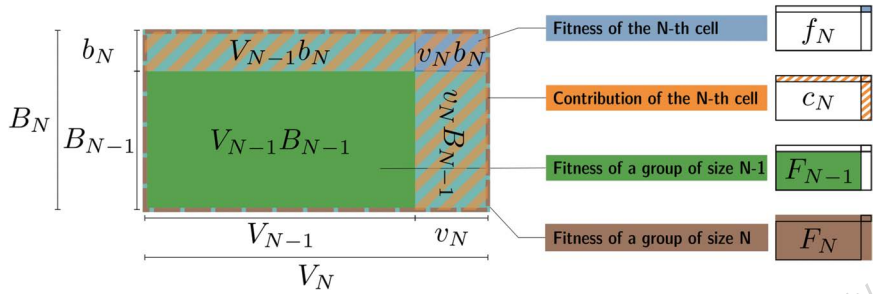

FIGURE 12.1 Geometric representation of collective fitness. As a consequence of Assumption 1 (i.e., fitness is the product of fecundity and viability) and Assumption 2 (i.e., collective traits are linear functions of their cell counterparts), it is possible to represent the fitness of a collective geometrically—as the area of a rectangle whose sides are its fecundity and viability—and to decompose it into the contribution of its constituent cells. Symbols are defined in Table 12.1.

to explore. Second, we present two interpretations of the *LHM* in the context of ETIs, with a particular focus on the metaphorical notion of "fitness transfer" and its limitations. Throughout, we illustrate our points with biological examples.

12.2 A STEP-BY-STEP GUIDE TO THE LIFE HISTORY MODEL OF DIVISION OF LABOR

12.2.1 Fitness is Viability Times Fecundity

Assumption 1 of the *LHM* is that the value of two life history traits characterizes any entity (e.g., a cell or a collective): their viability (v for cells and V for collectives, which measures their propensity to survive) and their fecundity (b and B,

TABLE 12.1
Definitions of the Symbols

Description	Symbol	Formula
Effort of the i-th cell toward viability	e_i	(parameter)
Cell viability, cell fecundity (of the i-th cell)	v_i, b_i	$v_i := v(e_i)$; $b_i := b(e_i)$ v and b are an increasing and decreasing function of e_i, respectively.
Cell fitness (of the i-th cell)	f_i	$f_i := v_i b_i$
Average cell fitness (in a collective of size N)	$\overline{f}$	$\overline{f} := N^{-1}\sum_{i=1}^{N} f_i$
Collective fitness (of a collective of size N)	F_N	$F_N := V_N B_N$
Viability, fecundity of a collective of size N	V_N, B_N	$V_N := \alpha \sum_{i=1}^{N} v_i$; $B_N := \alpha \sum_{i=1}^{N} b_i$
Contribution of the i-th cell to collective fitness	c_i	$c_i := b_i V_{i-1} + B_{i-1} v_i + b_i v_i$

TABLE 12.2
Summary of Modeling Assumptions 1–5

1	Fitness is viability times fecundity.
2	Collective traits are linear functions of their cell counterparts.
3	There is a trade-off between cell viability and fecundity.
4	Cell contribution to the collective is optimal.
5	There is an initial reproductive cost in large collectives.

which measures their propensity to reproduce). Fitness can be defined as the product of those two components ($f = vb$ and $F = VB$). The effect of fitness components[1] on the evolutionary success of organisms lies at the center of life history theory (Stearns, 1992), notably through the study of the constraints that link them together (see Stearns, 1989; Assumption 3). Other components of fitness exist in life history theory; however, the *LHM* focuses solely on viability and fecundity.

Taking the viability and fecundity product to compute fitness is a common assumption in the literature (Sober, 2001). There are at least two ways to justify this choice: phenomenologically and mechanistically. First, the product of two quantities *phenomenologically* characterizes the way these two components interact in the context of fitness. One can visualize fitness geometrically as the area of a rectangle whose sides' lengths are v and b (Figure 12.1). This representation helps to illustrate why 1) to be maximal, a multiplicative function requires a "strong balance" (Michod et al., 2006) between the two components; and 2) if one of the two sides is smaller, the marginal benefit (the surface gain) of increasing the other side is also relatively small. Additionally, if either side (fecundity or viability) has length zero, the area (fitness) is nil (we return to this point below).

Second, the product between two terms measuring fecundity and viability also arises naturally in various *mechanistic* models of population dynamics. As an example, consider a simple deterministic two-stage model with newborns and fertile adults that all share the same traits. Further, consider that whether proportion v of individuals reach the reproductive stage is given by their viability v ($0 < v < 1$), and that all adults leave a number of offspring equal to their fecundity ($b > 0$). It follows naturally that, on average, an individual will have vb offspring and the population size will grow geometrically with ratio vb in each generation (provided that generations do not overlap). This growth rate vb is also called the *Malthusian parameter* of the population and is commonly identified as a fitness measure (Fisher, 1930, p. 22). However, in a real biological situation, there is typically no fixed proportion of the population dying at each generation, and individuals leave a varying number of offspring. Despite this, the product fecundity–viability (or equivalent ratio of fecundity to mortality) is not just a feature of simple models. It also appears in more complex, stochastic models, where those fluctuations are taken into account (Kot, 2001; Haccou et al., 2007).

In the *LHM*, any relevant entity is characterized by fitness, which is broken down into its components. To study the two-level system of cells and collectives of interest for the evolution of multicellularity, one must describe how those two levels relate to each other. This is the purpose of Assumption 2.

12.2.2 Collective Viability and Fecundity Are a Linear Function of Cell Viability and Fecundity

Assumption 2 of the *LHM*, which is perhaps the most controversial, is that a collective's viability and fecundity are considered proportional to the sum or average of its component cells' viability and fecundity, respectively. In other words, the relationship between cell and collective fitness components is considered linear. Thus, a collective composed of N cells indexed $1, 2 \ldots N$ will have the viability $V_N = \alpha \sum_{i=1}^{N} v_i$ and the fecundity $B_N = \alpha \sum_{i=1}^{N} b_i$, where α is a coefficient of proportionality. If we assume $\alpha = 1$, the collective trait is the sum of the individual traits (Michod et al., 2006). If $\alpha = N^{-1}$, the collective trait is the average individual trait (Michod, 2006). The value of the coefficient is a matter of simplifying expressions and is irrelevant for most results unless comparing collectives of different sizes. For ease of presentation throughout the rest of the chapter, let $\alpha = 1$. The assumption of linearity is of great significance in the construction of the *LHM* because it qualifies the relationship of traits (and, thus, fitness components) between the lower level (cells) and the higher level (collectives). Therefore, it permits the unambiguous definition of the *contribution* of the N-th cell to collective fitness (c_N).

Assuming $F_N = F_{N-1} + c_N$, we have:

$$c_N := b_N V_{N-1} + B_{N-1} v_N + b_N v_N, \tag{12.1}$$

where F_{N-1}, V_{N-1}, and B_{N-1} respectively refer to the fitness, viability, and fecundity of a collective composed only of the cells 1 to $N-1$. We can see that c_N is the sum of three terms. The first term on the right-hand side is the effect of the focal cell's fecundity in the context of the remainder of the collective's viability ($b_N V_{N-1}$). The second term is the effect of the focal cell's viability in the context of the remainder of the collective's fecundity ($B_{N-1} v_N$). Finally, the last term is the N-th cell's fitness ($b_N v_N = f_N$). These three terms can be visualized as the sum of the three blue rectangles in Figure 12.1, and the contribution as the hatched orange area.

It follows from Assumption 2 that the only way a cell can affect collective viability and fecundity (and, ultimately, fitness) is by its own viability and fecundity. Thus, the indexing order and relative position of cells are irrelevant in this model. Further, since the cells' indexing is purely formal, any cell's contribution to the collective can be computed in the same fashion. However, a cell's contribution is not limited to its own fitness (the third term on the right-hand side of Equation 12.1). This is because it depends on the traits carried by the remainder of the collective (the first and second terms in Equation 12.1). Thus, the contribution of a cell can be higher if one of its components "compensates" for the weakness of the other component at the collective level (or, more accurately, the $N-1$ other cells). As previously, this can be visualized as in Figure 12.1. The same v quantity leads to a larger area with the orange dotted border (the cell contribution) if B is large and V is small, compared to a large V and a small B.

One consequence of Assumption 2 is that a cell with nil fitness ($vb = 0$) does not necessarily make a nil contribution toward collective fitness. For instance, consider a cell with zero viability and fecundity of one. In this situation, only the last two

terms of Equation 12.1 are nil. This result might appear puzzling at first, particularly considering that cells with nil fitness might never exist or subsist in the population (provided further implicit but standard assumptions regarding population dynamics) (Godfrey-Smith 2011, Bourrat 2015b). However, it means that, even if a cell with nil fitness (or which tends toward zero) would quickly die, its contribution to collective fitness does not necessarily tend toward zero.

A cell's viability/fecundity contribution to collective fitness can be visualized by drawing isolines of fitness in the trait-space (see orange lines in Figure 12.3). An isoline of fitness is a curve in the space v, b that corresponds to a fixed value of collective fitness. An isoline can be thought of as the contour lines of a map. This allows us to visualize the potential contribution of any cell (i.e., any pair v, b) to an already existing collective. Note that the isolines are convex (see Box 12.1) and, provided that traits of the other cells are "balanced," form a "hill" with its crest following the first diagonal, when the two traits are balanced ($v = b$), and a valley close to the two axes when one of the traits is close to zero. The minimum contribution is the point (0,0) where it is null and, thus, $F_N = F_{N-1}$.

Another way to visualize how cells with low fitness can "compensate" for one another and yield a high collective fitness is through what has been named the *group covariance effect* (Michod, 2006). Rewriting the terms of the definition of collective fitness (Table 12.1) shows the relationship between F_N and the average cell fitness ($\overline{f} := N^{-1}\sum_{i=1}^{N} f_i = N^{-1}\sum_{i=1}^{N} v_i b_i$):

$$F_N = N^2\left[\overline{f} - cov\left(\boldsymbol{v},\boldsymbol{b}\right)\right] \tag{12.2}$$

Equation 12.2 shows that collective fitness is not simply proportional to the average of cell fitnesses $\overline{f}$, but that there is a corrective term due to the interplay of cells that can be identified as the sample covariance between the fecundity and viability of the N cells, which is defined as:

$$cov(\boldsymbol{v},\boldsymbol{b}) := \frac{1}{N}\sum_{i=1}^{N}\left(v_i - \overline{v}\right)\left(b_i - \overline{b}\right) = \overline{vb} - \overline{v}\times\overline{b},$$

with $\overline{v} := N^{-1}\sum_{i=1}^{N} v_i$, $\overline{b} := N^{-1}\sum_{i=1}^{N} b_i$ and $\overline{vb} := \overline{f}$ and noting that $\overline{v}\times\overline{b} = N^{-2}F_N$.

Equation 12.2 shows that, when covariance is nil, such as when all cells are phenotypically indistinguishable (Figure 12.2a) or have independent trait values, collective fitness is directly proportional to the sum of its constituent cells' fitnesses. However, when cell fecundity and viability are not independent of one another, covariance is not nil—it is either positive or negative. If it is positive (Figure 12.2b), cells with a high v also have a high b, resulting in what we call "all-or-nothing cells." The opposite is true if it is negative (Figure 12.2c), resulting in specialized germ or soma-like cells.

Cell–cell interactions compensate for cell heterogeneities only when the covariance is negative. This can be seen by tallying the relative weight of "individual effects" of the cells on collective fitness (i.e., the direct sum of cell fitnesses, in color in Figure 12.2) and "interaction effects" due to the cross product between cell traits (the rest, in white in Figure 12.2). When this is done, it becomes apparent that

BOX 12.1 TRADE-OFF CONVEXITY

A trade-off is a relationship linking two quantities that cannot simultaneously be maximal; often, if one increases, the other must decrease. In this chapter, those two quantities are the life history traits of an individual (viability v and fecundity b).

This relationship can be due to a variety of phenomena. A trade-off between size and nutrient intake might result from physical laws (e.g., diffusion), or the trade-off may arise from the resource allocation of an organism (with a given quantity of nutrient, only so many molecules might be synthesized, creating a natural trade-off between structural molecules, housekeeping, and reproductive machinery). Trade-offs may also be caused by the underlying genetic structure of the organism (e.g., a single regulator molecule acting on two pathways, making regulation of one and the other correlated, or the functional constraint of simultaneous cell division and flagellation in the case of *C. reinhardtii*), or through interaction with other species (the expression of a useful transporter might render the cell vulnerable to a certain type of virus). Consequently, trade-offs themselves might change during the evolutionary history of organisms.

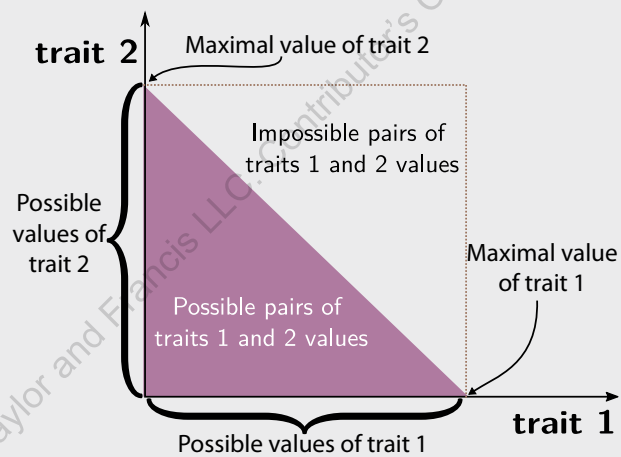

FIGURE 12.B1 Example of a trade-off between trait 1 and trait 2 with two degrees of freedom, represented by the purple surface.

This box gives a short introduction to the simple, deterministic one-dimensional trade-offs used in the *LHM* for the division of labor in multicellularity. Additional resources can be found in life history theory textbooks, such as Flatt and Heyland (2012). Two-dimensional trade-offs are represented conveniently by placing the two measures on the axis of a plane and shading the area of pairs of values that are possible within the confine of the trade-off (Figure 12.B1). This may result in a surface (two degrees of freedom) or a curve (one degree of freedom) depending on the number of free dimensions the trade-off allows.

Such a trade-off provides a straightforward definition of a "specialist" organism (with a maximal or close to the maximal value in a trait and, accordingly, a lower value for the other trait) and a "generalist" organism (with an intermediate value in both traits).

A particularly useful graphical way of analyzing a trade-off is to consider its position with respect to any segment defined by any two couple of trait values. The trade-off curve (or the edge of the surface) might coincide (Figure 12.B2.a), go below (Figure 12.B2.b), above (Figure 12.B2.c), or cross (Figure 12.B2.d) these segments.

When the curve coincides with all segments, the trade-off is said to be linear. In this case, the relationship between the two traits is proportional, reducing the value of trait 1 by a quantity x, and increasing the value of trait 2 by ax (where a depends on the slope of the curve and may depend on the scaling of the trait values). When the curve is below all segments, the trade-off is said to be convex. A small reduction in trait 1 has a different effect on trait 2 if the trait is close to the maximum value (small effect when compared to the linear) or the minimum value (large effect). Similarly, if the trade-off is above all segments, it is said to be concave. In this case, a small reduction in an optimal trait has a large effect on the other trait, whereas a small increase in a low trait has a small effect on the other trait. If the curve is above some segments and is below or crosses others, it is neither convex nor concave but can be studied in part by focusing on the different regions.

Intuitively, if the trade-off is convex, being a specialist (i.e., being on either axis) is the only way to reach high trait values, while being a generalist is "costly" in the sense that it entails a large reduction in trait value. This is reversed if the trade-off is concave—generalists enjoy a less pronounced reduction of their trait values with respect to specialists (being a specialist can be considered "costly" in the sense that the marginal cost of increasing a high value trait is relatively high compared to the case of a convex trade-off).

However, note that the convexity (or even the shape) of a trade-off does not make a prediction about the outcome of the evolutionary process on its own. It simply delimits the set of possible organisms. To be able to predict the outcome of the evolutionary process from such a trade-off, one must make additional assumptions. For instance, one could assume, as we do in the LHM,

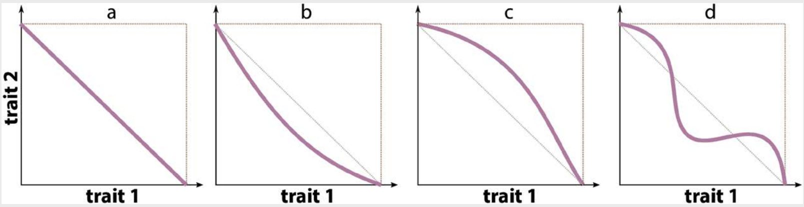

FIGURE 12.B2 a) Linear, b) convex, c) concave, d) composed trade-off.

that a fitness function F of the two traits exists and that the evolutionary dynamics reached an equilibrium state in which only the organisms with the highest fitness F are represented in the population. (In this case, one must determine the value within the set of possible individuals that gives the highest fitness). If density-dependent interactions are suspected to play a role, one possibility may be to define the invasion fitness of a rare mutant in a resident population for all pairs of points in the trade-off and look for evolutionary stable strategies, following the adaptive dynamics method (Geritz et al., 1998).

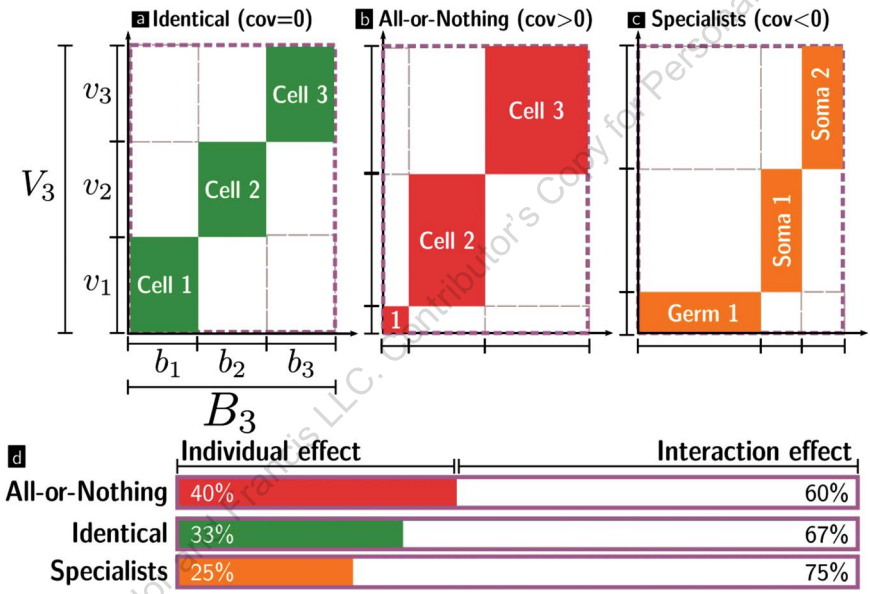

FIGURE 12.2 The covariance effect quantifies the extent to which collective fitness depends on intrinsic and interaction effects between cells. Three collectives composed of three cells are represented in the viability–fecundity space. The traits of each cell v, b are represented in color, and their fitness f is the area of a colored rectangle (green, red, orange). The three collectives have the same fitness $F_3 = V_3B_3$, represented by the area of the purple rectangle and the same average cell fitness $\overline{f}$. The collective fitness F_3 is the sum of the cell fitnesses (colored tiles, f) and of the interaction effects between cells (in white). **a)** a collective composed of three identical cells with equal traits (null covariance between v and b). **b)** a collective composed of "all-or-nothing" cells that would simultaneously have a high (low) viability and fecundity (positive covariance) (note that these cells are conceptual constructs; they are not biologically plausible). **c)** a collective composed of specialist cells with high fecundity and low viability (germ) or low fecundity and high viability (soma). **d)** the sum of cell fitnesses (colored area, labeled "individual effect") represents a larger fraction of the collective fitness (purple delimited area) when cells do not compensate for one another's weaknesses (all-or-nothing cells) as compared to when they do (specialists).

individual effects are relatively more important when the covariance is positive and relatively less important when the covariance is negative (Figure 12.2d). The converse is true for the interaction effects. Interaction effects are important because they explain how a collective can have high fitness, even if the fitness of its constituent cells is constrained to be low.

As stated earlier, Assumption 2 characterizes the relationship between cell and collective fitness in a more subtle way than simply taking the average. It also permits studying the combined effects of any set of cells (characterized by viability–fecundity pairs), as well as teasing apart a cell's direct contribution and its interactions with other cells in the collective by using the cell contributions (Equation 12.1) and the covariance effect (Equation 12.2). However, Assumption 2 is quite strong and, thus, comes at a steep price. In particular, it limits the range of phenomena that can be described satisfactorily by the model. As discussed below, biologically plausible scenarios of nonlinear and non-monotonic or, in general, higher-order interactions are impossible to describe within this framework due to this assumption. This limitation should be kept in mind by experimentalists and modelers alike.

A further limitation of Assumption 2 stems from the fact that it implies a monotonic relationship between cell and collective fitness. Thus, increasing the fecundity of one or all the cells of a collective of a given size is assumed to *always* increase the whole collective's fecundity by the same magnitude (up to the proportionality coefficient). In turn, this causes a net collective fitness increase, even though the return might be diminishing (when viability and fecundity are not well balanced). We can imagine that this assumption might not hold for all trait values. Increasing cell fecundity might increase collective fecundity by increasing the potential number of propagules the collective can produce. However, we might reasonably think that the fast proliferation of cells negatively interacts with the propagule-producing mechanisms when above a certain threshold.

Concerning the previous point, Assumption 2 also implies a kind of "beanbag" model of collectives, where the relative position and orders of cells cannot be captured. It might seem obvious for eukaryotes with sophisticated developmental dynamics and organ partitioning that a cell will have a different impact on the collective fate depending on its position and the nature of its neighboring cells. However, even relatively simple examples of multicellular organisms, such as heterocyst-forming filamentous cyanobacteria (Chapter 9), demonstrate why this is pervasive. In these species, the lack of combined nitrogen in the environment induces the formation of differentiated cells, heterocysts, which are devoted to the fixation of atmospheric N_2. Heterocysts exchange fixed nitrogen compounds for carbon products with the neighboring photosynthesizing (vegetative) cells of the filament. Crucially, heterocysts are not located at random spots in the filament; rather, they are spaced at regular intervals (Yoon & Golden, 1998). For example, in the model species *Anabaena* sp. PCC 7120, heterocysts are separated by 10–15 vegetative cells (Herrero et al., 2016). This ensures an adequate supply of fixed nitrogen compounds while maximizing the number of vegetative cells within a filament (Rossetti et al., 2010). Notably, while vegetative cells can divide and generate all other specialized cell types, heterocysts cannot divide and are terminally differentiated. Thus, we observe not only a metabolic division of labor but also a reproductive one, where heterocysts are comparable to the somatic and the vegetative cells to the

germ cells in multicellular eukaryotic organisms (Rossetti et al., 2010). This structure cannot be described accurately in the original *LHM* (but see Yanni et al., 2020)

Assumption 2 implies that increasing any individual trait is bound to increase its collective counterpart. Assumption 3 prevents the simultaneous increase of both viability and fecundity.

12.2.3 Trade-off between Cell Viability and Fecundity

Assumption 3 of the *LHM* posits that a cell with a particular value for viability is necessarily constrained on its counterpart value for fecundity. Consequently, this reduces the number of free dimensions in the model—the two traits cannot vary independently.

This assumption covers the intuitive point that a cell cannot simultaneously be highly fecund and highly viable (i.e., an all-or-nothing cell) if it has a finite amount of energy to allow both of these (biological) functions. There are many ways to implement a trade-off in a model. The *LHM* does this using a relatively simple, deterministic, and one-dimensional method. Consider that, besides viability and fecundity, there is a third "hidden" trait for a cell, noted e, that quantifies the *effort* or *investment* toward one of the two traits. Then, by definition, the viability is an increasing function of the effort, $v_i = v(e_i)$, and the fecundity a decreasing function of the effort: $b_i = b(e_i)$. Here, v and b are (mathematical) functions that must be specified by the modeler. For instance, a simple linear trade-off can be defined as $v(e) = e$ and $b(e) = 1 - e$ for $e \in [0,1]$.

If the notion of effort is essential for understanding the logic of the trade-off, it can be abstracted graphically when representing the trade-off in the (v,b) plane introduced in Assumption 2. The trade-off can be represented as a curve (purple in Figure 12.3)

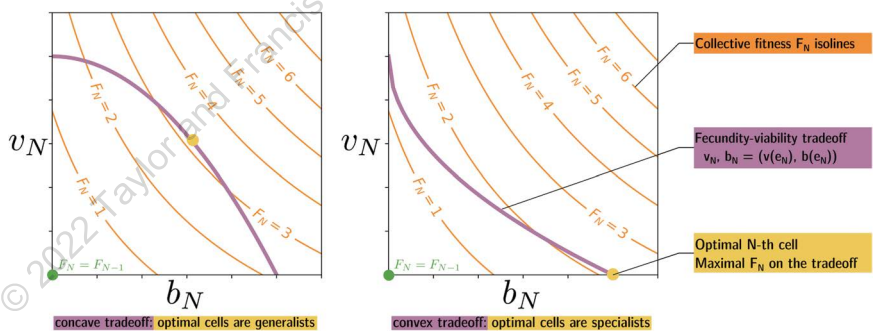

FIGURE 12.3 Isolines of fitness, trade-offs, and optimality of cell contributions. Representation in the plane (b_N, v_N) formed by the fecundity and viability of the N-th cell of a collective. As a consequence of Assumptions 1 and 2, collective fitness is a surface (represented by orange isolines) and, at the origin ($v_N = b_N = 0$), the collective fitness is minimal ($F_N = F_{N-1}$, green dot). As a consequence of Assumption 3, the values of v_N and b_N are constrained by a trade-off (purple line). As a consequence of Assumption 4, the model predicts that the traits favored by natural selection are those that yield the highest collective fitness (yellow disk) while satisfying the trade-off constraint. Concave (and linear) trade-offs (left) favor generalist cells (with balanced v, b), while convex trade-offs (right) favor specialist cells (with high v and low b, or vice versa).

constituting all the combinations of v_i, b_i given by all possible values of e_i. Different functional forms result in different trade-off shapes. Its shape and, in particular, its convexity are at the base of many strategies within the framework of life history theory (see Box 12.1 for a primer). We will return to this in discussing Assumption 5.

The notion of trade-offs in life history theory is an indubitably elegant way to incorporate an organism design's underlying constraints into a model. For instance, it can be used to account for the fact that the microtubule organizing center in the *Volvocaceae* cannot participate simultaneously in reproduction (through mitosis) and viability (through flagellar motility) (Koufopanou, 1994). While they are powerful theoretical tools, the existence of trade-offs is difficult to demonstrate, let alone quantify. One reason for this is that they can originate from many sources, such as physical (diffusion, buoyancy), genetic (metabolic pathways, regulations), or ecological (grazing, parasites) constraints. Moreover, trade-offs are not always set in stone. If physical constraints such as diffusion hardly change, mutation events can overturn other constraints—for instance, in the flagellate *Barbulanympha*, the microtubule organizing center can participate simultaneously in locomotion and reproduction (Buss, 1987). Note that, in the *LHM*, the shape of the trade-off changes with the size of the collective. This will be covered in more detail as part of Assumption 5.

Following Assumption 3, the set of all possible cells is reduced, as the trait of any new cell must be located on the trade-off curve. The model is not yet complete; natural selection acts on the organism in the context of these trade-offs, and its effect must be described. This is the purpose of Assumption 4.

12.2.4 Cell Contribution to the Collective is Optimal

So far, the role of natural selection has seldom been invoked in the *LHM*. We have only described the properties of cells and collectives and the diversity of traits they can exhibit, given some underlying constraints. Assumption 4 models the consequence of natural selection for this system—it assumes that all cells are optimal in terms of their contribution c to collective fitness. Formally, it means that the life history traits of any cell i within the collectives are such that the value of c_i is maximal:

$$e_i = argmax\, c_i$$

Note that optimality is an *assumption* rather than an outcome of the model.

Graphically, to find the values for a cell to contribute optimally to the collective, one must identify the intersection between the trade-off curve (purple in Figure 12.3) and the highest isoline of collective fitness (orange in Figure 12.3). This point is where a cell existing within the physiological constraints that link v and b has the highest fitness contribution. If the shape of the trade-off is sufficiently simple, there is a single optimal point and, thus, the model predicts the traits of any new cell based on Assumptions 1–4 plus the shape of the trade-off.

Of course, Assumption 4 could turn out to be incorrect if the cells are *not* optimal in their contribution to collective fitness. Cells might not be optimal for several reasons. For instance, the optimization of their traits might not occur independently from one another because they share the same underlying developmental program.

Alternatively, they might be "stuck" in another region of the trait space, in which case no viable mutation path would bring them to the optimal phenotype. Other reasons include that the trade-off's shape has recently changed due to changes in the environment or evolutionary forces (e.g., selection at another level or an evolutionary branching point) prevent the cells from reaching or remaining at the optimal phenotype.

Thus far, we have seen that the *LHM* assumes that the reproductive success of a collective depends on two fitness components (Assumption 1) that derive from their cell counterparts (Assumption 2), which are linked by underlying constraints (Assumption 3), and that natural selection is expected to favor optimal cells within this context (Assumption 4). The last piece of the puzzle is to qualify the shape of the trade-off—Assumption 5 does precisely this.

12.2.5 There is an Initial Reproductive Cost in Large Collectives

Assumption 5 states that small collectives have a linear or concave trade-off—favoring generalist cells—while large collectives have a convex trade-off—favoring division of labor. The distinction between linear, concave, and convex trade-offs is presented in Box 12.1. The mechanism proposed to explain why large collectives have a convex trade-off is the initial cost of reproduction. This assumption is critical because it characterizes the underlying constraints that bear on cell traits, but also ties them to the collective, in particular to collective size.

To understand Assumption 5, consider a cell specialized in viability (i.e., with a low fecundity) (Figure 12.4a). The mechanism for the initial cost of reproduction hinges on the assumption that, if this cell was investing more in fecundity than it currently does, it would reduce its viability but would *not* increase its fecundity (Figure 12.4b) until a threshold is reached (Figure 12.4c), after which it would increase (Figure 12.4d) until the cell is fully specialized in fecundity (Figure 12.4e).

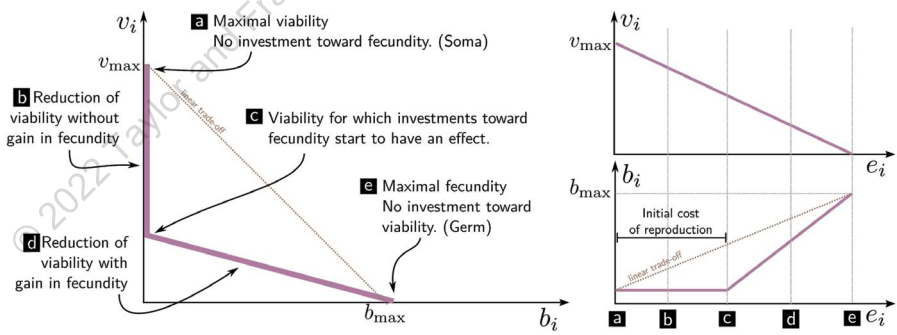

FIGURE 12.4 Initial reproductive cost as a model for a convex trade-off. Cell viability v_i and fecundity b_i are constrained, as represented by the purple curve corresponding to the possible combinations (v_i, b_i) under the trade-off modeled by the reproductive effort e_i. Starting from maximal investment toward viability (v_{max}, a), reduction in the viability effort has no effect on fecundity (b) until a threshold (c), where fecundity increases (d) up to the maximal fecundity allowed by the model (b_{max}, e). Contrast this with the simple linear trade-off (brown).

The relationship between group size and the shape of the trade-off between contribution to collective viability and fecundity is generally understood in terms of physical constraints. For instance, at the collective level, in the volvocine green algae, the enlargement of reproductive cells increases the downward gravitational force, increasing sinking; this is only overcome by the investment in more buoyant somatic cells (Solari et al., 2015). Thus, when colony size increases, a required initial investment toward buoyancy emerges that did not exist in unicellular organisms. This, in turn, explains how the trade-off, taken to be linear (or even concave) for single cells and small collectives, becomes convex when considering larger groups.

This is the last part of the *LHM*. As a consequence of Assumptions 1–5, large collectives favor the selection of specialist cells and, thus, division of labor.

12.3 DISCUSSION: FITNESS INTERPRETATIONS IN EVOLUTIONARY TRANSITIONS IN INDIVIDUALITY

The previous section presented a mechanism that promotes cell specialization between two life history traits (viability and fecundity) and, hence, a division of labor. This mechanism is based on the presence of a convex trade-off between the two traits due to the existence of an initial cost of reproduction in large collectives. This section places this model back in the broader context of ETIs by contrasting two interpretations of this phenomenon.

12.3.1 "Reorganization and Transfer of Fitness" Interpretation

A first interpretation of the *LHM* is based on the idea that the hallmark of an ETI is fitness reorganization/transfer/decoupling in the sense that cell specialization results in the lower-level cells "relinquish[ing] their autonomy in favor of the group" (Michod, 2005 p. 969; Michod et al., 2006, p. 258), resulting in a transfer of "fitness and individuality [from] the cell level to the group level" (ibid). We have tacitly assumed this interpretation throughout because it is the one with which the *LHM* was initially proposed when interpreting f_i as cell fitness and F_N as collective fitness.

This interpretation is rooted in the Multi-Level Selection 1–2 framework (Damuth & Heisler, 1988; Okasha, 2006), as cited in Michod (2005). In Multi-Level Selection 1 (MLS1) models, collective fitness is taken to be the average fitness of its members (or proportional to it), whereas in Multi-Level Selection 2 (MLS2), collective fitness cannot be defined in terms of particle survival and reproduction. Consequently, a new notion of fitness must be devised.

From this interpretation, the problem of explaining ETIs can be "reduced" to explaining the transition from an MLS1-like situation to an MLS2-like situation (Okasha, 2006, Chapter 8). Initially, this may appear to be an insurmountable hurdle because, in MLS1 (before the transition), cells are selected to have the highest cell fitness. In contrast, fully specialized cells in the model (after the ETI has occurred) have nil (or close to nil) fitness. The concept of *fitness transfer* (Michod, 2006) solves this problem by considering that, during an ETI, fitness between the two levels is reorganized—it is transferred from the lower level (the cells) to the higher

level (the collective). The transfer is achieved through germ and soma specialization (which results from the trade-off's convexity). When cells become specialized, they relinquish their fitness to the benefit of the collective. Further, during the process, collective fitness transitions from a mere average of cell fitness (MLS1) to a quantity, which is no longer the cell fitness average in the collective (MLS2) due to the covariance effect.

Although this interpretation is appealing, there are some problems associated with it. The main problem is that it seems to imply that fitness is a material quantity that can be transferred from one entity to another, comparable to a liquid that can be poured from one container to another. While, at first glance, this analogy may seem helpful for obtaining an intuitive idea of the problem, it contradicts our modern understanding of fitness as a predictor of evolutionary success (Bourrat, 2015a, 2015b, Bourrat, 2021a, b; Doulcier et al., 2021). First, it implies that some cells with nil fitness (or close to it) are not dead, contradicting the principle of natural selection. Second, because the evolutionary fates of cells and collectives are tied (by virtue of being made of the same biological substrate), it is difficult to determine how one level could ever be favored at the other's expense (Black et al., 2020; Bourrat, 2021a, b). This point is known in the philosophical literature as the "causal-exclusion principle." If a phenomenon is explained or described exhaustively at the lower level, one cannot appeal to the higher level to explain this phenomenon further. Doing so is either a form of "double counting" or requires assuming that strongly emergent properties are created ex nihilo at the higher level. Assuming the existence of strongly emergent properties raises a new range of issues because they contradict materialism, the idea that all causes are physical in nature.

Finally, the fitness transfer interpretation implies that cells constituting a multicellular organism can have different fitness values. This conflicts with the fact that those cells are clones and should, thus, have the same (inclusive) fitness (Bourrat, 2015b). To clarify this point, while a queen and a worker bee have different reproductive outputs, they have the same (inclusive) fitness. Evolutionarily, it does not make sense to say that the queen is more successful than the worker. Similarly, it does not make sense to say that a liver cell in a multicellular organism is fitter than a brain cell.

Recent work has helped to solve these issues by proposing a new interpretation of fitness at different levels of organization (Shelton & Michod, 2014, 2020). Following this new interpretation, the term "cell fitness" does not represent the cell's fitness within the collective but rather the one it *would have* if it were without a collective (counterfactual fitness). When cells have the same fitness they would have in the absence of the collective, no transition has occurred. However, when cells have different fitness, a transition has occurred (or at least been initiated). This constitutes a reasonable argument toward deciding whether an ETI happened (i.e., the state of cells and collectives). Crucially, this says nothing about the mechanism of the transition. There is no actual "decoupling" or "transfer" of fitness, other than in the loose metaphorical sense that the purely theoretical counterfactual fitness aligns or not with the actual fitness.

Metaphors and analogies are incredibly useful in biology because they allow us to build intuition of complex mechanisms by drawing parallels with other systems. *Fitness transfer* or *reorganization* implies the physical transfer of a material quantity

(with or without conservation). However, fitness is not transferred from one place (the cell) to another (the multicellular organism) in the way that heat, for instance, can be transferred. "Fitness transfer" might be used, but only in a loose metaphoric sense—that is, in the same way, teleological language in evolutionary biology is used in the context of a teleonomic explanation (Pittendrigh, 1958; Jacob, 1970). A further point worth mentioning is that metaphors may favor a specific interpretation that could obscure some aspects of the phenomena studied, such as how the sole focus on selection created the blind spots of the adaptationist program (Gould et al., 1979).

12.3.2 "Propensity" Interpretation

We favor an alternative interpretation of the *LHM*. This interpretation starts with the same mechanism—a convex trade-off between contribution to collective fecundity and collective viability will promote the emergence of individual specialist cells and, thus, a division of labor. It diverges from the previous one by its treatment of fitness, emphasizing how it emerges from cell *traits* rather than using it as a reified quantity of cells and collectives.

For the concept of fitness to qualify as a predictor in evolutionary biology, it cannot be reduced to an entity's actual success (i.e., its realized fitness). Instead, it must be tied to its *potential* success (or success in the long run). Without this point acknowledged, fitness is condemned to be tautological, as philosophers and biologists alike have long recognized (Manser, 1965; Popper, 1974; Smart, 1963; reviewed in Doulcier et al., 2021).[2] This conundrum has led to establishing several frameworks for the interpretation of fitness, one of which is the propensity interpretation of fitness (Brandon, 1978; Beatty, 1984; Pence & Ramsey, 2013). According to the propensity interpretation, fitness is a probabilistic property of entities summarizing their probability distribution of reproductive success (as defined by their demographic parameters: birth and death rates) in a given environment.

If we adopt this interpretation, the problems raised by the fitness transfer interpretation vanish. First, the problem of a collective's different (clonal) cells having different fitnesses disappears. Although their *realized* fitnesses (actual life history) might be different, their "true" fitnesses (potential life history) are equal because they relate to the potential success of the same genotype. Second, this interpretation does not appeal to fitness transfer or decoupling since cell and collective fitness are computed in expectation. Following the transfer of fitness interpretation, although this is not made explicit in the model, cell fitness and collective fitness are computed relative to different environments. In particular, collective-level demography (i.e., birth and death events of collectives) is typically ignored when computing cell fitness. Consequently, it becomes possible to define different values of fitness for the collective (F) and the cells (f). However, the fact that they are computed in different environments implies that they cannot legitimately be directly compared. When cell and collective fitnesses are computed in the same environment following the propensity interpretation—for instance, by factoring in collective events in the cell-level computation—they are necessarily equal (Bourrat, 2015a, 2015b; Bourrat et al., 2020). Cells and multicellular organisms are two levels of description of the same physical reality and cannot contradict one another, despite some claims to the contrary (e.g.,

Okasha, 2006).[3] Although conflicting processes might exist (e.g., segregation distortion locus, cancerous growth), fitness, properly computed to be comparable, must tally these conflicts and be coherent when referring to the same entity, regardless of the method of description.

Alternatively, one way to connect the interpretation we favor and the counterfactual fitness approach is to compare the fitness of free-living cells with cells within the collectives and observe *apparent decoupling* between these two environments: a decrease in free-living fitness and an increase in within-collective fitness (Bourrat, 2015a, 2015b, 2016; Bourrat et al., 2021; Bourrat, 2021a, 2021b). However, this apparent decoupling is rather a sign of *linkage* between the traits that contribute to free-living and within-collective fitness. The propensity interpretation of fitness can explain the same phenomena without invoking any "fitness transfer." If there is a "transfer," it is between the energetic investment of the cell toward different traits: from traits that provide no advantage to cells living in a collective (and potentially contributing to a free-living life cycle) toward traits that provide an advantage to the cells living in a collective (including vicarious advantages of cells with the same genotype).

Doing away with the reifying idea that fitness is something to be transferred and, more generally, treating the MLS1/MLS2 distinction as conventional—that is, two different ways to formalize the same idea—rather than as an evolutionary mechanism allows pursuing lines of inquiries that were more difficult to conceive within this framework. For instance, the focus on the relationship between cell and collective fitness leads naturally to the assumption that contributions to the collective fitness component are linear functions of their free-living counterparts (as was the case in Assumption 2). However, designing a mechanistic model naturally leads to relaxing this assumption. Traits of collectives are most certainly more complex than the arithmetic aggregation of individual quantities measured in the propagule or the fully developed collective. Rather, they are the result of internal developmental dynamics—that is, within-collective cellular ecological dynamics (Hammerschmidt et al., 2014; Rose et al., 2020). Selection of developmental mechanisms has long been recognized as a vital part of ETIs (Buss, 1987; Michod & Roze, 1997) and can be studied fully by models that describe the ecological dynamics within collectives explicitly (see Ikegami & Hashimoto, 2002; Williams & Lenton, 2007; Xie et al., 2019 for general cell communities; but see Doulcier et al., 2020 for an application to ETIs).

12.4 CONCLUSION

Division of labor is observed in complex organisms. The functions exhibited by multicellular organisms cannot be exhibited simultaneously by a single cell. Multicellularity solves this problem by allowing different subsets of cells to perform the different functions at once. The level of division of labor exhibited by a collective varies with the extent to which cells are specialized.

Natural selection favors specialist cells (hence, division of labor) if there is a convex trade-off between two equally important functions for cell fitness. The convexity of the trade-off is a consequence of two hypotheses: first, an energetic investment model in which a cell has limited energy to invest in two traits that contribute toward

each function and, second, an initial investment cost whereby a small investment in a trait does not translate immediately to an improvement of the function. The *LHM* predicts that collectives constituted of cells investing less energy in traits that contribute toward free-living fecundity and viability but more in traits that contribute toward fecundity and viability of collectives will progressively outcompete other collectives and become widespread.

This phenomenon has been interpreted as a "transfer of fitness" in the sense that individual cells relinquish their autonomy (investing less in free-living traits) to participate in life history traits of collectives (investing more in contribution toward collective function). During this "reorganization of fitness," cell fitness has been proposed to decrease while collective fitness increases. The fact that cell fitness and collective fitness do not change in the same direction has been named "fitness decoupling." However, this interpretation can be misleading because it conflicts with the concept of fitness as used in evolutionary biology. To fully appreciate the relevance of the *LHM* to ETIs, two things must be stressed. First, trade-offs occur between traits, not between fitnesses at different levels of organization. Second, fitness can only be defined with respect to a given entity (cell or collective) in a given environment and cannot be incoherent between the whole and the part. Thus, fitness cannot literally be "transferred" from individuals to collectives, even if, in retrospect, the traits that are adaptive in a collective environment would be detrimental to a free-living organism.

Trade-offs between life history traits are valid mechanisms—independently of the interpretation in terms of fitness transfer or steady state propensity, or even any other kind of interpretation one might propose (e.g., inclusive fitness, game theory, altruism). The interpretation chosen only represents a useful narrative for placing ETIs in the broader context of the evolution of complexity and allowing us to pursue subsequent questions, such as regarding developmental programs. Nonetheless, invoking a fitness concept that is consistent with the broader use of this term represents the primary reason for preferring one interpretation of the *LHM* to the other.

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NOTES

1. Or life history traits—the two terms are often used indistinguishably (Flatt & Heyland, 2012).
2. The propensity interpretation of probability is contentious in philosophy (Hájek, 2012). It produces a number of problems, some of which are inherited by the propensity interpretation of fitness (Godfrey-Smith, 2009; Bourrat, 2017). In recent years, several alternative interpretations of probabilities that play the same role as propensities and solve the issues of the propensity account have been proposed (e.g., Rosenthal, 2010; Lyon, 2011; Strevens, 2011; Abrams, 2012). Addressing the differences between these various interpretations in the context of fitness is beyond the scope of the present work. For our

purpose, we use "propensity" loosely as an entity's dispositional property to produce offspring (or equivalent terms in the aforementioned interpretations) without committing to any particular probability interpretation.
3. This claim admits a few theoretical exceptions, which are not relevant to ETIs.

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