

Fragmentation modes and the evolution of life cycles

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Abstract

Reproduction is a defining feature of living systems. Reproduction modes range from binary fission in bacteria to various modes of collective-level reproduction in multicellular organisms. However, the evolution of these modes and their adaptive significance is unclear. We develop a model in which groups arise from the division of single cells that do not separate, but stay together until the moment of group fragmentation. Fragmentation occurs via either complete or partial fission, resulting in a wide range of life cycles. By determining the relationship between life cycle and population growth rate, we define optimal fragmentation modes that have a surprisingly narrow class of solutions. Our model and results provide a framework for analysing the evolution of simple life cycles and for testing the adaptive significance of different modes of reproduction.

1 Introduction

A requirement for evolution – and a defining feature of life – is reproduction [Godfrey-Smith, 2009, Libby and Rainey, 2013, Hammerschmidt et al., 2014]. Perhaps the simplest mode of reproduction is

25 binary fission in unicellular bacteria, whereby a single cell divides and produces two offspring cells.
26 However, even a process as simple as this can underlie more complex modes of reproduction involving
27 life cycles comprised of recurring collective phases, or collective phases alternating with single cell
28 phases [Angert, 2005]. For example, in the bacterium *Neisseria*, a diplococcus, two daughter cells
29 remain attached forming a two-celled group that separates into two groups of two cells only after
30 a further round of cell division [Westling-Hägström et al., 1977]. *Staphylococcus aureus*, another
31 coccoid bacterium, divides in three planes at right angles to one another to produce grape-like clusters
32 of about 20 cells from which single cells separate to form new clusters [Koyama et al., 1977].

33 These are just a few examples of a large number of diverse “unicellular” reproduction modes, but
34 why should there be such a range of life cycles? Do these reproduction modes have adaptive signif-
35 icance or are they simply the unintended consequences of particular cellular processes underpinning
36 cell division? If adaptive, what selective forces shape their evolution? Do different life cycles provide
37 different opportunities to maximise intrinsic cell growth rate, or are collectives themselves the focal
38 units of selection?

39 A starting point to answer these questions is to consider benefits and costs of group membership.
40 Benefits may arise for various reasons. Cells within groups may be better able to withstand environ-
41 mental stress [de la Fuente-Núñez et al., 2013], escape predation [Boraas et al., 1998], or occupy new
42 niches [Bonner, 1982, Rainey and De Monte, 2014]. Also, via density dependent modes of gene reg-
43 ulation, cells within groups may gain more of a limiting resource than they would if alone [Williams
44 et al., 1992, Diggle et al., 2007]. On the other hand, cells within groups experience increased com-
45 petition and must also contend with the build up of potentially toxic waste metabolites [Groebe and
46 Mueller-Klieser, 1996, Stewart and Franklin, 2008]. Thus, it is reasonable to expect an optimal rela-
47 tionship between group size and mode of reproduction that is environment and organism dependent
48 [Tarnita et al., 2013, Rashidi et al., 2015, Kaveh et al., 2016].

49 Here we formulate and study a matrix population model [Caswell, 2001] representing a popula-
50 tion of groups of different size to consider all possible modes of group fragmentation. By determining
51 the relationship between life cycle and population growth rate, we show that there is, overall, a nar-
52 row class of optimal modes of fragmentation. When the process of fragmentation does not involve
53 costs, optimal fragmentation modes are characterised by a deterministic schedule and binary splitting,
54 whereby groups fragment into exactly two offspring groups. Contrastingly, when a cost is associated

55 with fragmentation, it can be optimal for a group to fragment into multiple propagules of equal size.

56 Our results show that the range of life cycles observed in simple microbial populations are likely
57 shaped by selection for intrinsic growth rate advantages inherent in different modes of group reproduc-
58 tion. These findings also have relevance for understanding the emergence of life cycles underpinning
59 the evolution of multicellular life.

60 **2 Methods**

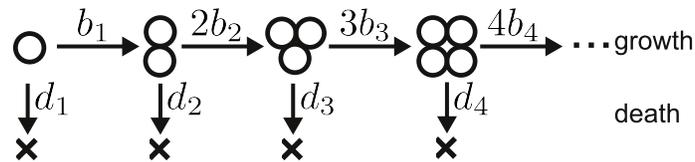
61 **2.1 Group formation and fragmentation**

62 We consider a population in which a single type of cell (or unit or individual) can form groups (or
63 complexes or aggregates) of increasing size by cells staying together after reproduction [Tarnita et al.,
64 2013]. We assume that the size of any group is smaller than n , and denote groups of size j by X_j .
65 Groups die at rate d_j and cells within groups divide at rate b_j ; hence groups grow at rate jb_j (Fig. 1.a).
66 The vectors of birth rates $\mathbf{b} = (b_1, \dots, b_{n-1})$ and of death rates $\mathbf{d} = (d_1, \dots, d_{n-1})$ make the costs
67 and benefits associated to the size of the groups explicit, thus defining the “fitness landscape” $\{\mathbf{b}, \mathbf{d}\}$
68 of our model.

69 Groups produce new complexes by fragmenting (or splitting), i.e., by dividing into smaller groups
70 (Fig. 1.b). We further assume that fragmentation is triggered by the growth of individual cells within
71 a given group. Consider a group of size j growing into a group of size $j + 1$. Such a group can either
72 stay together or fragment. If it fragments, it can do so in one of several ways. For example, a group
73 of size 4 can give rise to the following five “fragmentation patterns”: 4 (the group does not split but
74 stays together), 3+1 (the group splits into two offspring groups: one of size 3, and one of size 1), 2+2
75 (the group splits into two groups of size 2), 2+1+1 (the group splits into one group of size 2 and two
76 groups of size 1), and 1+1+1+1 (the group splits into four cells). Mathematically, such fragmentation
77 patterns correspond to the five partitions of 4 (a partition of a positive integer j is a way of writing j as
78 a sum of positive integers without regard to order; the summands are called parts [Andrews, 1998]).
79 We use the notation $\kappa \vdash \ell$ to indicate that κ is a partition of ℓ , for example $2 + 2 \vdash 4$. The number of
80 partitions of ℓ is given by ζ_ℓ , e.g., there are $\zeta_4 = 5$ partitions of 4.

81 We consider a vast space of life cycles comprising all possible ways groups can grow and fragment
82 into smaller groups. A “life cycle” (or “fragmentation mode”) assigns a probability q_κ to each possible

a) growth and death:



b) fragmentation:

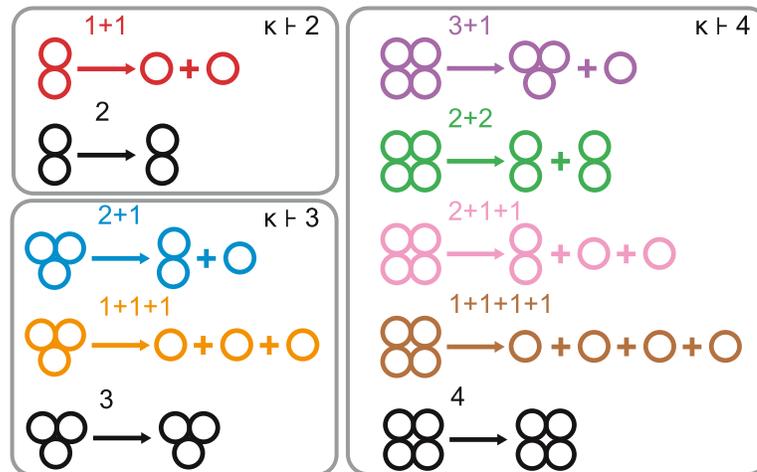


Figure 1: **Demographic dynamics depend on the growth, death, and fragmentation of groups.** (a) Groups of size i die at rate d_i and cells within them divide at rate b_i , hence groups grow at rate ib_i . (b) Fragmentation of groups occurs immediately after the reproduction of cells. Here we show all possible fragmentation patterns of groups of size $i = 2, 3, 4$. Each fragmentation pattern (determining the number and size of offspring groups) can be identified with a partition of i , i.e., a way of writing i as a sum of positive integers, that we denote by $\kappa \vdash i$. The last fragmentation pattern is i itself, in this case there is no actual fragmentation and cells stay together after individual reproduction.

83 fragmentation pattern (or partition) $\kappa \vdash 2, \kappa \vdash 3, \dots, \kappa \vdash n$. Such probabilities satisfy $\sum_{\kappa \vdash j+1} q_\kappa = 1$
 84 for $j = 1, \dots, n-1$, i.e., when growing from size j to $j+1$ one of the partitions $\kappa \vdash j+1$ (including
 85 staying together without splitting, $\kappa = (j+1)$) will certainly occur. Additionally, we impose $q_n = 0$ so
 86 that, when growing from size $n-1$ to size n , a group can no longer stay together and will necessarily
 87 fragment. It follows that a given life cycle or fragmentation mode can be represented by a set of
 88 vectors of the form

$$\mathbf{q} = \left\{ \underbrace{(q_2, q_{1+1})}_{\kappa \vdash 2}, \underbrace{(q_3, q_{2+1}, q_{1+1+1})}_{\kappa \vdash 3}, \dots, \underbrace{(q_n, q_{n-1+1}, q_{n-2+2}, \dots, q_{1+1+\dots+1})}_{\kappa \vdash n} \right\}. \quad (1)$$

89 As an illustration, consider the subset of stochastic strategies so that (i) with probability q a two-cell
 90 group grows to size three and then fragments according to fragmentation pattern 2+1, and (ii) with
 91 probability $1-q$ a two-cell group fragments according to fragmentation pattern 1+1. Such a set of
 92 strategies is represented by

$$\mathbf{q} = \{(q_2, q_{1+1}), (q_3, q_{2+1}, q_{1+1+1})\} = \{(q, 1-q), (0, 1, 0)\}. \quad (2)$$

93 We consider both deterministic and stochastic life cycles. For deterministic life cycles, splitting prob-
 94 abilities q_κ are either zero or one, so that only one fragmentation pattern with more than one offspring
 95 group occurs. This pattern can then be used to refer to the deterministic life cycle. For example,
 96 we represent the fragmentation mode $\{(1, 0), (0, 1, 0)\}$ by 2+1. The total number of deterministic
 97 fragmentation modes is

$$\sum_{j=1}^{n-1} (\zeta_{j+1} - 1), \quad (3)$$

98 which grows quickly with n : There are 128 deterministic fragmentation modes for $n = 10$, but
 99 1295920 for $n = 50$. The more general stochastic life cycles are characterised by some fragmentation
 100 pattern occurring with a probability between zero and one (Fig. 2).

101 2.2 Biological reactions and demographic dynamics

102 Together with the fitness landscape given by the vectors of birth rates \mathbf{b} and death rates \mathbf{d} , each
 103 fragmentation mode specifies a set of biological reactions. A number $n-1$ of reactions, of the type



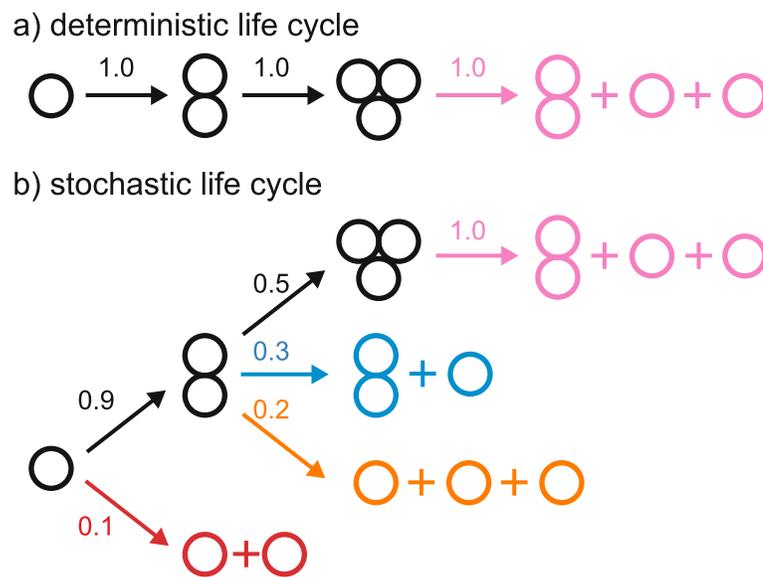
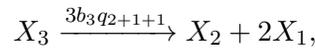


Figure 2: **Deterministic and stochastic life cycles.** (a) A deterministic life cycle (or fragmentation mode) given by $\mathbf{q} = \{(1, 0), (1, 0, 0), (0, 0, 0, 1, 0)\}$, whereby groups grow to size 3 and then split according to the fragmentation pattern 2+1+1. In a deterministic life cycle, fragmentation patterns are assigned a probability equal to either zero or one, so that fragmentation occurs only at a given group size (here, 3). (b) A stochastic life cycle given by $\mathbf{q} = \{(0.9, 0.1), (0.5, 0.3, 0.2), (0, 0, 0, 1, 0)\}$. In a stochastic life cycle, the probability of at least one fragmentation pattern is between zero and one.

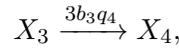
104 model the death of groups; these are independent of the fragmentation mode. An additional number
 105 of reactions, one per each non-zero element of the vector \mathbf{q} , models the birth of units and the growth
 106 or fragmentation of groups. These reactions are of the type



107 whereby a group of size j turns into a group of size $j + 1$ at rate jb_j , and then instantly divides with
 108 probability q_κ into offspring groups in a way described by fragmentation pattern $\kappa \vdash j + 1$, where
 109 parts equal to ℓ appear a number $\pi_\ell(\kappa)$ of times. These reactions depend on the life cycle, which
 110 specifies the probabilities of fragmentation patterns. For instance, the reaction



111 stipulates that groups of size 3, which grow to size 4 at rate $3b_3$, will split with probability q_{2+1+1} into
 112 one group of size 2 and two groups of size 1. The growth of a group without fragmentation is also
 113 incorporated in the set of reactions given by (5). For instance, the reaction



114 stipulates that groups of size 3, which grow to size 4 at rate $3b_3$, will not split with probability q_4 .

115 The sets of reactions (4) and (5) give rise to the system of differential equations

$$\dot{x}_i = \sum_{j=1}^{n-1} \sum_{\kappa \vdash j+1} q_\kappa \pi_i(\kappa) j b_j x_j - i b_i x_i - d_i x_i, \quad i = 1, 2, \dots, n-1, \quad (6)$$

116 where x_i denotes the abundance of groups of size i . This linear system can be represented in matrix
 117 form as

$$\dot{\mathbf{x}} = \mathbf{A} \mathbf{x}, \quad (7)$$

118 where $\mathbf{x} = (x_1, x_2, \dots, x_{n-1})$ is the vector of abundances of the groups of different size and

$$\mathbf{A} = \begin{pmatrix} b_1 \sum_{\kappa \vdash 2} q_\kappa \pi_1(\kappa) - b_1 - d_1 & 2b_2 \sum_{\kappa \vdash 3} q_\kappa \pi_1(\kappa) & \cdots & (n-1)b_{n-1} \sum_{\kappa \vdash n} q_\kappa \pi_1(\kappa) \\ b_1 \sum_{\kappa \vdash 2} q_\kappa \pi_2(\kappa) & 2b_2 \sum_{\kappa \vdash 3} q_\kappa \pi_2(\kappa) - 2b_2 - d_2 & \cdots & (n-1)b_{n-1} \sum_{\kappa \vdash n} q_\kappa \pi_2(\kappa) \\ 0 & 2b_2 \sum_{\kappa \vdash 3} q_\kappa \pi_3(\kappa) & \cdots & (n-1)b_{n-1} \sum_{\kappa \vdash n} q_\kappa \pi_3(\kappa) \\ 0 & 0 & \cdots & (n-1)b_{n-1} \sum_{\kappa \vdash n} q_\kappa \pi_4(\kappa) \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \cdots & (n-1)b_{n-1} \sum_{\kappa \vdash n} q_\kappa \pi_{n-1}(\kappa) - (n-1)b_{n-1} - d_{n-1} \end{pmatrix} \quad (8)$$

119 is the projection matrix determining the demographic dynamics of the population.

120 Note that the entries of the projection matrix \mathbf{A} are functions of the life cycle \mathbf{q} and the birth
 121 and death rates \mathbf{b} and \mathbf{d} . Since complexes can only split into complexes of equal or smaller size,
 122 $\pi_i(\kappa) = 0$ for all $\kappa \vdash j + 1$ and $i > j + 1$. Hence, the projection matrix \mathbf{A} has zero entries below the
 123 first subdiagonal. As an illustration, consider the subset of stochastic strategies represented by Eq. (2)
 124 and subject to the fitness landscape $\{\mathbf{b}, \mathbf{d}\} = \{(b_1, b_2), (d_1, d_2)\}$. In this simple case the projection
 125 matrix reduces to

$$\mathbf{A} = \begin{pmatrix} b_1 2(1 - q) - b_1 - d_1 & 2b_2 \\ b_1 q & -d_2 \end{pmatrix}. \quad (9)$$

126 2.3 Population growth rate

127 For any life cycle \mathbf{q} and any fitness landscape $\{\mathbf{b}, \mathbf{d}\}$, the projection matrix \mathbf{A} is essentially non-
 128 negative, i.e., all the elements outside the main diagonal are non-negative [Cohen, 1981]. This implies
 129 that \mathbf{A} has a real leading eigenvalue λ_1 with associated non-negative left and right eigenvectors \mathbf{v} and
 130 \mathbf{w} . In the long term, the solution of Eq. (7) converges to that of an exponentially growing population
 131 with a stable distribution, i.e.,

$$\lim_{t \rightarrow \infty} \mathbf{x}(t) = e^{\lambda_1 t} \mathbf{w}.$$

132 The leading eigenvalue λ_1 hence gives the total population growth rate in the long term, and its asso-
 133 ciated right eigenvector $\mathbf{w} = (w_1, \dots, w_n)$ gives the stable distribution of group sizes so that, in the
 134 long term, the fraction of complexes X_i in the population is proportional to w_i . The leading eigenvalue
 135 λ_1 is the largest solution of the characteristic equation $\det(\mathbf{A} - \lambda \mathbf{I}) = 0$. For example, the stochastic
 136 strategy represented by Eq. (2) and characterised by projection matrix (9) achieves a growth rate given
 137 by

$$\lambda_1 = \frac{b_1(1 - 2q) - (d_1 + d_2) + \sqrt{(d_1 + d_2 - (1 - 2q)b_1)^2 + 4b_1(2qb_2 + (1 - 2q)d_2)}}{2}. \quad (10)$$

138 In the particular case of a deterministic life cycle associated to fragmentation pattern $\kappa \vdash m$, the
 139 characteristic equation reduces to (Appendix A.1)

$$F_m(\lambda) - \sum_{i=1}^{m-1} \pi_i(\kappa) F_i(\lambda) = 0, \quad (11)$$

140 where

$$F_i(\lambda) = \prod_{j=1}^{i-1} \left(1 + \frac{d_j + \lambda}{j b_j} \right). \quad (12)$$

141 For instance, the growth rate of fragmentation mode 2+1+1 (illustrated in Fig. 2a) is given implicitly
 142 by the largest solution of $F_4(\lambda) - F_2(\lambda) - 2F_1(\lambda) = 0$. As Eq. (11) is a polynomial equation of degree
 143 $m - 1$, explicit formulas for the growth rate in terms of birth rates and death rates are only available
 144 for small m ($m \leq 5$). For instance, the growth rate of fragmentation modes 1+1, 1+1+1, and 2+1 are
 145 respectively given by

$$\lambda_1^{1+1} = b_1 - d_1, \quad (13a)$$

$$\lambda_1^{2+1} = \frac{-(b_1 + d_1 + d_2) + \sqrt{(b_1 + d_1 - d_2)^2 + 8b_1b_2}}{2}, \quad (13b)$$

$$\lambda_1^{1+1+1} = \frac{-(b_1 + 2b_2 + d_1 + d_2) + \sqrt{b_1^2 + 2b_1(10b_2 + d_1 - d_2) + (2b_2 - d_1 + d_2)^2}}{2}. \quad (13c)$$

146 For larger values of m , we solve Eq. (11) numerically.

147 2.4 Dominance and optimality

148 For a given fitness landscape $\{\mathbf{b}, \mathbf{d}\}$, we can take the leading eigenvalue $\lambda_1(\mathbf{q}; \mathbf{b}, \mathbf{d})$ as a measure
 149 of fitness of life cycle \mathbf{q} , and consider the competition between two different life cycles, \mathbf{q}_1 and \mathbf{q}_2 .
 150 Indeed, under the assumption of no density limitation, the evolutionary dynamics are described by two
 151 uncoupled sets of differential equations of the form (7): one set for \mathbf{q}_1 and one set for \mathbf{q}_2 . In the long
 152 term, \mathbf{q}_1 outcompetes \mathbf{q}_2 if $\lambda_1(\mathbf{q}_1; \mathbf{b}, \mathbf{d}) \geq \lambda_1(\mathbf{q}_2; \mathbf{b}, \mathbf{d})$; we then say that life cycle \mathbf{q}_1 dominates life
 153 cycle \mathbf{q}_2 and write $\mathbf{q}_1 \geq_{\lambda_1} \mathbf{q}_2$. We also say that strategy \mathbf{q}_i is optimal for given birth rates \mathbf{b} and death
 154 rates \mathbf{d} if it achieves the largest growth rate among all possible fragmentation modes, i.e., $\mathbf{q}_i \geq_{\lambda_1} \mathbf{q}_j$
 155 for all \mathbf{q}_j in the set of life cycles.

156 2.5 Two classes of fitness landscapes: fecundity landscapes and viability landscapes

157 Fitness landscapes capture the many advantages or disadvantages associated with group living. These
 158 advantages may come either in the form of additional resources available to groups depending on their
 159 size or as an improved protection from external hazards. For the sake of simplicity, we consider two
 160 classes of fitness landscape, each representing only one of these factors. In the first class, that we call
 161 “fecundity fitness landscapes”, the group size affects only the birth rates of cells, while death rates
 162 remain independent of the group size; for convenience, we impose $d_i = 0$ for all i . In the second
 163 class, that we call “viability fitness landscapes”, the group size affects only death rates, while birth

164 rates remain independent of the group size; in this case, we assume $b_i = 1$ for all i .

165 **3 Results**

166 **3.1 Optimal life cycles are deterministic and characterised by binary fragmentation**

167 We find that stochastic life cycles are dominated by a deterministic life cycle, i.e. for a given stochas-
168 tic life cycle and fitness landscape it is always possible to find at least one deterministic life cycle
169 that achieves a larger growth rate. As an illustration, consider the two deterministic strategies 1+1
170 and 2+1, and a stochastic strategy q mixing between these two deterministic modes so that with prob-
171 ability q fragmentation is governed by 2+1 and with probability $1 - q$ it is governed by 1+1, i.e.,
172 a stochastic strategy represented by Eq. (2). For any mixing probability q and any fitness land-
173 scape $\{(b_1, b_2), (d_1, d_2)\}$, the growth rate λ_1^q of the corresponding stochastic strategy (given explicitly
174 by Eq. (10)) is between the growth rates of the deterministic strategies, $\lambda_1^{1+1} \leq \lambda_1^q \leq \lambda_1^{2+1}$ or
175 $\lambda_1^{2+1} \leq \lambda_1^q \leq \lambda_1^{1+1}$ holds. Hence, either the growth rate of 1+1 or the growth rate of 2+1 is larger
176 than the growth rate of the stochastic strategy (Appendix A.2). Indeed, we are able to show a more
177 general result: that for any fitness landscape and any maximum group size n , all stochastic strategies
178 are dominated by at least one deterministic strategy. This allows us to conclude that the optimal life
179 cycle is always a deterministic fragmentation mode (Appendix A.3).

180 We also find that, within the set of deterministic life cycles, “binary fragmentation” strategies
181 (whereby groups split into exactly two offspring groups) dominate “non-binary fragmentation” strate-
182 gies (whereby groups split into more than two offspring groups). To illustrate this result, consider
183 the simplest case of $n = 3$ and the three deterministic strategies 1+1 (binary), 2+1 (binary), 1+1+1
184 (non-binary). By comparing the growth rates of the strategies, given by Eq. (13), we find that
185 $\lambda_1^{1+1} \geq \lambda_1^{1+1+1}$ holds if $b_1 - b_2 \geq d_1 - d_2$ and that $\lambda_1^{2+1} \geq \lambda_1^{1+1+1}$ holds if $b_1 - b_2 \leq d_1 - d_2$. Thus,
186 for any fitness landscape, 1+1+1 is dominated by either 2+1 or by 1+1. More generally, we can show
187 that for any maximum group size n , any fitness landscape, and any non-binary fragmentation strategy,
188 one can find a binary fragmentation strategy achieving a greater or equal growth rate (Appendix A.4).
189 Hence, within the full space of strategies that we consider, only deterministic strategies characterised
190 by binary fragmentation strategies are optimal.

191 Taken together, our analytical results imply that the set of optimal strategies is countable and, even

192 for large n , relatively small. As the number of strategies increases rapidly with n , we consider the
193 proportion of deterministic strategies that can be optimal. While this is relatively high for small n
194 (e.g., $2/3 \approx 0.67$ for $n = 3$ or $4/7 \approx 0.57$ for $n = 4$), it decreases sharply with increasing n (e.g.,
195 $25/128 \approx 0.20$ for $n = 10$ and $625/1295920 \approx 0.00048$ for $n = 50$).

196 Fig. 3 illustrates a numerical test of our findings for groups of maximum size $n = 4$ and a fecundity
197 fitness landscape given by $\mathbf{b} = (1, b_2, 1.4)$ and $\mathbf{d} = (0, 0, 0)$. In line with our analysis, and for all
198 values of b_2 , the optimal life cycle is always characterised by deterministic binary fragmentation. In
199 particular, for $b_2 = 2$, the optimal life cycle is 2+2, whereby groups grow up to size 4 and then
200 immediately split into two bi-cellular groups (Fig. 3.a). Other deterministic binary fragmentation
201 strategies can also be optimal, depending on the value of b_2 (Fig. 3.b). For small values ($b_2 \lesssim 0.45$),
202 the production of bi-cellular complexes is too disadvantageous, hence the optimal life cycle is 1+1
203 (under which bi-cellular groups are not produced). For intermediary values ($0.45 \lesssim b_2 \lesssim 1.11$), the
204 reproduction efficiency of three-cellular groups mitigates the inefficiency of two-cellular groups, and
205 the strategy 3+1 becomes optimal. For larger values ($1.11 \lesssim b_2 \lesssim 3.52$), the optimal strategy is
206 2+2, where no independent cells are produced. Finally, for very large values ($b_2 \gtrsim 3.52$), the optimal
207 strategy is 2+1, which ensures that one offspring group remains at the most productive bi-cellular
208 state.

209 Fig. 4 shows the optimal life cycles for fecundity and viability landscapes and $n = 4$ (Ap-
210 pendix A.5). Under fecundity fitness landscapes where unicells have the largest birth rates, the optimal
211 life cycle is 1+1. In this case, unicells perform better than larger cell complexes, and the optimal life
212 cycle leads to populations consisting only of unicells. Under fitness landscapes where the birth rates
213 of bi- and three-cellular complexes are similar and much larger than those of unicells, the optimal
214 life cycle is 2+2. In this case, unicells perform worse than larger cell groups, and the optimal life
215 cycle ensures that the population does not contain unicells. Under fitness landscapes where two cells
216 have much higher birth rates than one cell or three cells, the optimal strategy is 2+1. In this case, the
217 optimal life cycle keeps one of the offspring groups at the most productive state. The same argument
218 holds for fitness landscapes where three cells have much higher birth rates than single cells or two
219 cells, but in this case the optimal life cycle is 3+1. Under viability fitness landscapes, the performance
220 of complexes improves with the decrease of the death rate. Thus, the map of optimal life cycles under
221 viability landscapes follows the same qualitative pattern as under fecundity fitness landscapes.

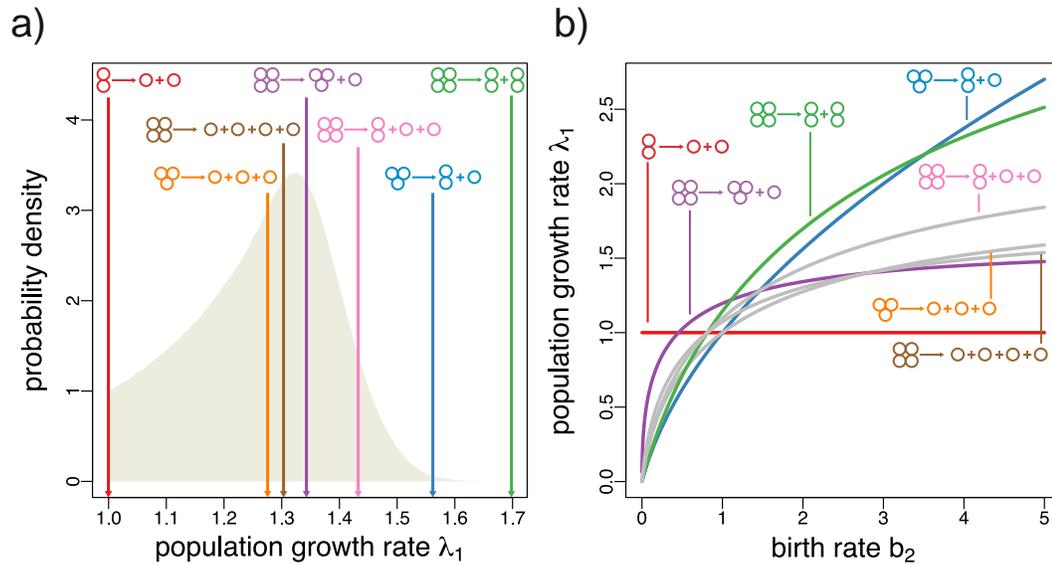


Figure 3: Deterministic binary fragmentation is optimal.

a) Empirical distribution of population growth rate of stochastic life cycles for $n = 4$ (generated from a sample of 10^7 randomly generated life cycles) subject to the fitness landscape $\{\mathbf{b}, \mathbf{d}\} = \{(1, 2, 1.4), (0, 0, 0)\}$. The population growth rates of all seven deterministic life cycles for $n = 4$ are indicated by arrows. Here, the deterministic life cycle 2+2 achieves the maximal possible growth rate among all possible life cycles. Random stochastic life cycles are generated as follows: The probabilities for growth without fragmentation are uniformly distributed. With the remaining probability, fragmentation occurs. The weight of each available fragmentation pattern is exponentially distributed.

b) Population growth rate (λ_1) for all seven deterministic life cycles for $n = 4$ subject to the fitness landscape $\{\mathbf{b}, \mathbf{d}\} = \{(1, b_2, 1.4), (0, 0, 0)\}$ as a function of the birth rate of bi-cells, b_2 . Each of the four life cycles characterised by binary fragmentation (1+1, 2+1, 2+2, and 3+1) can be optimal depending on the value of b_2 . Contrastingly, the three life cycles where a group fragments into more than two groups (1+1+1, 1+1+1+1, and 2+1+1) are never optimal.

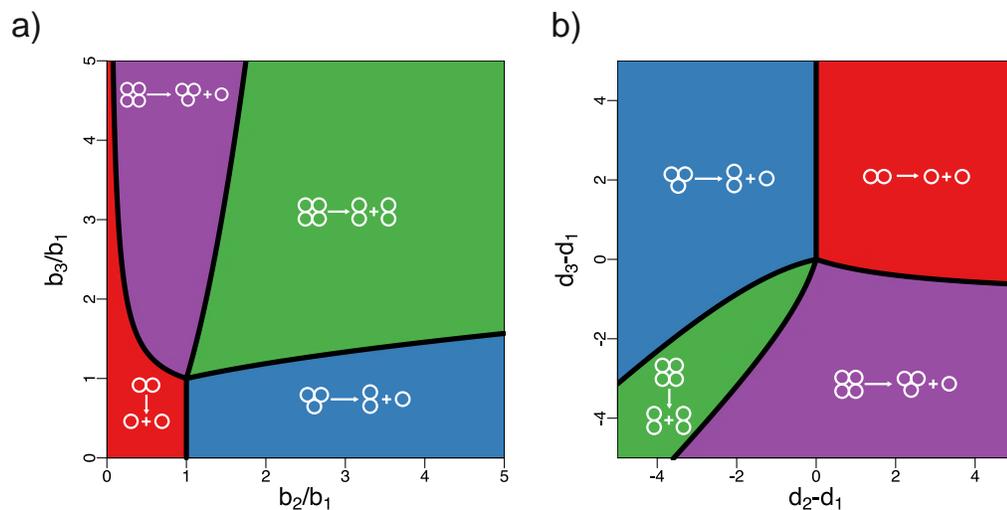


Figure 4: Optimal life cycles for fecundity and viability fitness landscapes. (a) Life cycles achieving the maximum population growth rate for $n = 4$ under fecundity fitness landscapes (i.e., $d_1 = d_2 = d_3 = 0$). In this scenario, the strategy 2+2 is optimal for most fitness landscapes. (b) Life cycles achieving the maximum population growth rate for $n = 4$ under viability fitness landscapes (i.e., $b_1 = b_2 = b_3 = 1$). In this scenario, life cycles emitting a unicellular propagule (1+1, 2+1, 3+1) are optimal for most parameter values. We use ratios of birth rates and differences between death rates as axes because one can consider $b_1 = 1$ and $\min(\mathbf{d}) = 0$ without loss of generality (Appendix A.1). Shaded areas are obtained from the direct comparison of the numerical solutions of characteristic equations in the form (11), lines are found analytically (Appendix A.5).

222 **3.2 Synergistic interactions between cells promotes the production of unicellular propag-** 223 **ules, while discounting interactions promote equal fragmentation.**

224 Next, we focus on monotonic fitness landscapes for which either the birth rate of cells increases with
225 group size (i.e., larger groups are more productive) or the death rate of groups decreases with group
226 size (i.e., larger groups live longer). In particular, we consider fecundity fitness landscapes given by
227 $b_i = 1 + Mg_i$ and $d_i = 0$ or viability fitness landscapes given by $b_i = 1$ and $d_i = M(1 - g_i)$,
228 where $g_i = [(i - 1)/(n - 2)]^\alpha$ is the group size benefit [Fromhage and Kokko, 2011] and $M > 0$
229 is the maximum benefit. The parameter α is the degree of complementarity between different units
230 (Fig. 5.a), it measures how important the addition of another unit is in producing the maximum pos-
231 sible benefit M [De Jaegher, 2017]. For $\alpha < 1$ the sequence g_i is strictly concave and the degree of
232 complementarity is low: each additional unit in the group contributes less to the per capita benefit of
233 group living [Hirshleifer, 1983], such that groups of all sizes achieve the same functionality as α tends
234 to zero. For $\alpha = 1$ the sequence g_i is linear, and each additional unit contributes equally to the fitness
235 of the group. Finally, for $\alpha > 1$, the sequence g_i is strictly convex and the degree of complementarity
236 is high, with each additional unit improving the performance of the group more than the previous unit
237 did. In the limit of large α , the advantages of group living materialise only when complexes achieve
238 its maximum size $n - 1$ [Hirshleifer, 1983].

239 We numerically compute the optimal fragmentation modes for $n = 20$ and the fitness landscapes
240 described above with $0.01 \leq \alpha \leq 100$ and $0.02 \leq M \leq 50$. We find that, for each value of α and M ,
241 the optimal life cycle is one where fragmentation occurs at the largest possible size (when the 20-th
242 cell is born), i.e., a fragmentation mode belonging to the set $S = \{10 + 10, 11 + 9, \dots, 19 + 1\}$. This
243 is because the maximum of the benefit sequence g_i is at $i = 19$, so that groups of maximum size
244 perform better, either by achieving the largest birth rate per unit (fecundity landscape) or the lowest
245 death rate (viability landscape).

246 For low degrees of complementarity, the optimal life cycle is always to split into equally sized
247 offspring groups. The intuition behind this result is that for very low complementarity, all multicel-
248 lular groups ($i \geq 2$) have similar performance, while unicellular groups ($i = 1$) are at a significant
249 disadvantage. Therefore, the optimal fragmentation mode is to ensure that both offspring groups are
250 as large as possible, and hence of the same size. Contrastingly, for high degrees of complementarity,
251 the optimal life cycle is always to fragment into one large group and one unicell. Here, the intuition is

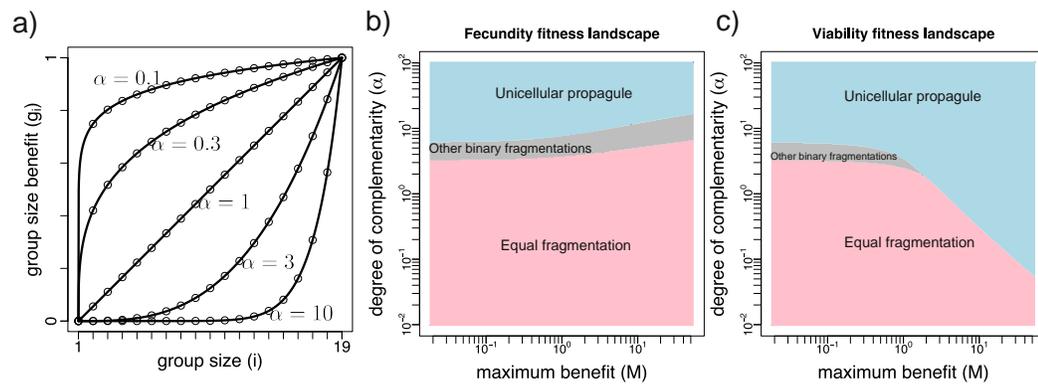


Figure 5: “Equal fragmentation” and “unicellular propagule” fragmentation modes are often optimal under monotonic fitness landscapes. (a) Group size benefit $g_i = [(i - 1)/(n - 2)]^\alpha$ as a function of group size for different values of the degree of complementarity α and $n = 20$. (b) Optimal life cycles under fecundity fitness landscapes with $b_i = 1 + Mg_i$ as a function of maximum benefit M and degree of complementarity α . Equal fragmentation is optimal if the degree of complementarity α is not too large. Increasing the maximum group size benefits M also promotes the evolution of equal fragmentation. (c) Optimal life cycles under viability fitness landscapes with $d_i = M(1 - g_i)$ as a function of M and α . Unicellular propagules are optimal if the degree of complementarity α is sufficiently large. An increase in the magnitude of group size bonus M also promotes the evolution of unicellular propagule.

252 that only the largest group can reap the benefits of group living and so the optimal mode is to have at
253 least one offspring of very large size.

254 The optimal life cycle also depends on whether group size affects birth rate (fecundity landscapes)
255 or death rate (viability landscapes). If a larger group size leads to an increase in the birth rate, then
256 for low values of the maximum benefit M producing unicellular propagules is optimal only for high
257 degrees of complementarity ($\alpha \gtrsim 7$). Larger values of M further rise the critical value of α above
258 which the unicellular propagule is the optimal life cycle. Contrastingly, if a larger group size leads to
259 a decrease in the death rate, then unicellular propagules can be optimal even if the degree of comple-
260 mentarity is low ($\alpha \lesssim 1$). In general, benefits on the birth rate make equal fragmentation optimal under
261 more demographic scenarios. On the contrary, benefits on the death rate make unicellular propagule
262 optimal under more demographic scenarios.

263 **3.3 Costly fragmentation allows splitting into multiple offspring groups to be optimal**

264 Up to this point, we have assumed that fragmentation is costless. However, fragmentation processes
265 can be costly to the parental group undergoing division. Reproduction costs are apparent in the case of
266 *Volvox*, where somatic cells constituting the outer layer of the group die upon releasing the offspring
267 cells and are not passed to the next generation [Kirk, 2005]. Reproduction costs may also be less
268 apparent. For instance, group division may cost resources that would otherwise be available for the
269 growth of cells within a group.

270 To investigate the effect of fragmentation costs on the set of optimal life cycles, we assume that
271 exactly one cell is lost upon each fragmentation event, so that the total number of cells in offspring
272 groups is one less than the number of cells in the parental group. Mathematically, this implies that,
273 upon reaching size $i+1$, fragmentation patterns are described by partitions of i rather than by partitions
274 of $i+1$ (Appendix A.6).

275 For such kind of costly fragmentation, stochastic life cycles are still dominated by deterministic
276 life cycles (a proof similar to the one given in Appendix A.3 for costless fragmentation still holds
277 in this case). However, under costly fragmentation, the set of optimal life cycles can also comprise
278 instances of non-binary fragmentation (i.e., division into more than two offspring groups). This can be
279 readily illustrated for the case of $n=4$ where the mode 1+1+1 is optimal for a wide range of fitness
280 landscapes (Fig. 6).

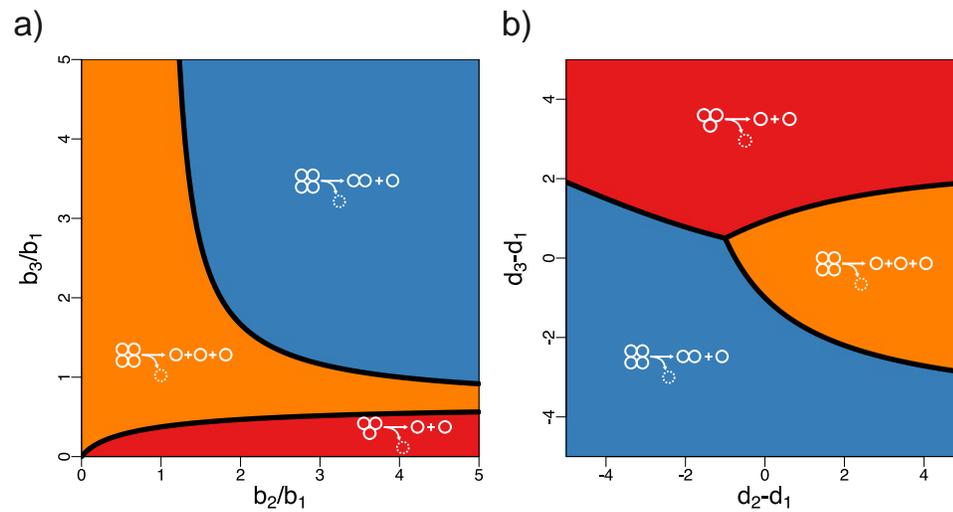


Figure 6: Optimal life cycles for fecundity and viability fitness landscapes under costly fragmentation.

Under costly fragmentation, each splitting event is associated with the loss of one cell. (a) Life cycles achieving the maximum population growth rate for $n = 4$ under fecundity fitness landscapes (i.e., $d_1 = d_2 = d_3 = 0$). (b) Life cycle strategies achieving the maximum population growth rate for $n = 4$ under viability fitness landscapes (i.e., $b_1 = b_2 = b_3 = 1$). In both classes of fitness landscapes, the life cycle 1+1+1, whereby 4-unit complexes produce 3 surviving independent units, is optimal under a wide range of fitness landscapes.

281 Another notable effect provided by the scenario of costly fragmentation is that life cycles involving
282 the emergence of large groups may be optimal even if groups do not grant any advantage to cells. If
283 fragmentation is costless, as we assumed above, fitness landscapes for which groups have inferior
284 performance in comparison with independent cells (that is, $b_i/b_1 \leq 1$ for fecundity landscapes or
285 $d_i - d_1 \geq 0$ for viability landscapes) lead to optimal life cycles where fragmentation occurs at the
286 minimal possible group size $i = 2$, so that no multicellular groups emerge in the population (see
287 Fig. 4). Contrastingly, in the case of costly fragmentation some of these fitness landscapes promote
288 fragmentation modes in which groups split at the maximal available size $n = 4$. Thus, the population
289 may contain multi-celled groups, even if these groups perform strictly worse than independent cells.
290 Such a counterintuitive behavior can be evolutionarily optimal, because the burden of inevitable cell
291 loss is indirectly shared among multiple offspring groups. Thus, the growth to a large size and then
292 fragmentation into multiple offspring groups minimizes the cost of cell loss and might be therefore
293 beneficial, even if the groups themselves are at a disadvantage.

294 We also identified optimal life cycles under costly fragmentation for larger group sizes and mono-
295 tonic fitness landscapes (Fig. 7). To obtain results comparable to the case of costless fragmentation,
296 we increased the group size limit by one, i.e., we assumed $n = 21$, so that the sum of offspring sizes
297 in both cases is equal to 20. Similarly to the case of costless fragmentation, all optimal life cycles are
298 such that splitting occurs only at the maximal possible size n . However, under costly fragmentation, a
299 new class of life cycle may be optimal: splitting into more than two offspring groups of similar size;
300 we call a life cycle within this class a “multiple fragmentation mode”. The most prominent mode
301 in this class is the fragmentation into multiple independent cells, i.e., $1+1+\dots+1$. This fragmentation
302 mode is promoted by fitness landscapes with low maximum benefit ($M < 1$). Under these fitness
303 landscapes, the group size has a small impact on the group performance, so the fragmentation cost
304 becomes the main factor determining the optimality of life cycles. The optimal mode to minimize
305 the fragmentation cost is to produce the maximal number of offspring per fragmentation event, i.e., to
306 fragment into independent cells.

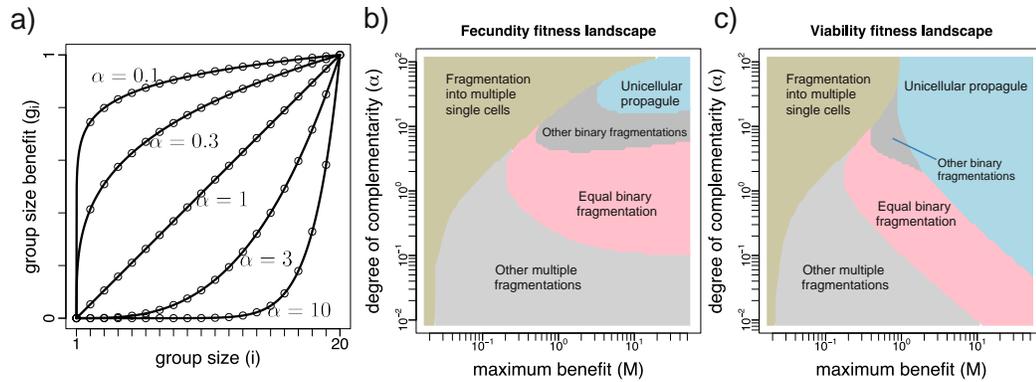


Figure 7: Multiple fragmentation into groups of similar size can be optimal under costly fragmentation and monotonic fitness landscapes. (a) Group size benefit $g_i = [(i - 1)/(n - 2)]^\alpha$ as a function of group size for different values of the degree of complementarity α and $n = 21$. (b) Optimal life cycles under fecundity fitness landscapes with $b_i = 1 + Mg_i$ as a function of maximum benefit M and degree of complementarity α . The fragmentation into multiple single cells is described by the fragmentation pattern $1+1+\dots+1$; other multiple fragmentation strategies have patterns $2+2+\dots+2$, $3+3+3+3+3+2$, $4+4+3+3+3+3$, $4+4+4+4+4$, $5+5+5+5$, and $7+7+6$. They are all coloured together as a single light gray area. (c) Optimal life cycles under viability fitness landscapes with $d_i = M(1 - g_i)$ as a function of M and α .

307 4 Discussion

308 Reproduction is such a fundamental feature of living systems that the idea that the mode of reproduc-
 309 tion may be shaped by natural selection is easily overlooked. Here, we analysed a matrix population
 310 model that captures the demographic dynamics of complexes that grow by staying together and repro-
 311 duce by fragmentation. The costs and benefits associated with group size determine whether or not
 312 a single cell fragments into two separate daughter cells upon cell division, or whether those daughter
 313 cells remain in close proximity, with fragmentation occurring only after subsequent rounds of division.

314 We showed that for each stochastic fragmentation mode there is a deterministic fragmentation
 315 mode that leads to higher cellular growth rate. Such a deterministic mode involves a regular sched-
 316 ule of group development and fragmentation. We also showed that, for any fitness landscape where
 317 fragmentation occurs without cell loss, the optimal fragmentation mode always involves binary frag-
 318 mentation. It is important to note that this does not mean that, for a given fitness landscape, a given
 319 mode with binary fragmentation will necessarily outperform a non-binary fragmentation mode. In-
 320 stead, for any given fitness landscape the best possible mode of reproduction will be one that involves

321 binary fragmentation. For example, with reference to Fig. 3*b* and for low b_2 , a group of three cells
322 that fragments into 1+1+1 outperforms three cells that undergo binary fragmentation producing 2+1.
323 However, 1+1+1 is outperformed by the unicellular life cycle 1+1, and also by groups of four cells
324 that fragment into 3+1.

325 A particularly intriguing finding is that the optimal life cycle under monotonic fitness landscapes
326 is generally of one of two types: “equal fragmentation”, which involves fission into two equal size
327 groups, and “unicellular propagule”, which involves the production of two groups, one comprised of
328 a single cell (Fig. 5). Equal fragmentation is favoured when there is a significant advantage associated
329 with formation of even the smallest group, whereas unicellular propagule is favoured when the benefits
330 associated with group size are not evident until groups become large. This makes intuitive sense:
331 when advantages arise when groups are small, it pays for offspring to be groups (and not single cells).
332 Conversely, when there is little gain until group size is large, it makes sense to maintain one group that
333 reaps this advantage.

334 Many multicellular organisms are characterised by a life cycle in which adult individuals develop
335 from a single cell [Grosberg and Strathmann, 1998]. Although passing through a unicellular bottleneck
336 is a requirement for sexual reproduction, life cycles with unicellular stages are also common in asexual
337 organisms such as multicellular algae and ciliates [Herron et al., 2013]. If multicellularity evolved
338 because of the benefits associated to group living, why do so many multicellular organisms begin their
339 life cycles as solitary (and potentially vulnerable) cells? Explanatory hypotheses include the purge
340 of deleterious mutations and the reduction of within-organism evolutionary conflict resulting from
341 clonality [Maynard Smith and Szathmáry, 1995, Grosberg and Strathmann, 1998]. Our results make
342 the case for an alternative (and perhaps more parsimonious) answer to this question: for relatively high
343 degree of complementarity on the number of cells of an organism, a life cycle featuring a unicellular
344 bottleneck is just the best way to guarantee that the “parent” group remains as large as possible to
345 reap off the maximum fecundity and/or viability advantages of group living. Our theoretical results
346 resonate with previous experimental work demonstrating that single-cell bottlenecks can be adaptive
347 simply because they maximise growth rate [Ratcliff et al., 2013].

348 Once cell loss upon fragmentation is incorporated as a factor in collective reproduction, a wider
349 range of fragmentation patterns become optimal. Such optimal life cycles include those where splitting
350 involves the production of multiple offspring (including multiple independent cells). These strategies

351 are optimal under conditions where there is negligible benefit associated with group size (Fig. 7).
352 Intuitively, this is because the production of multiple offspring allows the cost of cell loss to be spread
353 among many offspring.

354 This distribution of costs among offspring may also explain the tendency for life cycles involving
355 larger groups to outperform life cycles that fragment at smaller group size under fitness landscapes
356 that penalize groups of increasing size (e.g., when $b_1 > b_2 > b_3$). In the absence of a cost due to cell
357 loss, such landscapes favour the unicellular life cycle (Fig. 4a). With costs associated with cell loss,
358 not only is the unicellular life cycle excluded, but life cycles involving large groups can be favoured
359 over those involving smaller groups. This is evident in Fig. 6: under the fitness landscape given by
360 $\{\mathbf{b}, \mathbf{d}\} = \{(1, 0.9, 0.8), (0, 0, 0)\}$, groups that fragment at four cells outperform groups that fragment
361 at three cells. This reflects the fact that groups growing up to four cells before fragmentation distribute
362 the cost of cell loss among the three remaining cells, whereas a group that reaches three cells before
363 fragmentation distributes the cost among just two offspring cells.

364 Previous theoretical work has explored several questions related to the evolution of multicellular-
365 ity using matrix population models similar to the one proposed in this paper. In a seminal contribution,
366 Roze and Michod [2001] explored the evolution of propagule size in the face of deleterious and selfish
367 mutations. In their model, multicellular groups first grow up to an adult size and then reproduce by
368 splitting into equally sized groups, so that life cycle strategies can be indexed by the size of the propa-
369 gule size. In our terminology, this refers to the class of (deterministic) “multiple fragmentation modes”.
370 An important finding of Roze and Michod [2001] is that, even if large groups are advantageous, small
371 propagules can be often selected because they are more efficient at eliminating detrimental mutations.
372 Contrastingly, we did not study the effects of mutations, as cells in our model replicate faithfully.
373 Instead, we allowed for general fitness landscapes and the whole space of fragmentation modes, in-
374 cluding cases of asymmetric binary division (e.g. the “unicellular propagule” strategy) neglected by
375 Roze and Michod [2001]. Our results indicate that reproduction modes involving unicells often lead
376 to the maximum growth rate even when large group sizes confer fecundity or viability advantages
377 making small propagule sizes to either divide less efficiently or die at a higher rate. In particular,
378 we have shown that if fragmentation is costly, a strategy consisting of a multiple fragmentation mode
379 with propagule size one (i.e., the small propagule strategy studied by Roze and Michod [2001]) can
380 be adaptive for reasons different than the elimination of harmful mutations. Extending our model to

381 allow for mutations giving rise to heterogenous collectives characterised by within-organism conflict
382 or division of labor is a possible avenue of future research that would complement recent theoretical
383 efforts [Rashidi et al., 2015, Kaveh et al., 2016].

384 Closer to our work, Tarnita et al. [2013] investigated the evolution of multicellular life cycles via
385 two alternative routes: "staying together" (whereby offspring cells remain attached to the parent) and
386 "coming together" (whereby cells of different origins aggregate in a group). In particular, they studied
387 the conditions under which a multicellular strategy that grow complexes via staying together can out-
388 perform a solitary strategy whereby cells always separate after division. The way they model group
389 formation and analyze the resulting population dynamics (by means of biological reactions and matrix
390 models) is closely related to our approach; indeed, their solitary strategy is our binary strategy 1+1,
391 while their staying together strategy corresponds to a particular kind of binary stochastic strategy. The
392 questions we ask are however different. Tarnita et al. [2013] were concerned with the conditions under
393 which (multicellular) strategies that form groups can invade and replace (unicellular) strategies that
394 remain solitary; to do so they postulated specific reproductive modes and allowed for multicellular and
395 unicellular strategies to experience different birth and death rates (fitness landscapes). Contrastingly,
396 we aimed to understand what would be, for a common fitness landscape, the optimal reproduction
397 mode out of the vast space of fragmentation strategies comprising all possible deterministic and prob-
398 abilistic pathways by which complexes stay together and split apart. A key finding is that for any
399 fitness landscape, stochastic modes of fragmentation such as the particular staying together strategy
400 considered by Tarnita et al. [2013], will be outperformed by at least one deterministic fragmenta-
401 tion mode. With all the apparent generality of probabilistic reproduction modes, our model predicts
402 that life cycles shaped by natural selection will be characterised by a highly regulated developmental
403 program.

404 Other than diagnostic value, modes of fragmentation in bacteria have received little attention. The
405 theoretical framework developed here could serve as a null hypothesis against which the adaptive sig-
406 nificance of modes of fragmentation can be examined. It is of interest to note that two bacteria that
407 form groups and are well studied from a clinical perspective, *Neisseria gonorrhoeae* and *Staphylo-*
408 *coccus aureus*, both show evidence of deterministic fragmentation by binary fragmentation: *Neisseria*
409 *gonorrhoeae* divide into groups of two equal sizes [Westling-Häggström et al., 1977]. *Staphylococcus*
410 *aureus* divide into one large group plus a unicellular propagule [Koyama et al., 1977]. This leads to

411 questions concerning the nature of the fitness landscape occupied by these bacteria and the basis of
412 any collective level benefit as assumed by our model.

413 Although cell loss (apoptosis) is known in bacteria [Rice and Bayles, 2008], it is not usually, with
414 the exception of cyanobacteria [Rossetti et al., 2011], associated with fragmentation. However, cell loss
415 upon fragmentation occurs in *Volvox carteri*, a member of the volvacine algae, where the outer (soma)
416 cells, while contributing to collective viability, undergo senescence and die following the liberation of
417 the germ line cells [Kochert, 1968, Kirk, 2005]. In our model, such a mode of reproduction is possible
418 in instances where cell loss is associated with fragmentation. Within the volvacine algae, there are
419 species, such as *Gonium pectorale*, that also fragment into multiple propagules in the absence of cell
420 death. Although costs associated with cell loss are not evident, it is nonetheless likely that the process
421 of fragmentation is costly, for example, arising from production of enzymes for the degradation of the
422 cell matrix [Birkendal-Hansen, 1995, Basbaum and Zena, 1996].

423 While the model developed here is conceptually simple, it is readily extended and applied to the
424 study of more complex life cycles, including those involving specialized cell types, such as germ and
425 soma cells as found in many multicellular organisms. Our model is also amenable to exploring the
426 effects of developmental structure on the selective benefit of differing modes of reproduction.

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503 **A Appendix**

504 **A.1 Characteristic equation of a deterministic fragmentation mode**

505 Consider a deterministic fragmentation mode in which a group of size m grows to size $m + 1$ and
 506 fragments according to fragmentation pattern $\kappa \vdash m + 1$. The corresponding projection matrix is an
 507 $m \times m$ matrix of the form

$$\mathbf{A} = \begin{pmatrix} -b_1 - d_1 & 0 & \cdots & 0 & mb_m \pi_1(\kappa) \\ b_1 & -2b_2 - d_2 & 0 & \vdots & mb_m \pi_2(\kappa) \\ 0 & 2b_2 & -3b_3 - d_3 & 0 & mb_m \pi_3(\kappa) \\ 0 & 0 & \ddots & \ddots & \vdots \\ 0 & 0 & \cdots & (m-1)b_{m-1} & mb_m \pi_m(\kappa) - mb_m - d_m \end{pmatrix}.$$

508 The population growth rate is given by the leading eigenvalue λ_1 of \mathbf{A} , i.e., the largest solution of the
509 characteristic equation

$$\det(\mathbf{A} - \lambda\mathbf{I}) = 0. \quad (14)$$

510 By using a Laplace expansion along the last column of $\mathbf{A} - \lambda\mathbf{I}$, we can rewrite the left hand side of
511 the above expression (i.e., the characteristic polynomial of \mathbf{A}) as

$$\begin{aligned} \det(\mathbf{A} - \lambda\mathbf{I}) &= \sum_{i=1}^{m-1} (-1)^{i+m} mb_m \pi_i(\kappa) M_{i,m} + (-1)^{2m} (mb_m \pi_m(\kappa) - mb_m - d_m - \lambda) M_{m,m} \\ &= \sum_{i=1}^m (-1)^{i+m} mb_m \pi_i(\kappa) M_{i,m} - (mb_m + d_m + \lambda) M_{m,m} \end{aligned} \quad (15)$$

512 where $M_{i,m}$ is the (i, m) minor of $\mathbf{A} - \lambda\mathbf{I}$. For all $i = 1, \dots, m$, the minor $M_{i,m}$ is the determi-
513 nant of a block diagonal matrix, and hence equal to the product of the determinants of the diagonal
514 blocks. Moreover, each diagonal block is either a lower triangular or an upper triangular matrix, whose
515 determinant is given by the product of the elements in their main diagonals. We can then write

$$M_{i,m} = \prod_{j=1}^{i-1} (-jb_j - d_j - \lambda) \prod_{j=i}^{m-1} jb_j. \quad (16)$$

516 Substituting Eq. (16) into Eq. (15) and simplifying, we obtain

$$\begin{aligned} \det(\mathbf{A} - \lambda\mathbf{I}) &= (-1)^{m-1} \sum_{i=1}^m mb_m \pi_i(\kappa) \prod_{j=1}^{i-1} (jb_j + d_j + \lambda) \prod_{j=i}^{m-1} jb_j \\ &\quad - (-1)^{m-1} (mb_m + d_m + \lambda) \prod_{j=1}^{m-1} (jb_j + d_j + \lambda) \\ &= (-1)^{m-1} \left[\prod_{j=1}^m jb_j \right] \left(\left[\sum_{i=1}^m \pi_i(\kappa) \prod_{j=1}^{i-1} \left(1 + \frac{d_j + \lambda}{jb_j} \right) \right] - \prod_{j=1}^m \left(1 + \frac{d_j + \lambda}{jb_j} \right) \right). \end{aligned}$$

517 Replacing this expression into the characteristic equation (14), dividing both sides by $(-1)^m \prod_{j=1}^m jb_j$,
518 and simplifying, we finally obtain that the characteristic equation (14) can be written as

$$F_{m+1}(\lambda) - \sum_{i=1}^m \pi_i(\kappa) F_i(\lambda) = 0, \quad (17)$$

519 where

$$F_i(\lambda) = \prod_{j=1}^{i-1} \left(1 + \frac{d_j + \lambda}{jb_j} \right). \quad (18)$$

520 Note that two transformations preserve the solution of Eq. (17)

$$\mathbf{d} \rightarrow \mathbf{d} - r, \quad \lambda \rightarrow \lambda + r, \quad r \leq \min(\mathbf{d}),$$

521 and

$$\mathbf{d} \rightarrow s\mathbf{d}, \quad \mathbf{b} \rightarrow s\mathbf{b}, \quad \lambda \rightarrow s\lambda, \quad s > 0.$$

522 These transformations allow us to set $b_1 = 1$ and $\min(\mathbf{d}) = 0$ without loss of generality.

523 A.2 Mixing between 1+1 and 2+1 is dominated

524 To show that the life cycle mixing between fragmentation modes 1+1 and 2+1 with probability q and
 525 represented by Eq. (2) is dominated, consider its growth rate λ_1^q as a function of q , as given by Eq.
 526 (10). We have $\lambda_1^q(0) = \lambda_1^{1+1}$ and $\lambda_1^q(1) = \lambda_1^{2+1}$. A sufficient condition for q to be dominated by either
 527 1+1 or 2+1 is then that $\lambda_1^q(q)$ is monotonic in q . To show that this is the case, note that the derivative
 528 of λ_1^q with respect to q is given by

$$\frac{d\lambda_1^q}{dq} = b_1 \left(-1 + \frac{(2q-1)b_1 + 2b_2 + d_1 - d_2}{\sqrt{((2q-1)b_1 + d_1 + d_2)^2 + 4b_1(2qb_2 - (2q-1)d_2) - 4d_1d_2}} \right),$$

529 and that such expression is equal to zero if and only if

$$b_1 - b_2 = d_1 - d_2 \tag{19}$$

530 which is independent of q . It follows that λ_1^q is either nonincreasing or nondecreasing in q , and hence
 531 that it attains its maximum at either $q = 0$, $q = 1$, or (when (19) is satisfied) at any $q \in [0, 1]$. Hence,
 532 q is dominated by either 1+1 or 2+1.

533 A.3 Stochastic fragmentation modes are dominated

534 For any fitness landscapes, stochastic fragmentation modes are dominated by at least one deterministic
 535 mode. In other words, the optimal life cycle is deterministic. To prove this, consider the set of
 536 partitions $\kappa \vdash j$ for a given j , fix the probabilities of fragmentation patterns $\nu \vdash i \neq j$ to arbitrary
 537 values, and focus attention on the function

$$\lambda_1^j : S_j \rightarrow \mathbb{R},$$

538 mapping probability distributions in the ζ_j -simplex $S_j \subset \mathbb{R}^{\zeta_j}$ (specifying the probabilities of all
 539 partitions $\kappa \vdash j$) to the dominant eigenvalue λ_1^j of the associated projection matrix \mathbf{A} . Our goal is to
 540 show that, for any j , λ_1^j is a quasiconvex function, i.e., that

$$\lambda_1^j(\eta\mathbf{x}_1 + (1-\eta)\mathbf{x}_2) \leq \max \left\{ \lambda_1^j(\mathbf{x}_1), \lambda_1^j(\mathbf{x}_2) \right\}$$

541 holds for all $\mathbf{x}_1, \mathbf{x}_2 \in S_j$ and $\eta \in [0, 1]$. Quasiconvexity of λ_1^j implies that λ_1^j achieves its maximum at
 542 an extreme point of S_j , i.e., at a probability distribution that puts all of its mass in a single fragmenta-
 543 tion pattern. Quasiconvexity of λ_1^j for all j then implies that the maximum growth rate λ_1 is achieved
 544 by a deterministic fragmentation mode, and that stochastic fragmentation modes are dominated.

545 To show that λ_1^j is quasiconvex, we restrict the function to an arbitrary line and check quasicon-
 546 vexity of the resulting scalar function [Boyd and Vandenberghe, 2004, p. 99]. More precisely, we aim
 547 to show that the function

$$\ell(t) = \lambda_1^j(\mathbf{u} + t\mathbf{v}),$$

548 is quasiconvex in t for any $\mathbf{u} \in S_j$ and $\mathbf{v} \in \mathbb{R}^{\zeta_j}$ such that $\mathbf{u} + t\mathbf{v} \in S_j$. We hence need to verify that

$$\ell(\tau t_1 + (1 - \tau)t_2) \leq \max\{\ell(t_1), \ell(t_2)\} \quad (20)$$

549 holds for $\tau \in [0, 1]$.

550 To show this, note that the function $\ell(t) = \lambda_1^j(\mathbf{u} + t\mathbf{v})$ is given implicitly as the largest root of the
 551 characteristic polynomial

$$p(\lambda) = \det(\mathbf{A} - \lambda\mathbf{I}), \quad (21)$$

552 where the probabilities of fragmentation specified by $\mathbf{u} + t\mathbf{v}$ appear in the $(j - 1)$ -th column of the
 553 projection matrix \mathbf{A} (see Eq. (8)).

554 The right hand side of Eq. (21) can be written using a Laplace expansion along the $(j - 1)$ -th
 555 column of $\mathbf{A} - \lambda\mathbf{I}$, i.e.,

$$\det(\mathbf{A} - \lambda\mathbf{I}) = \sum_{i=0}^{n-1} (-1)^{i+j-1} (a_{i,j-1} - \delta_{i,j-1}\lambda) M_{i,j-1}, \quad (22)$$

556 where $\delta_{i,j-1}$ is the Kronecker delta and $M_{i,j-1}$ is the $(i, j - 1)$ minor of \mathbf{A} , i.e., the determinant of
 557 the submatrix obtained from \mathbf{A} by deleting the i -th row and $(j - 1)$ -th column. Each minor $M_{i,j-1}$
 558 is independent of t because the only entries of \mathbf{A} that depend on t appear in the $(j - 1)$ -th column.
 559 Moreover, each entry $a_{i,j-1}$ is either zero or a linear function of t . Hence, $p(\lambda)$ is a polynomial on λ
 560 with coefficients that are linear in t , i.e., of the form

$$p(\lambda) = \sum_{k=0}^{n-1} (\alpha_k + \beta_k t) \lambda^k, \quad (23)$$

561 for some α_k, β_k . Moreover, since the leading coefficient must be $(-1)^{n-1}$ (the matrix \mathbf{A} is $(n - 1) \times$
 562 $(n - 1)$), it follows that $\alpha_{n-1} = (-1)^{n-1}$ and $\beta_{n-1} = 0$.

563 Denote by $p_\tau(\lambda)$, $p_1(\lambda)$, and $p_2(\lambda)$ the characteristic polynomials corresponding to, respectively,
 564 the probability distributions given by $\mathbf{u} + [\tau t_1 + (1 - \tau)t_2] \mathbf{v}$, $\mathbf{u} + t_1 \mathbf{v}$, and $\mathbf{u} + t_2 \mathbf{v}$. From Eq. (23),
 565 these are given by

$$p_\tau(\lambda) = \sum_{k=0}^{n-1} (\alpha_k + \beta_k [\tau t_1 + (1 - \tau)t_2]) \lambda^k = \sum_{k=0}^{n-1} \alpha_k \lambda^k + [\tau t_1 + (1 - \tau)t_2] \sum_{k=0}^{n-1} \beta_k \lambda^k \quad (24a)$$

$$p_1(\lambda) = \sum_{k=0}^{n-1} (\alpha_k + \beta_k t_1) \lambda^k = \sum_{k=0}^{n-1} \alpha_k \lambda^k + t_1 \sum_{k=0}^{n-1} \beta_k \lambda^k \quad (24b)$$

$$p_2(\lambda) = \sum_{k=0}^{n-1} (\alpha_k + \beta_k t_2) \lambda^k = \sum_{k=0}^{n-1} \alpha_k \lambda^k + t_2 \sum_{k=0}^{n-1} \beta_k \lambda^k \quad (24c)$$

566 Subtracting Eq. (24b) from Eq. (24a), and Eq. (24c) from Eq. (24a), we can write

$$p_\tau(\lambda) - p_1(\lambda) = (t_2 - t_1)(1 - \tau) \sum_{k=0}^{n-1} \beta_k \lambda^k,$$

$$p_\tau(\lambda) - p_2(\lambda) = (t_1 - t_2)\tau \sum_{k=0}^{n-1} \beta_k \lambda^k.$$

567 Note that the signs of these differences are always different, i.e., either (i) $p_\tau(\lambda) - p_1(\lambda) \geq 0$ and
 568 $p_\tau(\lambda) - p_2(\lambda) \leq 0$, or (ii) $p_\tau(\lambda) - p_1(\lambda) \leq 0$ and $p_\tau(\lambda) - p_2(\lambda) \geq 0$. In the first case, we have
 569 $p_1(\lambda) \leq p_\tau(\lambda) \leq p_2(\lambda)$ and in the second we have $p_2(\lambda) \leq p_\tau(\lambda) \leq p_1(\lambda)$, i.e., for each λ , $p_\tau(\lambda)$
 570 lies between $p_1(\lambda)$ and $p_2(\lambda)$, or, equivalently

$$p_\tau(\lambda) \leq \max \{p_1(\lambda), p_2(\lambda)\}, \quad (25)$$

571 for all λ . Since λ_1^j is the largest root of $p(\lambda)$, and since $p_\tau(\lambda)$, $p_1(\lambda)$, and $p_2(\lambda)$ all have the same
 572 sign in the limit when λ tends to infinity (their leading coefficients are all equal to $\alpha_{n-1} = (-1)^{n-1}$),
 573 condition (25) obviously implies condition (20), thus proving our claim. See Fig. 8 for an illustration.

574 **A.4 Non-binary fragmentation modes are dominated by binary fragmentation modes**

575 For any fitness landscape, binary group splitting achieves a larger growth rate than splitting into more
 576 than two offspring groups. To prove this, consider positive integers m, j, k such that $m > j + k$, an
 577 arbitrary partition $\tau \vdash m - (j + k)$, and the following three deterministic fragmentation modes:

- 578 1. $\kappa_1 = j + k + \tau \vdash m$, whereby a complex of size m fragments into one complex of size j , one
 579 complex of size k and a number of offspring complexes given by partition τ .

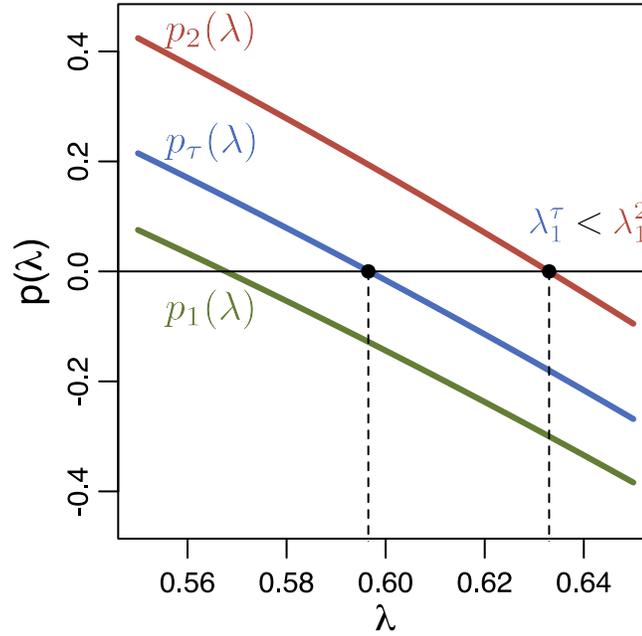


Figure 8: **Population growth rate λ_1 is quasiconvex.** Consider two fragmentation modes \mathbf{q}_1 and \mathbf{q}_2 which differ only in the probabilities of fragmentation patterns at a single size j . Then, for any $0 \leq \tau \leq 1$ and corresponding fragmentation mode $\mathbf{q}_\tau = \tau\mathbf{q}_1 + (1 - \tau)\mathbf{q}_2$, the polynomials $p(\lambda)$ given by Eq. (21) satisfy either $p_1(\lambda) \leq p_\tau(\lambda) \leq p_2(\lambda)$ or $p_2(\lambda) \leq p_\tau(\lambda) \leq p_1(\lambda)$. Thus, \mathbf{q}_τ leads to a lower growth rate than either \mathbf{q}_1 or \mathbf{q}_2 , i.e., either $\lambda_1^\tau \leq \lambda_1^1$, or $\lambda_1^\tau \leq \lambda_1^2$ holds. Here, $j = 3$, $\mathbf{q}_1 = \{(0.9, 0.1), (0.5, 0.5, 0), (0, 0, 0, 1, 0)\}$, $\mathbf{q}_2 = \{(0.9, 0.1), (0.5, 0, 0.5), (0, 0, 0, 1, 0)\}$, and $\tau = 0.6$. Note that the life cycle corresponding to \mathbf{q}_τ is schematically illustrated in Fig. 2b. The fitness landscape is given by $b_i = 1/i$, $d_i = 0$ for all i .

580 2. $\kappa_2 = (j + k) + \tau \vdash m$, whereby a complex of size m fragments into one complex of size $j + k$
 581 and a number of offspring complexes given by partition τ .

582 3. $\kappa_3 = j + k \vdash (j + k)$, a binary splitting fragmentation mode whereby a complex of size $j + k$
 583 fragments into two offspring complexes: one of size j and one of size k .

584 Fragmentation mode κ_1 leads to a number of offspring groups equal to

$$n_1 = \sum_{\ell=1}^{m-k-j} \pi_{\ell}(\tau) + 2,$$

585 fragmentation mode κ_2 to a number of offspring groups equal to

$$n_2 = \sum_{\ell=1}^{m-k-j} \pi_{\ell}(\tau) + 1 = n_1 - 1,$$

586 and fragmentation mode κ_3 to a number of offspring groups equal to two. Denoting by λ_1^i the leading
 587 eigenvalue of the projection matrix induced by fragmentation mode κ_i , we can show that, for any
 588 fitness landscape, either $\lambda_1^1 \leq \lambda_1^2$ or $\lambda_1^1 \leq \lambda_1^3$ holds. This means that a fragmentation mode with more
 589 than two parts is dominated by either a fragmentation mode with one part less or by a fragmentation
 590 mode with exactly two parts. By induction, this implies that, for any fitness landscape, the optimal
 591 fragmentation mode is always one within the class of binary splitting strategies.

592 To prove the statement above, let us define the polynomial $p_i(\lambda)$ as the left hand side of Eq. (17)

593 with $\kappa = \kappa_i$, so that λ_1^i is the largest root of $p_i(\lambda)$. We obtain

$$p_1(\lambda) = F_m(\lambda) - \sum_{\ell=1}^{m-j-k} \pi_{\ell}(\tau) F_{\ell}(\lambda) - F_j(\lambda) - F_k(\lambda) \quad (26a)$$

$$p_2(\lambda) = F_m(\lambda) - \sum_{\ell=1}^{m-j-k} \pi_{\ell}(\tau) F_{\ell}(\lambda) - F_{j+k}(\lambda) \quad (26b)$$

$$p_3(\lambda) = F_{j+k}(\lambda) - F_j(\lambda) - F_k(\lambda). \quad (26c)$$

594 These polynomials satisfy the following two properties. First,

$$\lim_{\lambda \rightarrow \infty} p_i(\lambda) = \infty, \quad (27)$$

595 as the leading coefficient of the left hand side of Eq. (17) is given by $1/(n!b_1 \dots b_n)$, which is always
 596 positive. Second,

$$p_1(\lambda) = p_2(\lambda) + p_3(\lambda). \quad (28)$$

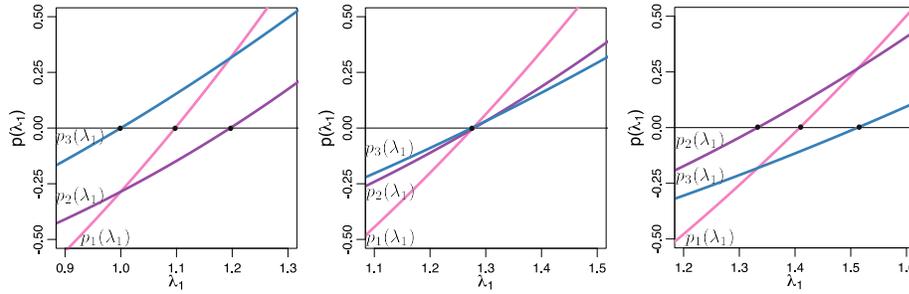


Figure 9: The population growth rate induced by a fragmentation mode with more than two offspring groups is weakly dominated. Consider the characteristic polynomials $p_i(\lambda_1)$ for partitions $\kappa_1 = 2 + 1 + 1$, $\kappa_2 = 3 + 1$ and $\kappa_3 = 2 + 1$. *Left:* Fitness landscape $\mathbf{b} = (1, 1, 1.4)$, $\mathbf{d} = (0, 0, 0)$. Since $p_2(\lambda_1^1) < 0$, κ_1 is dominated by κ_2 ($\lambda_1^1 < \lambda_1^2$ holds). *Center:* Fitness landscape $\mathbf{b} = (1, 2.6 - \sqrt{1.3}, 1.4)$, $\mathbf{d} = (0, 0, 0)$. Since $p_1(\lambda_1^1) = p_1(\lambda_1^2) = p_1(\lambda_1^3)$, κ_1 is weakly dominated by κ_2 ($\lambda_1^1 \leq \lambda_1^2$ holds). *Right:* Fitness landscape $\mathbf{b} = (1, 1.9, 1.4)$, $\mathbf{d} = (0, 0, 0)$. Since $p_3(\lambda_1^1) < 0$, κ_1 is dominated by κ_3 ($\lambda_1^1 < \lambda_1^3$ holds).

597 Evaluating Eq. (28) at λ_1^1 , and since $p_1(\lambda_1^1) = 0$, it then follows that

$$p_2(\lambda_1^1) = -p_3(\lambda_1^1).$$

598 Hence, only one of the following three scenarios is satisfied: (i) $p_2(\lambda_1^1) < 0 < p_3(\lambda_1^1)$, (ii) $p_2(\lambda_1^1) =$
 599 $p_3(\lambda_1^1) = 0$, or (iii) $p_2(\lambda_1^1) > 0 > p_3(\lambda_1^1)$. If $p_2(\lambda_1^1) < 0 < p_3(\lambda_1^1)$, and by Eq. (27) and Bolzano's
 600 theorem, $\lambda_1^1 \leq \lambda_1^2$ holds. Likewise, if $p_2(\lambda_1^1) > 0 > p_3(\lambda_1^1)$, then $\lambda_1^1 \leq \lambda_1^3$ holds. Finally, if
 601 $p_2(\lambda_1^1) = p_3(\lambda_1^1) = 0$, then both $\lambda_1^1 \leq \lambda_1^2$ and $\lambda_1^1 \leq \lambda_1^3$ hold. We conclude that either $\lambda_1^1 \leq \lambda_1^2$ or
 602 $\lambda_1^1 \leq \lambda_1^3$ must hold. See Fig. 9 for an illustration.

603 A.5 Optimality maps for $n = 4$

604 For $n = 4$ there are four deterministic fragmentation modes, denoted by their fragmentation patterns
 605 1+1, 2+1, 2+2, and 3+1. From Eq. (11), their characteristic polynomials are given by

$$1 + 1 : p_{1+1}(\lambda) = F_2(\lambda) - 2F_1(\lambda), \quad (29a)$$

$$2 + 1 : p_{2+1}(\lambda) = F_3(\lambda) - F_2(\lambda) - F_1(\lambda), \quad (29b)$$

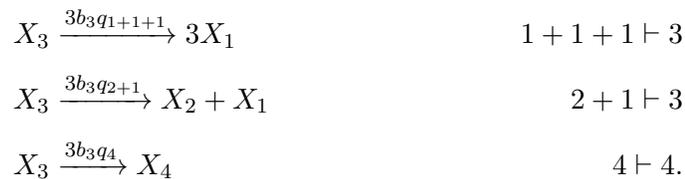
$$2 + 2 : p_{2+2}(\lambda) = F_4(\lambda) - 2F_2(\lambda), \quad (29c)$$

$$3 + 1 : p_{3+1}(\lambda) = F_4(\lambda) - F_3(\lambda) - F_1(\lambda). \quad (29d)$$

606 The optimality maps shown in Fig. 4 were obtained by computing numerically the largest root
 607 of the characteristic polynomials and comparing such different values for birth rates (fecundity land-
 608 scapes) or death rates (viability landscapes). For fecundity landscapes, we tested fitness landscapes of
 609 the form $\{\mathbf{b}, \mathbf{d}\} = \{(1, b_2, b_3), (0, 0, 0)\}$ and values b_2 and b_3 taken from a rectangular grid of size
 610 300 by 300 with $b_2 \in [0, 5]$ and $b_3 \in [0, 5]$. For viability landscapes, we tested fitness landscapes of
 611 the form $\{\mathbf{b}, \mathbf{d}\} = \{(1, 1, 1), (5, d_2, d_3)\}$ and values d_2 and d_3 taken from a rectangular grid of size
 612 300 by 300 with $d_2 \in [0, 10]$ and $d_3 \in [0, 10]$.

613 A.6 Costly fragmentation

614 For costly fragmentation, one cell is lost upon the fragmentation event. In this case the biological
 615 reactions are still given by Eqs. (4) and (5). However, under costly fragmentation the sum of sizes
 616 of offspring groups is one less than the size of parent group. Therefore, in the reaction describing
 617 the group splitting, κ is a partition of j (and not of $j + 1$ as it was under the costless fragmentation).
 618 Note, that in the reaction describing group growth, κ is still a trivial partition of $j + 1$. Thus, for costly
 619 fragmentation the set of available outcomes of growth of a group of size j is given by all partitions of
 620 j having at least two parts and the trivial partition of $j + 1$. For instance, the reactions modeling the
 621 birth of units and the growing and fragmentation of groups of size 3 are:



622 The combined probability of all outcomes of aggregate growth must be equal to one. In the case
 623 of costless fragmentation, this condition has been given by $\sum_{\kappa \vdash j+1} q_\kappa = 1$ for $j = 1, \dots, n - 1$.
 624 For costly fragmentation this condition changes to $\sum_{\kappa \vdash j'} q_\kappa = 1$ for $j = 1, \dots, n - 1$, where $j' =$
 625 $j \setminus j \cup (j + 1)$ denotes the set of partitions of j with at least two parts together with the trivial partition
 626 $(j + 1)$. The analogs of Eqs. (6) and (8) are changed accordingly.

627 Finally, note that for deterministic fragmentation mode, the characteristic equation is still the one
 628 derived in Appendix A.1 with the sole exception that, in Eq. (17), $\kappa \vdash m$ (rather than $\kappa \vdash m + 1$ as it
 629 was the case for costless fragmentation).