Sib cannibalism can be adaptive for kin

József Garay

MTA-ELTE Theoretical Biology and Evolutionary Ecology Research Group and Department of Plant Systematics, Ecology and Theoretical Biology,

L. Eötvös University, Pázmány P. sétány 1/C

H-1117 Budapest, Hungary (garayj@ludens.elte.hu)

Zoltán Varga

Institute of Mathematics and Informatics,

Szent István University, Páter K. u. 1.

H-2103, Gödöllő, Hungary (Varga.Zoltan@gek.szie.hu)

Manuel Gámez

Center for Agribusiness Biotechnology Research,

Almeria University, Ctra. Sacramento s/n,

ES-04120 Almeria, Spain (mgamez@ual.es)

Tomas Cabello

Center for Agribusiness Biotechnology Research,

Almeria University, Ctra. Sacramento s/n,

ES-04120 Almeria, Spain (tcabello@ual.es)

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Abstract

Sib cannibalism seems to be paradoxical behaviour, since it decreases the survival rate of the closest relative juveniles, so the rate of sib cannibalism changes the demography of the cannibal phenotype. In the general kin demographic selection model presented here, the long-term growth rate of a phenotype is determined by a Leslie matrix that depends on the life history strategy, and a uniform density-dependent selection process takes place, keeping the total population size at the level of the carrying capacity. Using this model, where different phenotypes are described by different Leslie matrices, we point out that the phenotype optimizing the phenotypic long-term growth rate will select out any other optimizing phenotype. We find that sib cannibalism is adaptive if the sib cannibal can decrease its developmental time, and the shorter development time can increase the rate of survival from sib cannibal juvenile to adult, and also when sib cannibalism increases fecundity in the adult stage. Cannibalism between the closest relatives can be considered as a mutualistic kin strategy when the benefit of cannibalism is greater than the cost of it.

Keywords: collector hypothesis, kin demography, Leslie model

1. Introduction

Cannibalism is surprisingly frequent in nature (Fox 1975; Polis 1981). Polis (1981) asked the question: "*Why is cannibalism relatively common?"* To get a possible answer to this problem, we will concentrate on the energetic and/or nutritional benefit (Manica 2002) of cannibalism. Our starting point comes from Richardson *et al*. (2010): in the case of non-carnivorous cannibal insects, *"Cannibalism can be adaptive by improving growth rate, survivorship, vigor, longevity, and fecundity"*. Our aim is to get some classical Darwinian insight into why cannibalism can win in the struggle for existence.

Cannibalism has at least two evolutionary advantages. The first advantage is the gain in energy and/or nutrition, in which cannibalism is similar to predation. The second advantage is that, if a cannibal phenotype can mainly eat non-relative conspecific (Bonsall & Klug 2011; Parsons *et al*. 2013), this strategy increases the relative advantage over a non-cannibal phenotype, in a way that is similar to Hamiltonian spite (Hamilton 1970; Garay & Móri 2011). What is more, the positive synergistic interference of these two advantages should radically increase the selection advantage of cannibalism. We note that cannibalistic snails are unable to distinguish between sib, other kin and non-kin eggs (Baur 1987; *cf*. Fea *et al.* 2014). Thus the question arises of whether, without the second advantage, cannibalism can be adaptive. From an evolutionary point of view, cannibalism within the closest relatives (*cf*. Hamilton 1964) seems paradoxical behaviour at first glance; for example, sib cannibalism directly decreases the number of sibs, and thus also the number of cannibalism genes. However, is this really the case in all possible selection situations?

For the analysis of the two advantages of cannibalism described above, there are two main classes of mathematical descriptions of cannibalism. The first of these uses differential equations (Argasinski & Broom 2014; Bonsall & Klug 2011; Dennis *et al*. 2001; Klug &

Bonsall 2007). This method can easily deal with the density-dependent interaction between cannibal and non-cannibal phenotypes. The second class uses matrix models (Skurdal *et al*. 1985; Stenseth 1985), which are a kind of modified version of the classical Leslie model. Our model belongs to the second class, since we will not consider the interaction between phenotypes.

For many arthropods, cannibalism is a normal phenomenon, not an anomaly. It has been documented in many insect orders, including *Odonata, Orthoptera, Thysanoptera, Hemiptera, Trichoptera, Lepidoptera, Diptera, Neuroptera, Coleoptera* and *Hymenoptera*. It occurs among predatory species and herbivores, and involves predation by the mobile adults and larvae or nymphs on each other and on immobile eggs and pupae (Capinera 2008; Richardson *et al*. 2010). Our examples are inspired by the knowledge about insects (and in the general reasoning we shall also use terms related to insects), but we think that the demonstrated effects could work for other species, too.

We consider a species with non-reproductive juvenile and reproductive adult stages with overlapping generations, where the females lay more eggs than the carrying capacity. More precisely, the density resulting from oviposition together with the intrinsic (selection-independent) survival exceeds the carrying capacity. When interaction between different phenotypes takes place, we have to consider density- or frequency-dependent interactions, which makes the calculation harder. Furthermore, we do not have enough information to model the genetic background of cannibalism, and thus we consider an asexual model. Thus our simplifying assumption is that *there is only cannibalistic interaction between the offspring of a female, the population is asexual and the intensity of cannibalism as the strategy of a phenotype is a hereditary trait*. We consider only sib cannibalism (the closest relative juveniles eat each other), and our results are based on the following:

Collector Hypothesis. Consider the case when each individual collects nutrients alone, and the accumulated nutrient mass determines the speed of development and the individual survival rate (*cf*. Joyner & Gould 1987; Capinera 2008; Santana *et al*. 2012). Nutrients are limited: the collection of nutrients is a long process and/or the food is imperfect, meaning that individuals are malnourished, and the collection of the quantity of the limited nutrients necessary for development takes a long time. Our basic idea is that, in this selection situation, sib cannibalism can be considered as an effective "feeding strategy" for the cannibal phenotype, in the sense that the cannibalized juvenile has "collected nutrients" in its body for its surviving sibling. The Collector Hypothesis is based on the following effects (see e.g. Richardson *et al.* 2010):

a) Accelerating effect on development. If the collection of energy and nutrients sufficient for development takes a long time, and the development strictly depends on the consumed nutrient mass, then sib cannibalism can increase the speed of nutrient collection for development. The development time is therefore decreased by sib cannibalism (*cf*. Duelli 1981; Joyner & Gould 1985; Klug & Bonsall 2007; Michaud 2003).

b) Survival effect on development. When adult females deposit more eggs than the carrying capacity, sib cannibalism can paradoxically increase the total survival rate under the following conditions. If, without cannibalism, the survival rate is very small, then sib cannibalism can increase the survival rate on the part of the siblings, and, finally, the number of surviving cannibal siblings can be higher than that of the surviving non-cannibal siblings. The survival rate of the sib cannibal phenotype can increase for the following three reasons. First, they grow more rapidly (Santana *et al*. 2012), and thus the most risky juvenile stage is shorter (*cf*. polyphenic cannibalism in tadpoles). Secondly, they have more nutrients, because the nutrients of siblings that either starved to death or became injured or infected by parasites

would be lost without cannibalism (*cf*. Storz 2004). Thirdly, a reduction in the local density of siblings reduces the liberation of odour hints (semiochemicals), possible predation and the risk of parasitism (Chapman *et al*. 1999, 2000; Guerra-Sanz & Cabello 2008).

c) Effect on fecundity. In insects there are examples for which sib cannibalism between juveniles can increase the fecundity of the surviving sib cannibal nymphs during their adult stage (*cf*. Duelli 1981; Al-Zubaidi & Capinera 1983).

In order to model the dynamics of cannibalism appropriately, we worked out a general kin demographic selection model (as explained in Section 2) that can be used to find out which phenotype will win in the struggle for life. Using this model, in Section 3, we give theoretical examples of when sib cannibalism is evolutionarily successful. We finish the paper with our conclusions.

2. Kin demographic selection model

Here we introduce a general kin demographic selection model, based on the following *Basic Assumptions*: (a) different phenotypes, determined by hereditary life history strategies, do not interact: there are interactions only within the kin (such as between a female and its offspring); (b) the survival rate depending on total density is the same for all individuals, independently of their phenotype and age; and (c) the interactions within the kin and the density-dependent survival process are independent.

The new kin demographic selection model is a combination of the following two sub-models. In the first sub-model the non-interacting females produce offspring, and the interactions (determining the demographic parameters of the different phenotypes) take place only within the kin – for example, between the offspring of the same given single female. Here, the main point is that the phenotype-dependent demographic parameters (the fecundities and survivals

of the Leslie model) determine the next age-classified state vector of the phenotype. In the second sub-model, a random survival process reduces the total population size to the fixed value of the carrying capacity. Here, the main point is that the density-dependent survival process has a uniform effect on the demographic parameters of all individuals of all phenotypes. This condition is reasonable for our modelling methodology if we want to see the effect of the interaction within the considered kin on the demography. Indeed, selection depending on phenotype or age class might mask the effect we are discussing.

Using Basic Assumption (c) we can combine the above two sub-models in the kin demographic selection model, which, although the "intrinsic population growth" and the selection occur in two independent "steps", can be considered as a particular density-dependent limited growth model where the demographic parameters depend uniformly on the total density of the system.

2.1. Sub-model 1: Life history strategy dependent Leslie model

First we consider a general model for optimization by life history trait. We start from the classical Leslie model, considering a species where age structure and phenotypes are different only in their life history traits. Under our Basic Assumptions (a)-(c), the different phenotypes have different, not density-dependent Leslie matrices that depend on the life history trait. Thus we consider a Leslie matrix for each phenotype, where both the survival rates $0 < \omega_i(s) \leq 1$ and the fecundities $\alpha_i(s) \geq 0$ depend on the phenotype's life history trait, parametrized by $0 \leq s \leq 1$:

$$L(s)=\begin{pmatrix} \alpha_1(s) & \alpha_2(s) & \dots & \alpha_{n-1}(s) & \alpha_n(s) \\ \omega_1(s) & 0 & \dots & 0 & 0 \\ 0 & \omega_2(s) & & \dots & \dots \\ \dots & & \dots & 0 & 0 \\ 0 & \dots & 0 & \omega_{n-1}(s) & 0 \end{pmatrix}. \quad (1)$$

Observe that the model parameters depend both on the age i (e.g. Metcalf & Pavard 2007; Proaktor *et al.* 2007) and the life history trait s, at the same time. We emphasize that in this general framework a number of different life history traits (e.g. timing of reproduction, resource allocation, offspring care, cannibalism only within the kin, etc.) can be investigated.

Now the question arises of what is the appropriate mathematical definition of fitness (*cf.* Brommer 2000; Brommer *et al.* 2002; Caswell 1982, 2001; Schaffer 1974; Stenseth 1985) in the Leslie model. Selection takes place on different phenotypes. Thus, we have to point out first that different fitness functions, in general, can define different phenotypes. For this purpose we concentrate now on two phenotypes defined by the two different "objective functions", phenotypic long-term growth rate and life reproductive success.

Phenotype A maximizes the phenotypic long-term growth rate. It is well-known that in the Leslie model the phenotypic long-term growth rate is equal to the unique positive dominant eigenvalue of the Leslie matrix (see e.g. Caswell 2001), which is the unique positive solution of the following characteristic equation of the above Leslie matrix:

$$P(\lambda)=\lambda^n-\alpha_1(s)\lambda^{n-1}-\alpha_2(s)\omega_1(s)\lambda^{n-2}-\dots-\alpha_n(s)\omega_1(s)\dots\omega_{n-1}(s)=0 \quad (2)$$

Clearly, for all fixed s equation (2) has a unique positive solution $\lambda(s)$. Thus phenotype A has a trait s^* that maximizes the phenotypic long-term growth rate; formally,

$$\lambda^*=\lambda(s^*)=\max_s \lambda(s). \quad (3)$$

Phenotype B maximizes life reproductive success (the average number of offspring of a given individual during its life), given by:

$$R(s) = \alpha_1(s) + \alpha_2(s)\omega_1(s) + \alpha_3(s)\omega_1(s)\omega_2(s) + \ldots + \alpha_n(s)\omega_1(s)\ldots\omega_{n-1}(s). \quad (4)$$

So phenotype B has a trait $\hat{s}$ that maximizes life reproductive success,

$$R(\hat{s}) := \max_s R(s). \quad (5)$$

Observe that the long-term growth rate of phenotype A is not less than that of phenotype B, formally, $\lambda(\hat{s}) \le \lambda(s^*)$.

Now the question arises of whether phenotypes A and B are different. In other words, do these two "objective functions" (phenotypic long-term growth rate and life reproductive success) take their maximum at the same s? The answer is that sometimes they do, and sometimes they do not, as can be seen from the following two Remarks. We emphasize that Remarks 1 and 2 have no direct connection with cannibalism.

Remark 1. Sometimes phenotypes A and B are the same. Consider the case where there is only one fertile age class, so the s-dependent Leslie matrix has the following form:

$$\begin{pmatrix} 0 & \ldots & 0 & \alpha_n(s) \\ \omega_1(s) & 0 & \ldots & 0 \\ 0 & \ldots & 0 & 0 \\ .. & 0 & \omega_{n-1}(s) & 0 \end{pmatrix}.$$

Here the individual life reproductive success is

$$R(s) = \alpha_n(s)\prod_{1}^{n-1}\omega_i(s),$$

and the phenotypic long-term growth rate is

$$\lambda(s) = \sqrt[n]{\alpha_n(s)\prod_{i=1}^{n-1}\omega_i(s)}.$$

In this case, our two objective functions (R and λ) take their maximum at the same s, since $\sqrt[n]{x}$ strictly increases with x. The octopus and many insects are examples belonging to this category.

Remark 2. In general, the above two objective functions take their maximum at different s. To see this, let us consider the following s-dependent Leslie matrix

$$\begin{pmatrix} 1+s & 1-s & 1-s \\ 1-s & 0 & 0 \\ 0 & 1 & 0 \end{pmatrix}.$$

Observe that this Leslie matrix describes a theoretical situation where there are two trade-offs, the first between fecundity and longevity, and the second between early and late reproduction.

Consider first phenotype B that maximizes life reproductive success, that is:

$$R(s) = 2s^2 - 3s + 3,$$

with maximum 3 at $\hat{s} = 0$ (see Figure 1). Thus phenotype B (with $\hat{s} = 0$) lives for three time units, producing one offspring during each time unit, the life reproductive success is 3, and phenotype B has the following Leslie matrix:

$$\begin{pmatrix} 1 & 1 & 1 \\ 1 & 0 & 0 \\ 0 & 1 & 0 \end{pmatrix},$$

with phenotypic long-term growth rate of 1.83.

Now, to check phenotype A, we consider the characteristic equation

$$P(\lambda) = \lambda^3 - (1+s)\lambda^2 - (1-s)^2\lambda - (1-s)^2 = 0.$$

The phenotypic long-term growth rate takes its maximum at $s^* = 1$ (see Figure 1). Phenotype A (with $s^* = 1$) lives for only one time unit and produces two offspring, so the phenotypic long-term growth rate is also 2.

Figure 1.

To see the difference in growth rate between phenotypes A and B, see Figure 2.

Figure 2.

The most important lesson from Remark 2 is that life reproductive success takes its maximum at $\hat{s} = 0$, while phenotypic long-term growth rate takes its maximum at $s^* = 1$. Thus the fitness concept is crucial in our situation, since different fitness concepts give quite different predictions.

At the end of this subsection we note that, if there is no limit to carrying capacity, then any s phenotype with $\lambda(s)>1$ would grow exponentially in the long run. We introduce the carrying capacity in the subsection below.

2.2. Sub-model 2: Selection model for uniform density-dependent survival according to carrying capacity

Let us consider phenotypes A and B, which have different strategies that determine the demographic parameters of the phenotype, implying different phenotypic long-term growth rates. According to the original Darwinian view, we need some density-dependent selection to keep the total density of these two phenotypes at the carrying capacity. Since in the selection situation under consideration (see Basic Assumptions (a)-(c)), there is no interaction between the phenotypes, and we are assuming that the phenotypes differ only in their demographic parameters, we can suppose that there is a uniform survival process, that is, that the survival rate corresponding to the carrying capacity is the same for all individuals. Now the question arises of which phenotype will win in the struggle for existence over the long selection time scale (*cf*. Garay & Varga 2005).

Let us suppose that phenotypes A and B develop according to Leslie models having the respective population vectors $x(t)$, $y(t)$, and matrices L_1, L_2, total densities $\|x(t)\| = \sum_i x_i(t)$, $\|y(t)\| = \sum_i y_i(t)$, and phenotypic long-term growth rates λ_1 and λ_2 with $\lambda_1, \lambda_2 > 1$ and $\lambda_1 > \lambda_2$. Then the relative frequency of phenotype B tends to zero, as is shown below.

Indeed, let us suppose that the subpopulations start from initial states $x(0)$ and $y(0)$, respectively, and that the time unit is chosen in such a way that in unit time the total density of the system always exceeds the carrying capacity K, in particular

$$\|L_1 x(0)\| + \|L_2 y(0)\| > K.$$

Now, by selection, the total density of the system reduces to K proportionally:

$$x(1) = \frac{K}{\|L_1 x(0)\| + \|L_2 y(0)\|} L_1 x(0),$$

$$y(1) = \frac{K}{\|L_1 x(0)\| + \|L_2 y(0)\|} L_2 y(0).$$

Indeed, obviously $\|x(1)\| + \|y(1)\| = K$.

We emphasize that in this model we consider the "intrinsic" survival (described by the Leslie matrices) and the survival under selection independently. However, this model can be formally considered as a particular Leslie-type model that depends on the total density of the system, where each demographic parameter in the Leslie matrices L_1 and L_2 is multiplied by $\frac{K}{\|L_1 x(0)\| + \|L_2 y(0)\|}$.

Similarly, for all t= 1, 2, 3,… we get our kin demographic selection model for the two different phenotypes:

$$\begin{aligned} x(t+1) &= \frac{K}{\|L_1 x(t)\| + \|L_2 y(t)\|} L_1 x(t) \\ y(t+1) &= \frac{K}{\|L_1 x(t)\| + \|L_2 y(t)\|} L_2 y(t) \end{aligned}. \qquad (6)$$

Now, for the proportion of phenotype B we obtain:

$$\frac{\|y(t)\|}{\|x(t)\| + \|y(t)\|} = \frac{1}{\frac{\|x(t)\|}{\|y(t)\|} + 1}.$$

Here

1
$$\frac{\|x(t)\|}{\|y(t)\|} = \left(\frac{\lambda_1}{\lambda_2}\right)^t \frac{\frac{\|x(t)\|}{\lambda_1^t}}{\frac{\|y(t)\|}{\lambda_2^t}}.$$

2 Since we can suppose that in both phenotypes the last two fecundities are positive, the Perron-
3 Frobenius theorem (see e.g. Caswell 2001) implies that both $\frac{\|x(t)\|}{\lambda_1^t}$ and $\frac{\|y(t)\|}{\lambda_2^t}$ tend to finite
4 positive limits as $t \to \infty$. Therefore, $\lim_{t\to\infty} \frac{\|x(t)\|}{\|y(t)\|} = \infty$, implying

5
$$\lim_{t\to\infty} \frac{\|y(t)\|}{\|x(t)\| + \|y(t)\|} = 0.$$

6 Thus if $\lambda_1 > \lambda_2$, the relative frequency of phenotype B tends to zero as *t* tends to infinity.

7 **2.3. Kin demographic selection model: Life history strategy dependent Leslie model**
8 **combined with uniform density-dependent survival according to carrying capacity**

9 Under our Basic Assumptions (a)-(c), from the combination of the models of Sections 2.1 and
10 2.2 (equalities (1) and (6)) we get the kin demographic selection model:

11
$$\begin{aligned} x(t+1) &= \frac{K}{\|L(s_1)x(t)\| + \|L(s_2)y(t)\|} L(s_1)x(t) \\ y(t+1) &= \frac{K}{\|L(s_1)x(t)\| + \|L(s_2)y(t)\|} L(s_2)y(t) \end{aligned}. \qquad (7)$$

12 Observe that, $\frac{K}{\|L(s_1)x(t)\| + \|L(s_2)y(t)\|}$ describes the uniform density-dependent survival of
13 each individual under selection, and matrix $L(s_i)$ describes the trait-dependent (but density-

1 independent) demography of the phenotype. According to our Basic Assumption (c), the
2 product of these two factors describes the demography of the different phenotypes.

3 Using the reasoning of Section 2.2, if there is a unique global maximum $\lambda(s)$, at strategy s_1,
4 then this strategy will be fixed in the long run. If the maximum is attained at, say, two
5 strategies s_1 and s_2, such that $\lambda(s_1) = \lambda(s_2) > \lambda(s)$, $s_1 \neq s_2 \neq s$, then strategies s_1 and s_2 are
6 neutral with respect to each other, but outperform any other different strategies.

7 We note that the often used assumption that the basic phenotypic long-term growth rate
8 equals 1 (e.g. Stenseth 1985, where $\lambda = 1$ is assumed, to exclude infinite population growth in
9 the classical Leslie model), is not consistent with our model. On the one hand, our question is
10 which phenotype will win. If both phenotypes have a long-term growth rate equal to 1, neither
11 of the phenotypes would win. Nevertheless, in our model, when the selection has already
12 fixed one phenotype only, its growth rate stabilizes at 1. On the other hand, in the selection
13 model presented, the total population growth rate is 1 for each time step (since the total size of
14 the population of the different phenotypes is fixed at the carrying capacity *K*) but during the
15 selection process the evolutionarily most successful phenotype has a higher growth rate than
16 the growth rate of the other phenotypes.

17 Summing up Section 2, in spite of the fact that the life reproductive success of phenotype B is
18 greater than that of phenotype A, phenotype B will die out on the time scale of selection. The
19 lesson is that the average number of an individual's offspring is not necessarily a good
20 definition of fitness, when generations overlap and adults have more than one reproductive
21 period. We emphasize that not only phenotype B, but also all other phenotypes, will die out, if
22 different fitness concepts (e.g. reproductive value, Fisher 1930) determine different
23 phenotypes.

Consequently, based on Remark 2, the phenotypic long-term growth rate can be considered as the Darwinian fitness. Therefore, we compare below the phenotypic long-term growth rates of cannibal and non-cannibal phenotypes.

3. Kin demographic selection models for sib cannibalism

Sib cannibalism has two kinds of effect: an immediate one, during the juvenile stage, and a delayed one, during the adult stage. We will investigate these effects separately.

3.1. Mechanism-based model for the derivation of the Leslie matrix, when sib cannibalism can change only the juvenile demographic parameters

To get an insight into the advantage of sib cannibalism between closest relatives, in this subsection we will consider the maximal sib cannibalism intensity.

For simplicity we assume that there is the same prey density for each plant. Assume that each female uses a different plant for oviposition, so that we can assume that on each plant there is one cohort at a time, or in other words that each female's juveniles are of the same size at every time moment. Let us fix the time unit (which is based on temperature and real time: it is "degree day" for insects). To compare different phenotypes we have to use the same time unit. To develop from nymph to adult, a juvenile needs energy E, and only the energy collection determines the developmental time. Assume that, independently of the size of the juvenile, during unit time, each juvenile collects energy e from its prey and, for non-cannibal phenotypes, $(E-e_0)/e=10$ time units are needed to develop from egg to adult, where e_0 is the energy contained in a new-born nymph. All adults live only two time units.

Non-cannibal phenotype. For simple and shorter notation we assume that the survival rate of a nymph does not depend on its size, i.e. $\omega_i = \omega_1$, for $i=1,2,\ldots,10$, adult stages take two time

units, with survival rate ω_2, and only adults produce α eggs, then we have the following Leslie matrix for the non-cannibal phenotype:

$$\begin{pmatrix}
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha & \alpha \\
\omega_1 & 0 & & & & & & & & & & 0 \\
0 & \omega_1 & 0 & & & & & & & & & 0 \\
0 & 0 & \omega_1 & 0 & & & & & & & & 0 \\
0 & & 0 & \omega_1 & 0 & & & & & & & 0 \\
0 & & & 0 & \omega_1 & 0 & & & & & & 0 \\
0 & & & & 0 & \omega_1 & 0 & & & & & 0 \\
0 & & & & & 0 & \omega_1 & 0 & & & & 0 \\
0 & & & & & & 0 & \omega_1 & 0 & & & 0 \\
0 & & & & & & & 0 & \omega_1 & 0 & & 0 \\
0 & & & & & & & & 0 & \omega_1 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \omega_2 & 0
\end{pmatrix}.$$

Observe that the survival rate of a non-cannibal nymph from egg to adult age is ω_1^{10}.

Sib cannibal phenotype. For simplicity, we suppose that non-conspecific predators totally consume what they kill, and that if a sibling dies for some other reason (such as an infection) it is not consumed by its cannibal sibling (e.g. it falls off the plant). During unit time, a surviving cannibal juvenile can also collect energy e from its prey, and we assume that the time unit is small enough such that a cannibal individual can eat only one of its sibs. Observe that now s=1 means that, in each time unit, each surviving sib cannibalizes one of its siblings. We assume that, on average, a killed sibling can only collect half the energy before it is killed by its cannibal sibling. Under these assumptions, sib cannibalism has two effects:

Accelerating effect. At the end of the first time unit, the collected energy is e_0+e+(e_0+0.5e), where (e_0+0.5e) is the energy content of the cannibalized sibling at the first time unit. Thus in the t-th time unit, the total collected energy is:

$$E(t+1) = 2E(t) + 1.5e,$$

and the series of values of collected energy is: $E(0) = e_0$, $E(1) = 2e_0 + 1.5e$, $E(2) = 4e_0 + 4.5e$, $E(3) = 8e_0 + 10.5e$. Under our assumption that collected energy determines developmental time, a sib cannibal juvenile can mature in only three time units, while a non-cannibal needs 10.

Juvenile's survival effect. We assume that dying without a cannibal attack and dying in a cannibal attack are independent events in the sense of probability theory, and thus that the survival rate is $\omega_1/2$ in each time unit. We should emphasize that we are not assuming that sib cannibalism can increase the survival rate of successful juveniles, since ω_1 does not increase with time.

Based on the above two effects, we get the following Leslie model for the sib cannibal phenotype:

$$\begin{pmatrix} 0 & 0 & 0 & \alpha & \alpha \\ \frac{\omega_1}{2} & 0 & 0 & 0 & 0 \\ 0 & \frac{\omega_1}{2} & 0 & 0 & 0 \\ 0 & 0 & \frac{\omega_1}{2} & 0 & 0 \\ 0 & 0 & 0 & \omega_2 & 0 \end{pmatrix},$$

Observe that the survival rate of a sib cannibal juvenile from egg to adult age is $\left(\frac{\omega_1}{2}\right)^3$.

There are two possible advantages of sib cannibalism:

Juvenile's survival advantage. In spite of the fact that during each time unit the survival rate strictly decreases, the survival rate from juvenile to adult can increase, since the development time radically decreases. In the present case, the survival rate of a non-cannibal juvenile ω_1^{10} is smaller than that of a sib cannibal $\left(\frac{\omega_1}{2}\right)^3$ if and only if $\omega_1 < 0.743$. This is illustrated by the following numerical example.

Example 1. If $\alpha = 10000$, $\omega_2 = 1$, $\omega_1 = 0.6$, then the phenotypic long-term growth rate of the non-cannibal phenotype (1.78) is less than that of the cannibal one (4.27).

Now the question arises of whether it is true that, whenever the expected number of offspring of a new-born non-cannibal juvenile surviving to adult stage is higher than that of a cannibal one, the phenotypic long-term growth rate of the non-cannibal phenotype is also higher. To answer this question, consider the following example:

Example 2. If $\alpha = 10000$, $\omega_2 = 1$, $\omega_1 = 0.9$, then the long-term growth rate of the non-cannibal phenotype (2.17) is less than that of the cannibal one (5.72).

In Example 2, although sib cannibalism radically decreases the number of surviving juveniles, cannibalism ensures a higher long-term growth rate. Why? The answer is explained below.

Accelerating advantage. Assume that the non-cannibal phenotype has 8 generations, while the cannibal phenotype has 20 generations, during one seasonal cycle. Clearly, in spite of the fact that the non-cannibal phenotype has more surviving offspring than the cannibal one after one generation, the cannibal phenotype can produce more offspring by the end of the reproductive period than the non-cannibal one. Indeed, let *X* and *Y* be the numbers of non-

cannibal and cannibal phenotype offspring in one generation, respectively, then at the end of the seasonal cycle $X^8 < Y^{20}$ for the total numbers of descendants could easily hold despite the inequality $X > Y$. More generally, $X^k < Y^l$ is equivalent to $l > k\frac{\ln X}{\ln Y}$, which is the condition for the accelerating advantage of cannibalism in this case.

Example 3. Let $\alpha = 100$, $\omega_2 = 1$, $\omega_1 = 1$. Then the phenotypic long-term growth rate of the non-cannibal phenotype (1.58) is less than that of the sib cannibal one (2.07). We note that in this example the life reproductive success of the non-cannibal phenotype is 200, while that of the sib cannibal one is 25, since all non-cannibal juveniles mature, while only one-eighth of the cannibal juveniles survive (7/8 of the siblings are cannibalized by their surviving siblings). So, life reproductive success cannot be considered as a definition of fitness.

The "paradoxical" lesson is this: when the developmental time radically decreases because of sib cannibalism, the phenotypic long-term growth rate can increase, even though the number of surviving juveniles decreases because of sib cannibalism.

3.2. Sib cannibalism can also change the juvenile and adult demographic parameters

As we have already mentioned, sib cannibalism between juveniles can increase longevity in the adult stage. To demonstrate the consequence of this delayed phenomenon, we have to ignore all the effects of Section 3.1 that can mask the delayed effect of sib cannibalism on longevity. Assume that sib cannibalism decreases the survival rate in juveniles, does not decrease the developmental time and can increase the survival rate of adults. Consider a theoretical example when both the juvenile and the adult stages take two time units. Under these assumptions we consider the following Leslie matrix for the cannibal phenotype:

$$\begin{pmatrix} 0 & 0 & \alpha & \alpha \\ \omega_1 - cs & 0 & 0 & 0 \\ 0 & \omega_1 - cs & 0 & 0 \\ 0 & 0 & \omega_3 + bs & 0 \end{pmatrix},$$

where the "cost" of sib cannibalism is $0 < c < \omega_1$ and the "benefit" of cannibalism is $0 < b < 1 - \omega_3$ (where the latter inequality guarantees the consistency of the present model). Here s denotes the intensity of sib cannibalism: $0 < s \le 1$ corresponds to the sib cannibal phenotype and s=0 means no sib cannibalism.

In the Appendix we show that sib cannibalism can be adaptive if the following sufficient condition holds:

$$2\frac{\lambda_0 + \omega_3}{\omega_1} < \frac{b}{c}. \qquad \text{(A.1)}$$

Therefore, for any set of parameters $\alpha, \omega_1, \omega_3$, condition (A.1) holds whenever the benefit/cost proportion is large enough, so a high benefit/cost proportion implies that sib cannibalism is adaptive. In the following numerical examples condition (A.1) will be satisfied.

Example 4. Let us consider a case when sib cannibalism radically increases the longevity in the adult stage:

$$\begin{pmatrix} 0 & 0 & 100 & 100 \\ 0.5 - 0.01s & 0 & 0 & 0 \\ 0 & 0.5 - 0.01s & 0 & 0 \\ 0 & 0 & 0.01 + 0.86s & 0 \end{pmatrix},$$

The parameters $\alpha = 100$, ω_1=0.5, ω_3=0.1, c=0.01, b=0.86 satisfy condition (A.1), implying that the sib cannibalism is adaptive. Furthermore, in this case, both the phenotypic long-term growth rate and the life reproductive success take their maxima at s=1 (see Figure 3).

Figure 3.

Example 5. Now consider different parameters, namely:

$$\begin{pmatrix} 0 & 0 & 100 & 100 \\ 0.5-0.06s & 0 & 0 & 0 \\ 0 & 0.5-0.06s & 0 & 0 \\ 0 & 0 & 0.1+0.8s & 0 \end{pmatrix}.$$

In this case the phenotypic long-term growth rate takes its maximum at s= 0.23, while the life reproductive success is maximal at s=1 (see Figure 4).

Figure 4

4. Conclusions

Darwinian selection of fitness notions

Using the kin demographic selection model introduced here, we have shown that the phenotype maximizing the phenotypic long-term growth rate will select out other different phenotypes (*cf*. Garay & Varga 2005). For instance, the phenotype maximizing its life reproductive success and the phenotype maximizing its reproductive value at every age (Fisher 1930; Schaffer 1974) are outperformed by the phenotype maximizing its phenotypic long-term growth rate, when these phenotypes are different.

Results assuming the Collector Hypothesis

The theoretical framework presented is strictly based on classical Darwinism for the following selection situation: there are neither density- nor frequency-dependent inter-phenotype interactions; cannibalism happens between the closest relatives; the generations overlap; and all other possible selection forces have the same effect on all individuals. We gave some theoretical examples of when sib cannibalism between the closest relatives can increase the phenotypic long-term growth rate. The basic biological assumption of our Collector Hypothesis is that it is mainly the food (nutrient) intake that determines the developmental time, the survival rate of pre-fertile juveniles, and the fecundity of adults. We found that if the developmental time is a strictly decreasing function of food consumed, then a sib cannibal can decrease its developmental time. Shorter developmental time can increase the survival rate from juvenile to adult. Consequently, the sib cannibalism is adaptive. Sib cannibalism can also be adaptive if it increases survival in the adult stage.

Essentially, the Collector Hypothesis claims that cannibalism could be an effective feeding strategy for the phenotype. For instance, Arctic char are often the only fish found in northern lakes, and small and cannibalistic giant chars form a "food web": the small char feed on zooplankton, while a cannibalistic giant char eats smaller char that are up to a third of its size (Byström 2006). In this case, cannibalism can be considered as a feeding strategy.

We hope that our prediction could be tested by experiments in species for which food can either shorten the development time or increase the survival in the adult stage, in each case radically enough to overcompensate for the negative effect of cannibalism.

Connection with kin selection theory

From the point of view of individuals, cannibalism is an antagonist behaviour; from the point of view of phenotypes, in a well-mixed population, cannibalism is a competitive behaviour

that can increase the relative advantage of the cannibal phenotype over the non-cannibal one. However, as we have shown, sib cannibalism between the closest relatives can be adaptive for kin. There is a harmony between our models and Hamilton's kin selection theory, since in our case interactions also happen only within kin. Sib cannibalism can be considered as an extreme version of mutualism, when some juvenile (collecting nutrient in its body for its sib) pays the extreme cost and the benefit is a higher long-term growth rate for its kin. So, if sib cannibalism is adaptive among the closest relatives, then cannibalism is even more adaptive among non-relatives. Thus, we think that the evolutionary advantage of cannibalism can occur whatever the relatedness. Nevertheless, there will surely be some effect of relatedness on the likelihood of it occurring. In the asexual case (when the genetic relatedness of the recipient to the actor is one), the actual version of the well-known Hamilton rule strictly depends on the details of the life history (see inequality (A.1)), since the generations overlap. We conjecture that in a sexual population sib cannibalism may also be adaptive, but this is the subject of a further paper.

Our model is strictly based on the fundamental idea of kin selection theory (favouring the reproductive success of a sib, even at a cost to the other sibs), but it is not the standard version of the kin selection model (since in our case the fitness is implicitly given). Moreover, the results for our model give some insight into the problem of competition between sibs (*cf.* Gardner & West 2004; West *et al.* 2002). In spite of the fact that sib cannibalism is a negative interaction between sibs, if it can decrease the development time, and consequently can increase the total survival rate of juvenile sibs, and furthermore can increase longevity in the adult stage, in the end it can increase the long-term growth rate for sib cannibalistic kin. As far as we know our kin demographic model is the first to have pointed out that a negative interaction between kin can be an evolutionarily successful strategy for the kin (*cf.* Frank 1998; Queller 1994).

Relationship to biological pest control

In biological pest control, a natural enemy of the pest (insect or mite) can be applied in two ways. If the biocontrol starts when there is a high pest density, an inundative release is applied in which a large number of adult agents are released for an immediate reduction of the pest. At a low initial pest density, by contrast, an inoculative agent release may be more cost efficient; the agents reproduce themselves through several generations to provide long-term control (see e.g. Hajek 2004). The long-term growth rate studied in the present paper then has obvious importance. Furthermore, in the case of an inoculative release every single female may find a different plant for oviposition. Since certain agents also display sib cannibalistic behaviour (see e.g. Michaud 2005), the kin demographic model and the maximization of the phenotypic long-term growth rate may have importance for biological control, as well.

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18

Appendix

Consider the following Leslie matrix:

$$\begin{pmatrix} 0 & 0 & \alpha & \alpha \\ \omega_1 - cs & 0 & 0 & 0 \\ 0 & \omega_1 - cs & 0 & 0 \\ 0 & 0 & \omega_3 + bs & 0 \end{pmatrix}.$$

The phenotypic long-term growth rate is the unique positive solution $\lambda(s)$ of the following characteristic equation for all fixed s:

$$\lambda^4 - \alpha(\omega_1 - cs)^2\lambda - \alpha(\omega_1 - cs)^2(\omega_3 + bs) = 0.$$

Now the question arises of when sib cannibalism can be adaptive. Formally, we obtain below a sufficient (but not necessary) condition, implying that $\lambda(s)$ is an increasing function at $s = 0$. In the latter case, changing the intensity of the cannibalism from zero to a small positive value increases the phenotypic long-term growth rate. Using the implicit function theorem we get

$$\begin{aligned} \lambda'(s) &= -\frac{\frac{\partial}{\partial s}\left[\lambda^4 - \alpha(\omega_1 - cs)^2\lambda - \alpha(\omega_1 - cs)^2(\omega_3 + bs)\right]}{\frac{\partial}{\partial \lambda}\left[\lambda^4 - \alpha(\omega_1 - cs)^2\lambda - \alpha(\omega_1 - cs)^2(\omega_3 + bs)\right]} \\ &= -\frac{2\alpha c(\omega_1 - cs)\lambda - \alpha(\omega_1 - cs)\left[-2c(\omega_3 + bs) + (\omega_1 - cs)b\right]}{4\lambda^3 - \alpha(\omega_1 - cs)^2}. \end{aligned}$$

Hence, at $s = 0$, with $\lambda_0 = \lambda(0)$, we get

$$\lambda'(0) = \frac{-2\alpha c\omega_1\lambda_0 + \alpha\omega_1\left[-2c\omega_3 + \omega_1 b\right]}{4\lambda_0^3 - \alpha{\omega_1}^2}.$$

Since from the characteristic equation it is easy to obtain $4\lambda_0^3 - \alpha\omega_1^2 > 0$, for $\lambda'(0) > 0$, a necessary and sufficient condition is

$$-2\alpha c\omega_1\lambda_0 + \alpha\omega_1\left[-2c\omega_3 + \omega_1 b\right] > 0,$$

which can be guaranteed by

$$2\frac{\lambda_0 + \omega_3}{\omega_1} < \frac{b}{c}. \tag{A.1}$$

Summing up, inequality (A.1) is a sufficient condition for the adaptivity of cannibalism in the model in Section 3.2.

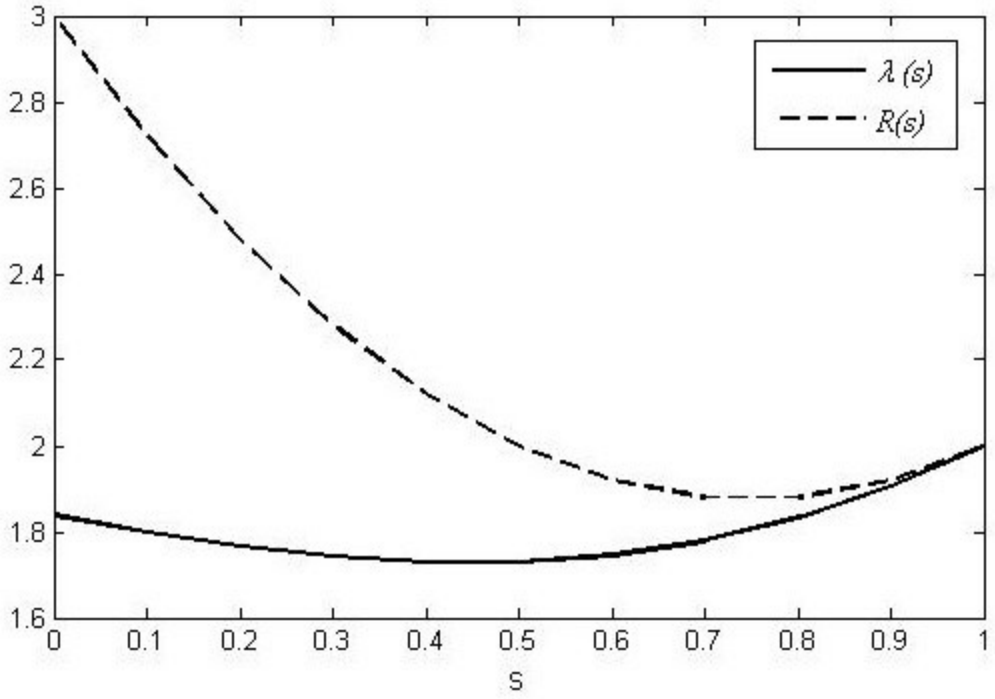


Figure 1. The phenotypic long-term growth rate $\lambda(s)$ is the positive root of the characteristic polynomial $P(\lambda) = \lambda^3 - (1+s)\lambda^2 - (1-s)^2\lambda - (1-s)^2$. The life reproductive success $R(s)$ is always greater than the phenotypic long-term growth rate $\lambda(s)$, except when $s = 1$, when they are equal. Observe that life reproductive success and the phenotypic long-term growth rate take their maxima at different extreme values of the intensity of cannibalism.

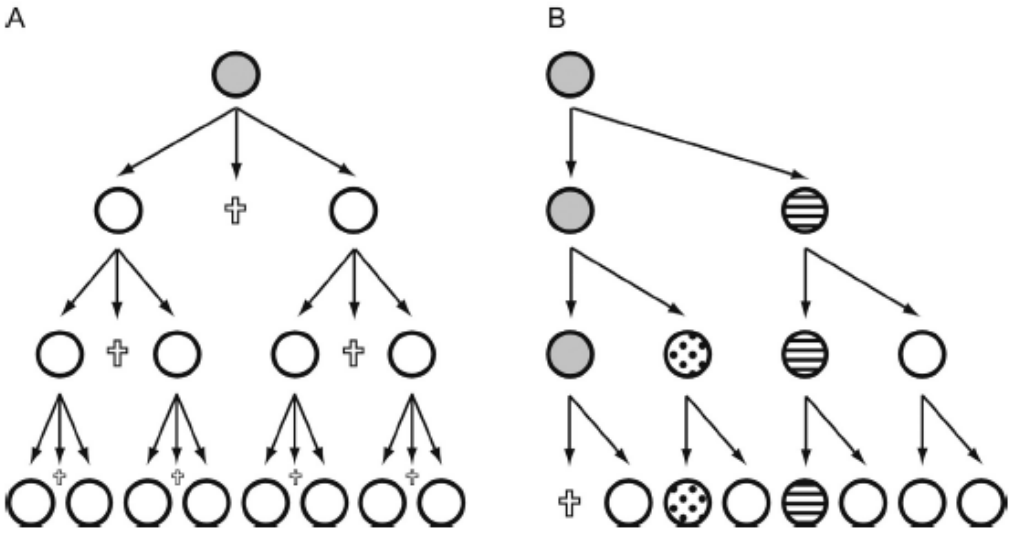


Figure 2. Tree A corresponds to phenotype A ($s^* = 1$) living one time unit and having two offspring. Tree B corresponds to phenotype B ($\hat{s} = 0$) living three time units and having one offspring in each time unit. Observe that at the third time unit phenotype A has 8, and phenotype B has only 7 individuals.

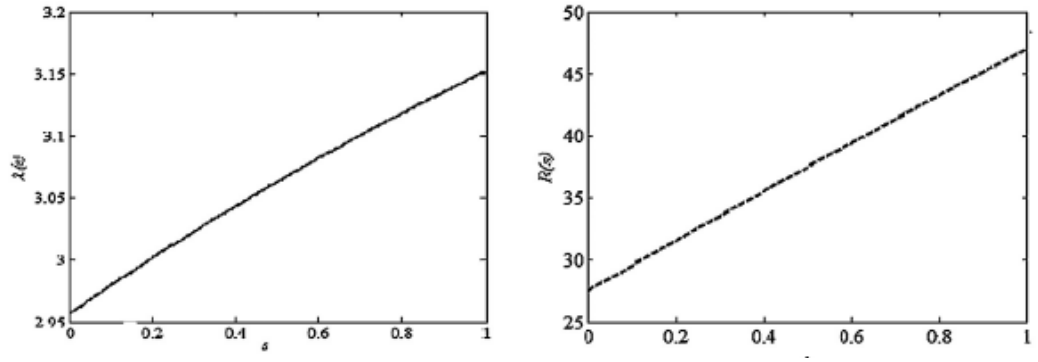
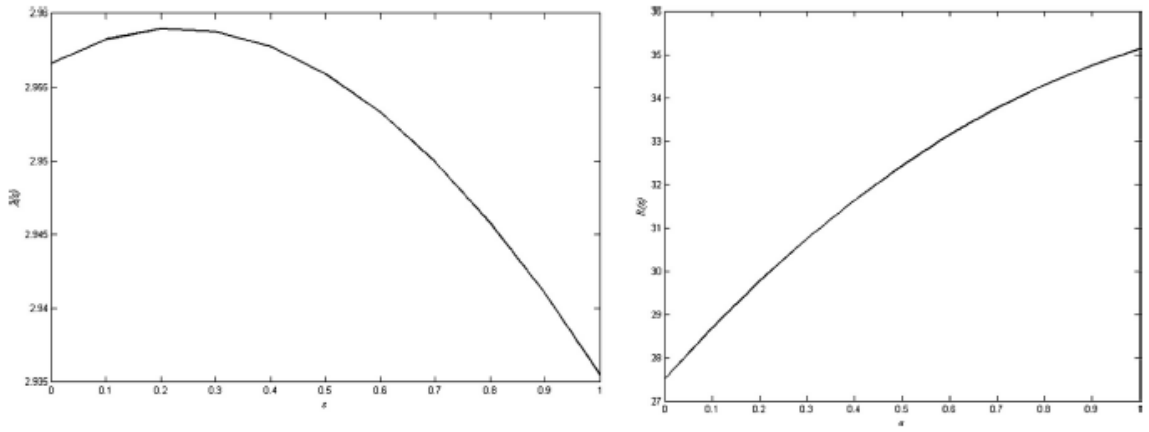


Figure 3. In Example 4, phenotypic long-term growth rate and life reproductive success take their maxima at the maximal sib cannibalism intensity.

1



2

3 Figure 4. In Example 5, phenotypic long-term growth rate and life reproductive success take

4 their maxima at different sib cannibal intensities.