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Optimal energy allocation to growth, reproduction, and production of defensive substances in plants: a model

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ABSTRACT

Question: Does allocation to the production of defensive substances in plants affect such characteristics as growth rate, time of maturation, size at maturity, current and lifetime reproductive success?

Numerical method: Dynamic programming algorithm to analyse the optimal energy allocation to growth, reproduction, and production of defensive substances.

Key assumptions: The production of defensive substances requires energy that should be diverted from other processes. The plants live in a non-seasonal environment. Defensive substances are non-degradable substances (e.g. tannins).

Predictions: Optimal energy allocation responds to level of herbivory, intrinsic properties of a plant to produce defensive substances (α), and external mortality. High levels of herbivory and low external mortality promote the production of defensive substances. Low herbivory pressure, as well as high external mortality, make the production of defensive substances non-optimal and favour the simultaneous allocation of energy to growth and reproduction. Plants with this strategy lose their vegetative mass after maturation. A negative exponential decrease in herbivory with an increase in the concentration of defensive substances promotes simultaneous allocation of energy to growth and reproduction after maturation for some values of α and some efficiencies of defensive substances.

Keywords: dynamic state variable model, herbivory, life history, resistance, resource allocation, tolerance.

INTRODUCTION

In this study, I approach the production of defensive substances from the life-history and optimal energy allocation points of view. I assume that defence against herbivores requires energy that, once invested, could not be reallocated to other processes such as growth or

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reproduction. Therefore, higher concentrations of defensive substances should bring about costs (e.g. lower fitness). Whereas some studies have either confirmed or suggested the existence of costs (Coley *et al.*, 1985; van Dam *et al.*, 1996; Lehtilä and Strauss, 1999; Cornelissen and Fernandes, 2001; Coviella *et al.*, 2002; Ruiz *et al.*, 2002, Hoballah *et al.*, 2004; Rudgers *et al.*, 2004; Joshi and Vrieling, 2005; Pratt *et al.*, 2005; Lewis *et al.*, 2006; Osier and Lindroth, 2006), others have not provided such evidence (Ågren and Schemske, 1992; Vrieling and van Wijk, 1994). Bergelson and Purrington (1996) reviewed the results of 58 studies of plant resistance to herbivores, pathogens, and herbicides and found costs in 33% of them. Later, Strauss *et al.* (2002) updated and extended this analysis with the results from 33 studies on resistance to herbivores only, and detected costs in 82% and 76% of studies with and without a controlled genetic background, respectively.

The existence of trade-offs between defence and growth (Osier and Lindroth, 2006), seed mass and leaf removal (Spotswood *et al.*, 2002), level of damage and fruit number (Pratt *et al.*, 2005), growth rate and salicortin (Orians *et al.*, 2003) or sinigrin concentration (Lewis *et al.*, 2006), percentage of leaves attacked, shoot length, growth rate, and tannin concentration (Cornelissen and Fernandes, 2001), as well as between different direct resistance traits (Rudgers *et al.*, 2004), also suggests a conflict between allocation to defence and to other compartments. Obviously, the production of defensive substances or other modes of defence – as with any other physiological process – should involve an investment of energy, but such expenditure is difficult to measure in both laboratory and field studies. Strauss *et al.* (2002) detected high costs of resistance ranging from 6% to 45% and from 8.7% to 73% in controlled background and natural population studies, respectively.

Defensive substances have evolved in many plants and apparently they are neither waste products nor do they serve any other known function of the plant (see Coley *et al.*, 1985). This fact, together with the evidence of costs and trade-offs, obviates the application of an allocation model that assumes the existence of different energy sinks, one of them being the production of defensive substances.

Some optimal defence models preclude the co-existence of two kinds of herbivory arrestment, i.e. resistance and tolerance. The former implies the production of either defensive substances or structures and the latter the arrestment of damage due to tissue re-growth. Tolerant plants do not allocate resources to defensive substances but re-grow rapidly after tissue damage following disturbance. Some models explicitly assume the existence of a conflict between the allocation of energy to different sinks (de Jong, 1995; Iwasa and Kubo, 1997; de Jong and van der Meijden, 2000), and others the existence of costs in terms of resources without explicitly taking into account the allocation of energy (e.g. van der Meijden, 1996; Fornoni *et al.*, 2004). Other models look at the distribution of defensive substances among leaf age classes by applying a demographic approach: they treat leaves of a single plant as a population and apply a modification of the Euler-Lotka equation. Then, an analogue of the Malthusian parameter that expresses either the growth rate of the plant (van Dam *et al.*, 1996) or the assimilative value of the leaf (Lambdon and Hassall, 2005) is maximized.

I assume here that the production of defensive substances requires energy, which is diverted from other processes such as growth and reproduction. If this is the case, a strong trade-off should exist between energy allocation to defence and allocation to other processes. I study the outcomes of some trade-offs, including between growth rate and time of maturation. Allocation to the production of defensive substances is analogous to the investment in resistance. I do not focus here on the problem of tolerance, although some results may be referred to as 'tolerance'.

Some optimal energy allocation models have already approached the problem of

herbivory. Klinkhamer *et al.* (1997) studied the effect of herbivory on reproductive success, survival to the following year, and efficiency of recovery from the storage compartment. They focused on the effect of the plant's tissue loss on the evolution of the reproductive schedule (semelparity vs. iteroparity). Iwasa and Kubo (1997) and de Jong and van der Meijden (2000) modelled the effect of unpredictable disturbance of generalist herbivores on re-growth dynamics after disturbance (tolerance) rather than the optimal concentration of defensive substances in the plant's tissues. They assumed that re-growth occurs due to the relocation from the storage sink and analysed the optimal growth curves. Yamauchi and Yamamura (2004) modelled optimal energy allocation to growth, reproduction, and nutrient acquisition by plants. They included herbivory in their model, although they focused on the problem of 'grazing optimization' and nutrient recycling due to the action of herbivores rather than optimal defence. All of the above allocation models examined the effect of herbivory and/or disturbance on optimal energy allocation without taking into account either the concentration of defensive substances in the plant's body or the optimal allocation of energy necessary for constant tissue replacement after loss during life (no relocation from storage to growth).

Another important topic that has received much attention is inducible versus constitutive resistance in plants (van Dam *et al.*, 1993, 2000; van Dam and Vrieling, 1994; Strauss *et al.*, 2002). In the model I propose here, the extent to which a plant responds to herbivore pressure is set by its intrinsic properties to produce defensive substances, the intensity of herbivory, and external mortality. The existence of an intrinsic genetic component and/or genetic variation regarding resistance or a genotype–environment interaction is evidenced or suggested by several studies (Vrieling *et al.*, 1993; van Dam and Vrieling, 1994; Orians *et al.*, 1996, 2003; Tiffin and Rausher, 1999; Fornoni *et al.*, 2003; Meaux and Mitchell-Olds, 2003; O'Reilly-Wapstra *et al.*, 2004; Lewis *et al.*, 2006; Osier and Lindroth, 2006). I evoke here the existence of two possible modes of genetic determination of the physiological defensive equipment: (i) the determination of an intrinsic physiological mechanism that modifies the proportions of energy invested in defensive substances, or (ii) the determination of the proportion of energy devoted to the production of defensive substances. In the former case, we expect the plastic response of a plant to different levels of herbivory pressure. In the latter case, a plant will respond in the same way independently of the level of herbivory. I also suggest that plants exhibiting an inducible response to the presence of herbivores have a physiological component of such a plastic response. Analogously, plants that do not induce the production of defensive substances in the presence of herbivores are unable to change the proportion of energy allocated to this activity. Indeed, the results of van Dam *et al.* (1993) suggest the existence of different response mechanisms in different plant species.

In the model, I also address the question of the tolerance to herbivory even when it is not exactly compatible with its 'classic' definition, which defines this trait as the capacity to repair tissues after damage. I do not include storage in the model, although I show that when defence is not optimal, and the plant loses tissues, it is optimal – under some conditions – to allocate energy to growth after maturation. Even when it is not a 'true' re-growth from storage, it can be considered tolerance in the sense that it is 'non-resistance'. Several studies have previously proposed that both tolerance and resistance should be considered 'operational' definitions (for a review, see Mauricio, 2000). It is generally believed that tolerance minimizes the effect of herbivory on plant fitness (see Tiffin and Rausher, 1999; Tiffin and Inouye, 2000) because energy is not diverted to the production of defensive substances. This is associated with the optimality of resistance (production of defensive substances),

i.e. a defensive mode that diverts energy from other processes and thus negatively affects fitness. This is not an accurate definition, since the re-growth also diverts energy from other processes and thus affects fitness negatively.

The model ascertains when either resistance or non-resistance (tolerance?) is an optimal solution. The optimal concentration of defensive substances in the body is an outcome of optimal energy allocation.

Theoretical as well as laboratory work often neglects the effect of external (environmental) mortality (e.g. predators, diseases, parasites). However, several theoretical studies have demonstrated that this factor determines the allocation pattern (Cichoń, 1997; Cichoń and Kozłowski, 2000; Janczur *et al.*, 2000). I show here that plants that evolve under high external mortality have different patterns of defence than those that evolve in low-mortality environments.

THE MODEL

For simplicity, I consider an asexual plant. The basic assumption is that defence against herbivores is an energy-demanding process. It implies not only the production of toxic defensive substances but also the production of such structural defence as spines and pubescence. Another important point is that active defence diverts energy from other processes. The loss of vegetative tissues reduces reproductive output, since production is a function of plant size. Again, for simplicity, I assume that only one kind of generalist herbivore is grazing the plant, or that all herbivores have the same grazing rate. I apply the dynamic programming algorithm to examine optimal energy allocation to growth, reproduction, and defence against herbivores of an organism living in a constant environment (no seasons, constant population size). This algorithm involves the division of the entire lifespan into short intervals (2% of the lifespan in the model) and the optimization of energy allocation in each interval. I also study the consequences of optimal energy allocation for some life-history traits, such as plant tissue mass dynamics, adult size and age at maturity, as well as the concentration of defensive substances in the plant's tissues.

In an evolutionary context, an optimal decision involves the maximization of lifetime (present and future) reproductive success (fitness), since the strategy that produces greater offspring survival will transmit more genes to the next generations (Taylor *et al.*, 1974). In this paper, the word 'strategy' denotes a schedule of energy allocation to different processes changing dynamically throughout a plant's life.

I express the lifetime allocation to offspring (seed) production using the reproductive value. Assuming constant seed size ($w_0 = 1$), this is a measure of fitness for populations at equilibrium regulated by density dependence early in life (Mylius and Diekmann 1995), which is almost intrinsic for this class of problems (e.g. Kozłowski and Uchmański, 1987; Kozłowski and Wiegert, 1987; Heino and Kaitala, 1996; Cichoń, 1997; Teriokhin, 1998; Kozłowski and Teriokhin, 1999).

I assume that the maintenance costs have priority, and that only surplus energy is allocated to the above processes. The production is the difference between assimilation and metabolic rate, both of which are size-dependent:

$$P(w) = A - R = w^{0.6} - 0.1w^{0.7} \tag{1}$$

where w is size at age t measured in energy units. If respiration exceeds assimilation, no surplus energy is available and all the energy is used for maintenance alone. The values of the parameters are not derived from any real biological situation, but the results are

qualitatively similar as long as the function $P(w)$ remains concave. I describe the fractions of energy assigned to each physiological process using the control vector $\boldsymbol{U}(t) = (u_1(t) \ldots u_n(t))$ with two constraints:

$$0 \le u_i(t) \le 1 \qquad \text{and} \qquad u_i(t) + \ldots + u_n(t) = 1 \tag{2}$$

where i is an arbitrary number assigned to each activity and n is the number of energy sinks in the model. Hereafter, for the sake of easier notation, I will use u_i instead of $u_i(t)$, even though the control vector is time-dependent.

The plant in the model allocates the amounts $u_1P(w(t))$ and $u_2P(w(t))$ of energy to growth and to reproduction respectively (Fig. 1), both measured in energy units, even though the latter can be easily converted to seed number of a constant size. The herbivore harvests fraction h of the plant's mass, i.e. the plant loses an amount hw of the vegetative tissues in a time unit. This brings about a decrease in reproductive output, since the production rate is size-dependent. To arrest the negative effect of herbivory on fitness, the plant

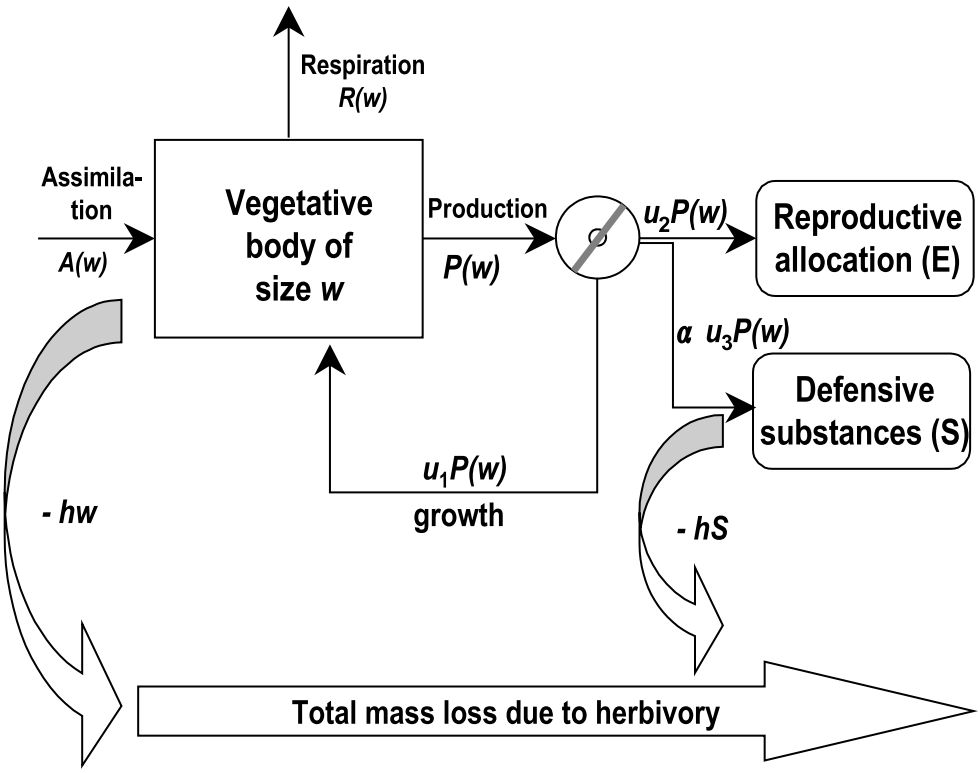

Fig. 1. Flow chart of energy allocation. A plant of vegetative size w assimilates energy at rate $A(w)$. After satisfying all living expenses, fractions u_1, u_2, and u_3 (where $u_3 = 1 - u_1 - u_2$) of surplus energy $P(w)$ can be allocated to growth, reproduction, and the production of defensive substances respectively. The rate of production of defensive substance depends also on the intrinsic property of a plant (α). Note that lower α implies a higher intrinsic production cost of defensive substances. At each moment of life, fraction h of a vegetative body is removed by a generalist herbivore. Production ($P(w)$) is the difference between assimilation ($A(w)$) and respiration ($R(w)$).

invests the fraction $u_3 = 1 - (u_1 + u_2)$ of the surplus production P to the production of defensive substances (or non-productive defensive structures). This investment decreases h, the concentration of defensive substances in the body.

I define the system by the control vector regarding the plant's allocation decision as well as by the state vector that describes its response to such a decision. The elements of the state vector are plant size, the amount of defensive substances in the plant, and the amount of resources devoted to reproduction. I describe the dynamics of the state vector by a system of differential equations. For clarity of presentation, I use different letters for each element of the state vector here:

$$\begin{aligned} \frac{dw}{dt} &= u_1 P(w) - hw \\ \frac{dE}{dt} &= u_2 P(w) \\ \frac{dS}{dt} &= \alpha u_3 P(w) - hS \end{aligned} \tag{3}$$

where w represents vegetative mass in energy units minus energy contents of defensive substances, E is energy for reproduction, S is the pool (not concentration) of defensive substances, hw is the loss of plant mass due to herbivory, hS is the corresponding loss of defensive substances, and the coefficient α is the internal, physiological property (e.g. genetically fixed) of the plant, measuring the efficiency of production of defensive substances (the amount of defensive substances produced from a unit of energy, e.g. in $\mu g \cdot J^{-1}$; note that the defensive substances production cost is the inverse of α). In different numerical examples, α varies from 5 to 15. Several empirical studies have confirmed the existence of differences in the efficiency of production of defensive substances (α in the model) in either closely related species or different populations of the same species (e.g. Agrawal *et al.*, 1999; Rousi *et al.*, 1996; Vrieling *et al.*, 1993; van Dam and Vrieling, 1994).

I assume here that the proportion h of the vegetative tissues removed by herbivores decreases either linearly or exponentially with the amount of defensive substances. In both cases, h decreases after the concentration $C = S/w$ of defensive substances in the vegetative body reaches the threshold concentration $C_N = 0.1$. The herbivory rate is maintained at the minimum level $h_{\min} = 0.01$ when the concentration of defensive substances is greater than $C_K = 10$ in the linear model. Thus for the linear model:

$$h = \begin{cases} h_{\max} & for \quad C \le C_N \\ \dfrac{h_{\max}(C_K - C)}{C_K - C_N} & for \quad C_N < C < C_K, \\ h_{\min} & for \quad C \ge C_K \end{cases} \tag{4}$$

and for the negative exponential model:

$$h = \begin{cases} h_{\max} & for \quad C \le C_N \\ h_{\max} e^{-g(C - C_N)} & for \quad C > C_N \end{cases},$$

where the parameter g changes in the model from 0.1 to 0.6 (Fig. 2a and b for linear and negative exponential model respectively). In different numerical examples, I make the

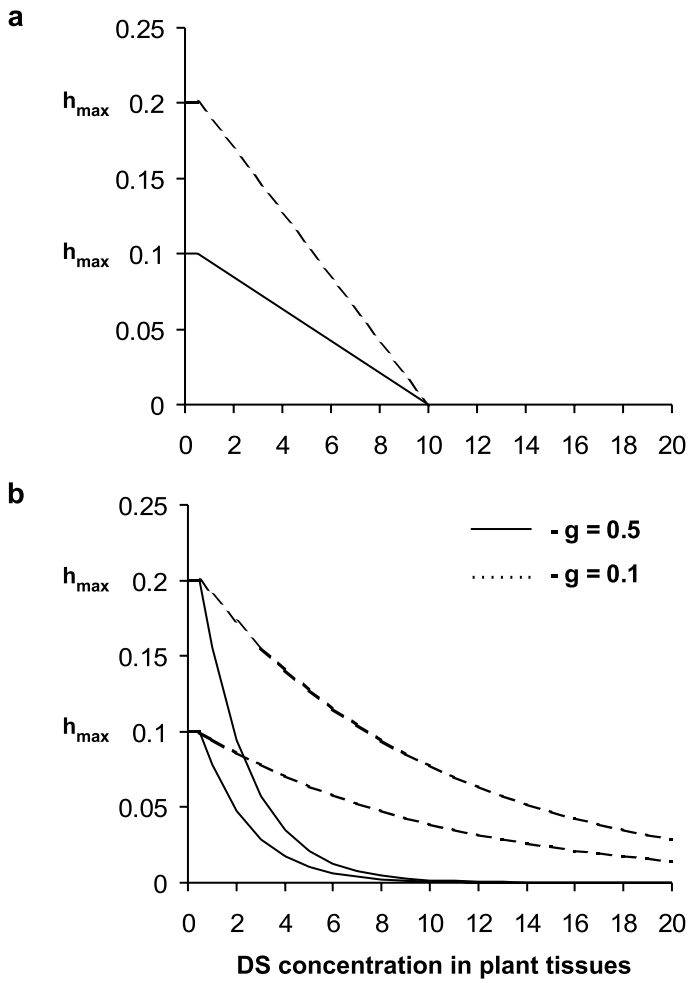

Fig. 2. Linear (a) and negative exponential (b) effect of the concentration of defensive substances (DS) in the plant tissues on herbivory rate (h). h_{max} is the herbivory rate when defensive substances are absent and is called 'herbivory pressure'. In both models, the threshold concentration $C_N = 0.1$. g is the effectiveness of defensive substances in repelling herbivores.

maximum herbivory rate (h_{max}) constant during an organism's life. Hereafter, I will call h_{max} 'herbivory pressure' and the value h in equation (3) the 'herbivory rate'. Also, I will call the negative exponential model the 'exponential model'. I assume that herbivory changes the assimilation rate (first term in equation 1) only through its effect on vegetative size. Since the assimilation rate is a function of the leaf area index (LAI) (Gholz, 1982; Primack, 1979; Webb *et al.*, 1983), this assumption is reasonable. A lower assimilation rate due to higher cumulative body loss is already included in the first term of equation (1). I maintain constant threshold concentrations in the linear model.

I assume that defensive substances are non-degradable compounds like tannins, i.e. they are lost only through harvesting, not through chemical decomposition.

In an ecological context, mortality should always be considered, since only an organism that survives until maturation will reproduce. I consider here two mortality components: external mortality q_{ex} and age-dependent mortality $m(t)$. The corresponding part of the survivorship function defined by external mortality is:

$$L_1 = e^{-tq_{ex}} \tag{5}$$

where q_{ex} is the external (age-independent) mortality rate. External mortality is constant during the plant's life, and varies from $q_{ex} = 0.0001$ to $q_{ex} = 0.05$ between different numerical examples. To describe the age-dependent mortality rate, I use the function given by Kozłowski and Teriokhin (1999):

$$m = q_0 \frac{T}{T - t}$$

with the corresponding survival function (6)

$$L_2 = e^{-\int_0^t m(x)dx} = \left(\frac{T - t}{T} \right)^{Tq_0}$$

where m is the age-dependent component of mortality rate, and T is maximum longevity or intrinsic lifespan (e.g. physiologically determined). Mortality (m) defined this way tends to infinity when t approaches T. For values of q_0 close to zero, m is very low for almost the entire lifespan and goes quickly to infinity at the end of life. When q_0 increases, m increases earlier in life. Throughout this paper, I assume that $q_0 = 0.022$. The latter mortality term is rather obvious, since it is easy to interpret it as a determinant of the maximum physiological lifespan: when m grows exponentially to infinity, the plant dies (see also Kozłowski and Teriokhin, 1999; Janczur *et al.*, 2000). The former term (q_{ex}), however, requires a comment. In contrast to the case of animals, the concept of external mortality in plants is not obvious, since plant depredation leading to its instantaneous death is not as common as it is in animals, because grazing animals (or invertebrate herbivores) often only inflict partial damage and do not eat the entire plant. However, plants that maintain a small size even during the adult stage are often completely devoured. Furthermore, predator herbivores are not the only source of external mortality in plants. The pressure of parasites or other disease-causing factors, such as fungi, viruses, and bacteria, is probably even stronger. In summary, such non-intrinsic factors that increase the probability of death of a plant, except the action of herbivores, are sources of external mortality.

The evolutionary optimality principle involves the maximization of present and future reproductive success (Taylor *et al.*, 1974). This implies a straightforward use of the dynamic programming method (Bellman, 1957; McNamara and Houston, 1996), which permits finding of the optimal fractions of energy allocated to growth, reproduction, and production of defensive substances at each instant in life, starting from its end and moving back towards the beginning. In a population at equilibrium, which I assume here (see Gadgil and Bossert, 1970), the future reproductive success at age $t = T - 1$ is that calculated for age $t = T$, taking into account the probability of surviving between $T - 1$ and T. Total (present and future) reproductive value (objective function) is given by the recursive equation that also defines the algorithm of the numerical solution:

$$V(w_{T-1}, S_{T-1}, T-1, T) = \max_U \left\{ \int_{T-1}^{T} u_2 P(w(x))dx + \max_U [V(w_T, S_T, T, T)] \right\} p(T) \tag{7}$$

where x is the plant's age, T is maximum physiological lifespan, $V(w_{T-1}, S_{T-1}, T-1, T)$ is state-vector-dependent expected reproductive success at the instant $T-1$ (present and future reproductive value), $V(w_T, S_T, T, T)$ is state-vector-dependent future reproductive value in the next time interval for an individual that survives until time T, $p(T) = e^{-(q_{ex}+m)}$ is the probability of survival from age $T-1$ to age T, and $\overset{\max}{U}$ is the value of the control vector that maximizes the net reproductive output in time unit T for a plant of size w_T. Moving backwards, $T-1$ and T are exchanged with $T-2$ and $T-1$ in the next step, and so on towards the beginning of life. The transition from the set of state variables $\{w_{T-1}, S_{T-1}\}$ to the set $\{w_T, S_T\}$ depends on the strategy attained, i.e. the proportions of energy invested in each sink.

I set the initial vegetative size to $w(0) = 1$ and the concentration of defensive substances at sprouting to $S(0) = 0$. I assume that reproductive energy is liberated at the beginning of the next time interval. I numerically solve equation (3) for the time interval $(t, t+1)$ using the Runge-Kutta method (Press *et al.*, 1989). [For a detailed description of the dynamic programming algorithm see, for example, Bellman (1957), McNamara and Houston (1996), and Janczur *et al.* (2000).]

NUMERICAL EXAMPLES

Concentration of defensive substances and lifetime reproductive success

Optimal energy allocation is represented here as the relative height of the area under the survival curve, i.e. an arithmetic product of the proportion of energy invested in each process and the probability of survival until a given age (Figs. 3 and 4). One can see not only the energy allocation schedule, but also the proportion of individuals in the population with a given energy allocation (Cichoń, 1997). The solid line represents the concentration of defensive substances in the vegetative tissue, resulting from the allocation pattern, herbivory, and growth. In the linear model (Fig. 3), the optimal concentration of defensive substances in the vegetative tissues increases quickly early in life until it reaches a concentration higher than concentration C_K sufficient to entirely repel the herbivores (oversteering). Afterwards, through decreasing fluctuations, the concentration of defensive substances drops to C_K and remains at this level, providing complete protection against herbivory. This pattern is independent of the values of α, h, and q_{ex}. For the same intensity of herbivory, a low intrinsic production cost of defensive substances (high α) promotes a faster increase in these substances in the body than a moderate one, even though in the former case less allocation to defensive substances is optimal (compare Figs. 3a and 3c). This is also true for the negative exponential model (compare Figs. 4a and 4c). In the exponential model, the maximum concentration of defensive substances varies for different combinations of α and g, because the rate of herbivory decreases slowly to zero with increasing defensive substances, and there is no threshold concentration providing full protection against herbivores. When allocation to growth after maturation is optimal, the concentration of defensive substances decreases until allocation to growth stops completely, because growing tissues 'dilute' defensive substances and such substances are never produced after maturation (Figs. 4c, d, f). The concentration of defensive substances increases when α increases and – for a given α – decreases with a higher g (Figs. 5a, b). The concentration of defensive substances is higher when herbivory pressure is higher and is inversely correlated with lifetime reproductive success, because higher herbivory pressure implies a lower value

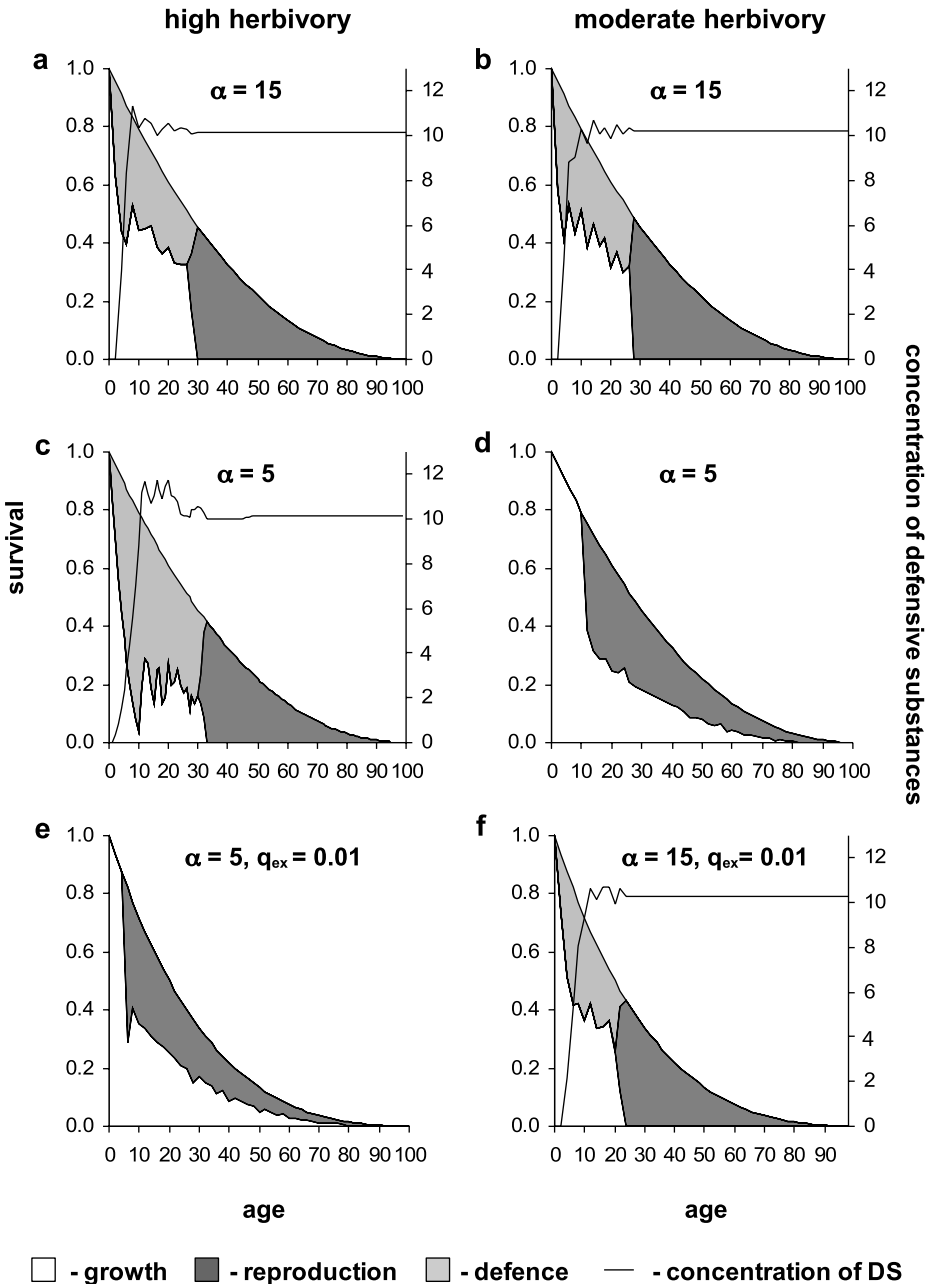

Fig. 3. Examples of optimal energy allocation in the linear model under high ($h_{max} = 0.2$) and moderate ($h_{max} = 0.1$) herbivory pressure for plants characterized by different intrinsic defensive substance production efficiencies (α) and living under different external mortality rates (q_{ex}). If not mentioned, external mortality is maintained at a low level ($q_{ex} = 0.0001$). The solid line represents the concentration of defensive substances. To illustrate the allocation of energy in an age-structured population, proportions of energy are placed under the survival curve. It is not optimal to produce defensive substances when either herbivory rate is moderate (d) or external mortality is high (e).

of the latter (Figs. 5a, c and b, d). Lifetime reproductive success is higher when herbivory pressure is moderate and is never equal for resistant and non-resistant plants (Figs. 5c, d).

External mortality also affects the concentration of defensive substances in young plants: when defensive substances are cheap (high α), higher external mortality promotes a slower increase in defensive substances (compare Figs. 3b and 3f). When defensive substances are costly, non-resistance is optimal when external mortality and herbivory pressure are high (compare Figs. 3c and 3e).

Optimal energy allocation

Figures 3 and 4 show fluctuations of the proportions of energy allocated to different sinks before maturation. These fluctuations are the result of somatic tissue loss due to herbivory: the plant should continuously adjust its allocations to a new vegetative size. Here I show the results of the model that assumes either linear (Fig. 3) or exponential (Fig. 4) effects of defensive substances on herbivory. It is evident that the optimal energy allocation depends on the intrinsic properties of the plant to produce toxic substances (α) as well as on herbivory pressure (h_{max}) and external mortality rate (q_{ex}). When allocation to defensive substances is optimal, a simultaneous allocation to growth is also optimal. When allocation to defensive substances does not occur, simultaneous allocation to growth and reproduction after maturation is optimal (Figs. 3d, e). Optimal allocations for the exponential model are qualitatively similar (Fig. 4). Interestingly, in the exponential model, some combinations of the above parameters bring about a simultaneous allocation to growth and reproduction after maturation even when allocation to defensive substances occurs earlier, although the fraction allocated to growth after maturation is very low (Figs. 4b, c, d, f). The period of mixed allocation to growth and reproduction is longer when either α and g are lower or herbivory intensity is higher (Figs. 5e, f). Note that the highest bars (Figs. 5e, f, left-hand side) represent non-resistant plants that lose their tissues and allocate energy simultaneously to growth and reproduction during most of their lifespan.

The production of defensive substances always ceases or almost ceases at maturation. This is an outcome of the assumption that defensive substances are stable compounds (e.g. tannins) and their replenishment is not necessary. Interestingly, in none of the cases does the allocation to the production of defensive substances stop before maturation. However, allocation to defensive substances does not always start just after germination in the exponential model (Figs. 4d, f). In other words, to be optimal, an immature plant should start allocation to defensive substances either at germination or later, and extend such allocation to maturation.

For all combinations of parameters when herbivory is absent ($h_{max} = 0$), the allocation to the production of defensive substances is not optimal. In this case, a 'bang–bang' switching from growth to reproduction occurs as predicted by general life-history theory. In the linear model, high herbivory pressure ($h_{max} = 20\%$) makes the allocation to defensive substances optimal for either high or moderate intrinsic defensive substances production efficiency (α = 15 and α = 5 respectively; compare Figs. 3a, b, c). When herbivory pressure is moderate ($h_{max} = 10\%$), allocation to defence is optimal when the plant's intrinsic production of defensive substances is energetically efficient (α = 15; Fig. 3b), and is not optimal when this efficiency is moderate (α = 5; Fig. 3d). Interestingly, in the latter case, simultaneous energy allocation to growth and reproduction occurs (Fig. 3d). For a given herbivore pressure, less energy is used to provide full protection if α is high (compare Fig. 3a and Fig. 3c), whereas

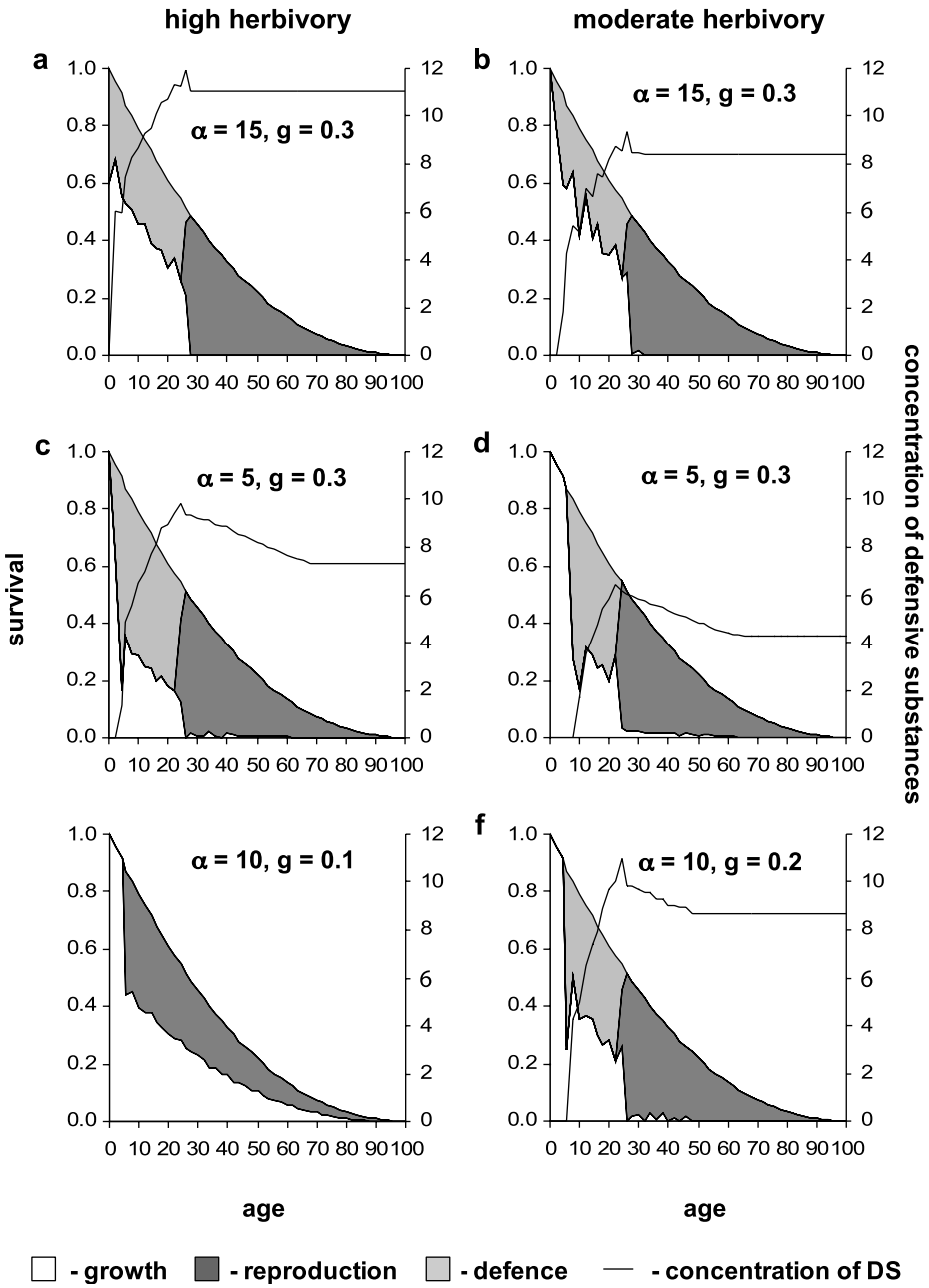

Fig. 4. Examples of optimal energy allocation in the exponential model under high ($h_{max} = 0.2$) and moderate ($h_{max} = 0.1$) herbivory rate for plants characterized by different intrinsic defensive substance production efficiencies (α) and different efficiencies of defensive substances to repel herbivores (g). External mortality is maintained at a low level ($q_{ex} = 0.0001$). The solid line represents the concentration of defensive substances. To illustrate the allocation of energy in an age-structured population, proportions of energy are placed under the survival curve. Some combinations of α and g produce simultaneous energy allocation to growth and reproduction after maturation (b, c, d, f). It is not optimal to produce inefficient defensive substances (with low g) even when the intrinsic production cost of defensive substances is low, i.e. α is high (e).

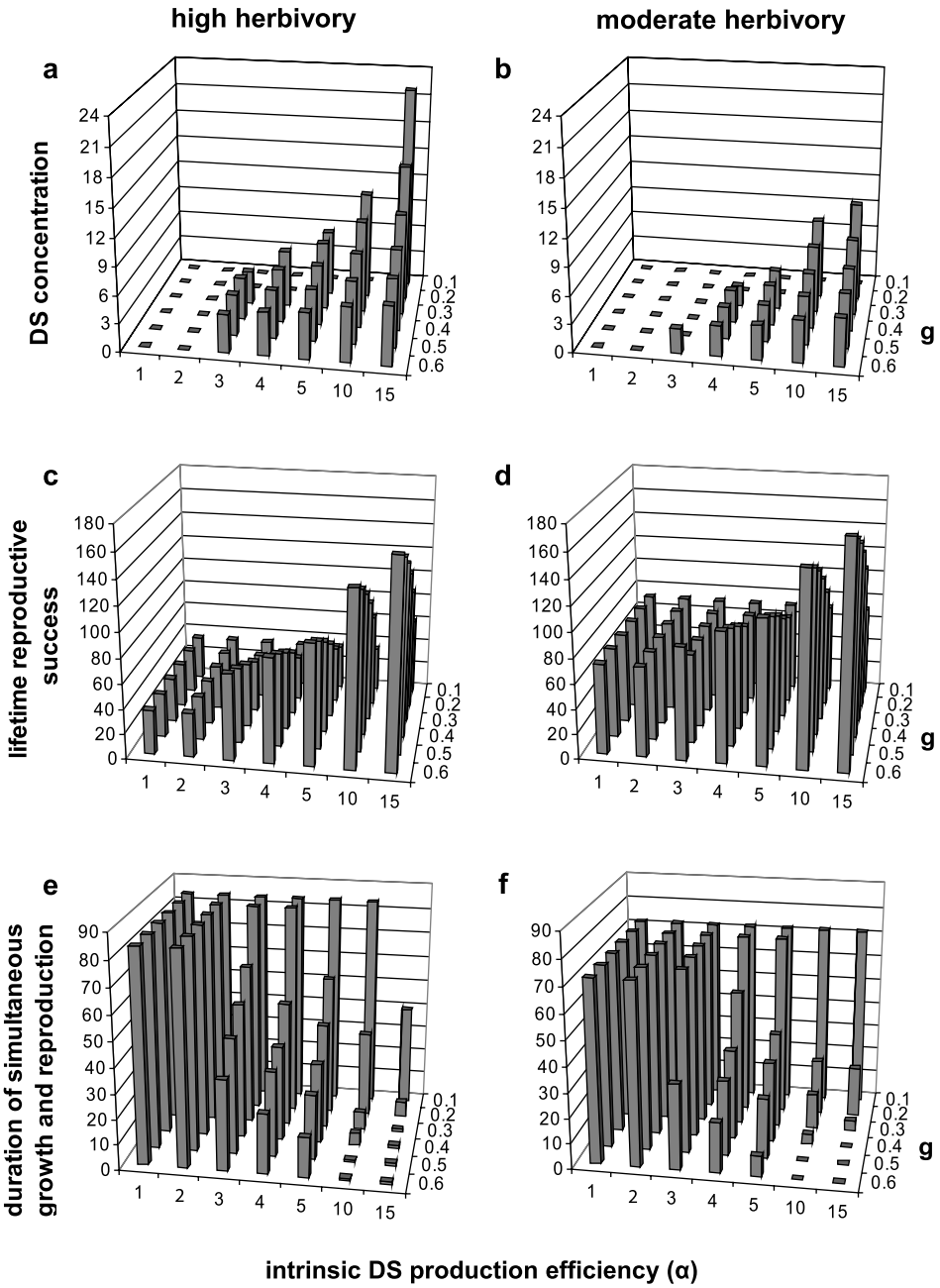

Fig. 5. Concentration of defensive substances (DS content/vegetative size in energy units), lifetime reproductive success (energy units), and duration of energy allocation to growth after maturation (% of the lifespan) for different intrinsic defensive substance production efficiencies (α) and efficiency of defensive substances to repel herbivores (g). The columns of the high 0 on (a) and (b) concern plants that do not produce defensive substances. External mortality is maintained at a low level ($q_{ex} = 0.0001$). An exponential effect of defensive substances on herbivores is assumed here.

herbivory pressure does not change allocation to defensive substances for high α (compare Fig. 3a and Fig. 3b).

The efficiency of defensive substances in repelling herbivores can be analysed easily in the exponential model: a higher value of the parameter g in equation (4) accounts for a faster decrease of herbivory rate with an increase in the concentration of defensive substances, i.e. higher g implies a higher effectiveness of the defence. Cheaper and more efficient defensive substances (higher α and g) make lower allocation to the defence against herbivores optimal (compare Figs. 4a and b with Figs. 4c and d), and make a shorter simultaneous allocation to growth and reproduction optimal after maturation (Figs. 5e, f, front right-hand side). When a plant produces inefficient defensive substances only (low g), the lack of allocation to defence is optimal even when defensive substances are cheap (high α) (compare Fig. 4e with Fig. 4c). Such non-resistance is optimal when the plant physiology allows for the production of inefficient defensive substances even when they are cheap (low g and high α), but also efficient defensive substances even when they are costly or moderately costly (low or moderate α) (Figs. 5e, f, left-hand and back side).

External mortality rate changes the allocation pattern. High external mortality makes the allocation to defensive substances optimal when the production of such substances is not costly (high α) and herbivory pressure is moderate (Fig. 3f). Under a moderate production cost of defensive substances (moderate α), non-resistance and the simultaneous allocation to growth and reproduction are optimal (Fig. 3e). External mortality changes the survival curve (Figs. 3e, f) as well as the age at maturity, according to general life-history theory (compare Figs. 3b–f with Figs. 3c–e). Obviously, since herbivory affects plant mass only and not mortality rate, neither herbivory rate nor the production cost of defensive substances changes the survival curve (Figs. 3a through d).

In summary, three qualitative allocation patterns exist: (i) simultaneous allocation to growth and production of defensive substances followed by pure allocation to reproduction; (ii) simultaneous allocation to growth and production of defensive substances followed by simultaneous allocation to growth and reproduction (even when allocation to the former is very low); and (iii) allocation to growth followed by simultaneous allocation to growth and reproduction without the production of defensive substances. In the third case, the period of pure growth can be very short (Fig. 3e, Fig. 4e). For each defensive substances production efficiency α under constant external mortality, there exists a herbivory pressure h_{max} below which the allocation to the production of defensive substances is never optimal; this critical h_{max} is lower for higher defensive substances production efficiency (Fig. 6).

Plant tissue dynamics

I present here plant tissue dynamics for the linear model. Optimal energy allocation affects growth. This is because enhanced allocation to defence subtracts energy from growth. Figure 7 shows that under no herbivory, the plant grows faster. The plant tissue dynamics curve for high herbivory pressure ($h_{max} = 20\%$) is qualitatively similar, but the rate of increase in tissue is slower and maturation is delayed: this can be attributed almost entirely to allocation to the production of defensive substances, since the loss of vegetative mass occurs only early in life, when the concentration of defensive substances is below the threshold concentration (Fig. 3c). The production of defensive substances prevents the plant from being eaten, thus no loss in somatic tissues is observed at the adult stage (compare $h_{max} = 0$ and $h_{max} = 0.2$ in Fig. 7a). The curves for low and moderate herbivory

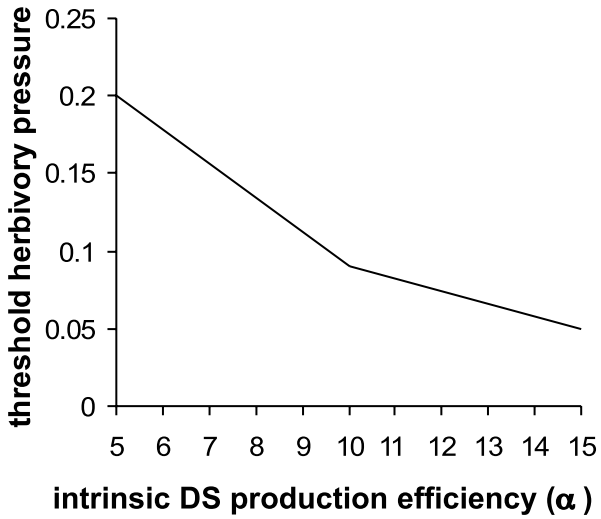

Fig. 6. Threshold herbivory pressure (vertical axis), below which the allocation to the production of defensive substances (DS) is never optimal, for plants characterized by different intrinsic defensive substance production efficiencies (α, horizontal axis). External mortality is maintained at a low level ($q_{ex} = 0.0001$). A linear effect of defensive substances on herbivores is assumed here.

pressure decrease after maturation, because it is not optimal to produce defensive substances (non-resistance). The tissue mass dynamics of the seedling is thus determined by the difference between growth and herbivory rates. After maturation, it is not optimal to replace all lost vegetative tissues, which causes a gradual decrease in mass. Increasing herbivory pressure between $h_{max} = 0.01$ and $h_{max} = 0.1$ (with no allocation to defensive substances being optimal) makes earlier maturation and more vigorous replenishment of lost tissues optimal. As a result, the line describing plant tissue mass is positioned lower, and it is flatter; also, maximum size is attained earlier in life (compare $h_{max} = 0.05$ with $h_{max} = 0.1$ in Fig. 7a). The plant's decision concerning the production of defensive substances has important implications for its actual and lifetime reproductive success. For example, a plant that produces defensive substances under high herbivory has a higher seed production for a considerable fraction of the lifespan than a plant that does not produce defensive substances under weaker herbivory pressure (compare $h_{max} = 0.2$ with $h_{max} = 0.1$ or $h_{max} = 0.05$ in Fig. 7b). Nevertheless, the latter has obviously a higher lifetime reproductive success (Fig. 7c). Figure 7c shows also that a lower intrinsic production cost of defensive substances (higher α) makes starting production of such substances optimal at lower herbivore pressure.

Interspecific level

I regard here as a plant species the combination of the intrinsic energetic efficiency of the plant to produce defensive substances (α) and the efficiency of the defensive substances to repel herbivores (g in the exponential model). Figure 5 shows the combined effect of these two parameters together with the effect of herbivory pressure on the concentration of defensive substances in the tissues, lifetime reproductive success, and duration of

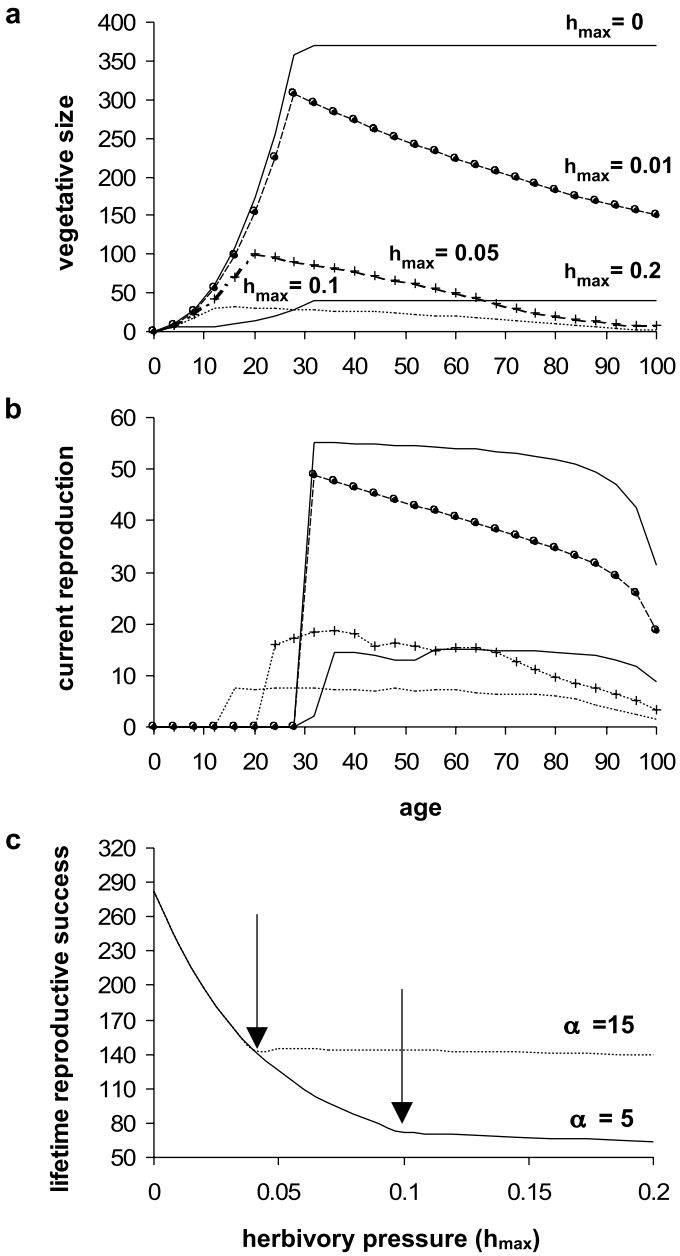

Fig. 7. Tissue mass dynamics curves (a), current reproduction (b), and lifetime reproductive success (c) of a plant living under different herbivory pressure (h_{max}). Arrows indicate the minimum h_{max} below which the allocation to the production of defensive substances is never optimal. Intrinsic production efficiency of defensive substances is maintained constant in (a) and (b) ($\alpha = 5$). Growth curves are qualitatively similar when either herbivory is absent ($h_{max} = 0$) or the production of defensive substances is optimal under high herbivory ($h_{max} = 0.2$). Non-resistance is optimal when herbivory is less intense. The loss of vegetative body under low $h_{max} = 0.1$ is compensated by a higher lifetime reproductive success for $\alpha = 5$, even when current reproduction during most of the lifespan is lower for $h = 0.1$. Lifetime reproductive success is higher for a broader interval of h_{max} in plants that defend themselves at lower cost ($\alpha = 15$). A linear effect of defensive substances on herbivores is assumed here. External mortality is maintained at a low level ($q_{ex} = 0.0001$).

simultaneous allocation to growth after maturation. The latter variable was registered as the moment of the last allocation to growth when fluctuations occurred. For non-resistant species, this period is frequently extended to almost all of the lifespan (Fig. 4e). For resistant species, it differs from zero when energy is allocated simultaneously to growth and reproduction after maturation (Figs. 4c, d, f). Each bar represents an optimal species with a characteristic combination of α, g, and h_{max}. By comparing the same combination of α and g in the left and in the right column, one can observe the response of different plant species to increased herbivory pressure. The optimal concentration of defensive substances is higher when herbivory pressure is higher, internal efficiency to produce defensive substances is better (i.e. defensive substances are cheaper), and protection by defensive substances is less efficient (Figs. 5a, b, back right corner). The proportion of species that do not produce defensive substances is lower when herbivory pressure is higher (Figs. 5a, b, left-hand side and backward part), i.e. at higher herbivory, higher variability of defence strategies is observed.

Lifetime reproductive success is always higher when the parameters make defence optimal; however, it is inversely correlated with the concentration of defensive substances (compare Figs. 5a, b and Figs. 5d, e, left-hand side). Higher herbivory pressure promotes a longer duration of allocation to growth after maturation when defence is not optimal (Figs. 5c and f, left-hand side). Moderate values of α and g, as well as higher herbivory pressure, promote simultaneous allocation to growth and reproduction after maturation.

DISCUSSION

Cost of defence

One of the aims of this study was to assess defence against herbivores from the life-history point of view, i.e. explaining when an optimal plant should invest energy in such protection. However, I also highlight other life-history implications of herbivory, including optimal adult plant size, optimal age at maturity, and optimal growth dynamics under different herbivory and mortality rates, that are outcomes of the costs assumed. There is empirical evidence of the cost of defence. Lewis *et al.* (2006) compared the native European population of *Alliaria petiolata* with that introduced in North America. Plants from the native population produced more defensive substances (sinigrin) and were smaller. In their introduced range, plants experienced reduced herbivory. Rudgers *et al.* (2004) found a strong negative correlation between investment in gossypol glands and investment in trichome cover across the clade Gossypieae that could be attributable to the costs of defence, negative genetic correlation or redundancy of their anti-herbivore action. Hoballah *et al.* (2004) reported a similar negative relationship between the intensity of induced emission of volatile defensive substances and plants' dry mass when comparing six maize inbred lines differing in the extent of emission of volatile defensive substances. The lines that released a larger amount of volatile defensive substances also contained more non-volatile terpenoids. Additionally, plants induced to produce volicitin exhibited a small but significant reduction in seed production. Strauss *et al.* (2002) reported that 82% of studies with a controlled genetic background demonstrated significant fitness reduction associated with resistance.

Age-dependent concentration of defensive substances

Iwasa and colleagues' (1996) dynamic optimization model shows that the concentration of defensive substances is higher in younger leaves. They studied the effect of either negative exponential or hyperbolic defence function on optimal defence, and maximized the plant's growth as an increase in the rate of a population of leaves of different ages. They assumed that defensive substances are non-degradable compounds, reallocated at no cost from older to younger leaves, and that the rate of photosynthesis decreases with a leaf's age to virtually zero. To make the model analytically tractable, the whole plant grows exponentially. I do not assume here any particular growth curve, but rather the shape of the production equation (1): the growth curve is an effect of optimal energy allocation. Similar to the model of Iwasa *et al.* (1996), when protection against herbivores is not complete, the concentration of defensive substances is lower for older plants (Figs. 4c, d, f). However, the cause is different: in their model it is an effect of both a lower photosynthesis rate in older leaves as well as of the reallocation of defensive substances to younger leaves, whereas in the present model it results from 'dilution' of the defensive substances in the plant's tissue due to allocation to growth after maturation. This 'dilution' is an effect of non-linearity of the defence function (Fig. 2b) that produced simultaneous allocation to growth and reproduction after maturation: for the same intrinsic defensive substances production efficiency (α), a slower decay in the rate of herbivory with increasing concentration of defensive substances promotes a longer simultaneous allocation period (Figs. 5e, f). Contrary to their model, the present model shows that generally very young plants will have lower concentrations of defensive substances in their tissues. Indeed, in the entire leaf set of *Erica australis*, *Cistus laurifolius*, *Quercus pyrenaica*, *Rosa canina* (Ammar *et al.*, 2004), *Q. serrata*, *Q. semecaprifolia*, *Q. glauca*, and *Q. ilex*, the concentration of condensed tannins was lower at the beginning of the season before increasing later (Makkar *et al.*, 1990). Likewise, the concentration of ellagitanin (Salminen *et al.*, 2002) or total tannins (Feeny, 1970) in leaves of *Q. robur*. Cornelissen and Fernandes (2001) observed lower tannin concentrations and 60% higher attack frequency by chewing herbivores on young than on mature leaves of *Bauhinia brevipes*. A similar tendency for phenolic compounds was observed in *Acacia tortilis* (Gowda and Palo, 2003) and in *Q. glauca* (Makkar *et al.*, 1990). Several studies reported opposite results. Van Dam and co-workers (1994, 1995, 1996) found that young leaves of *Cynoglossum officinale* contained higher concentrations of pyrrolizidine alkaloid. Salminen *et al.* (2004) observed higher concentrations of most of the hydrolysable tannins in young than in mature leaves of *Q. robur*. The model shows that these results are very unlikely when defensive substances are absent in seeds: before maturation, an optimal plant should grow and produce defensive substances simultaneously and a certain time interval is required for such substances to reach a threshold concentration, even when it is likely to be very short. Interestingly, in the study by Salminen *et al.* (2004), two compounds from the hydrolysable tannin group had a dynamic similar to that predicted by the model: their concentration increased very quickly early in life, reached a peak, and decayed later on (Figs. 4c, d, f). Higher concentrations of defensive substances in younger leaves, as evidenced by some studies, is probably an effect of: (i) non-null concentration of defensive substances in the seed, (ii) very rapid increase in defensive substances in young plants not perceived by the researchers, and/or (iii) non-linear shape of the defence function (Fig. 2b) resulting in allocation to growth after maturation and thus a 'dilution' of defensive substances in the tissues. In such cases, the concentration of defensive substances is lower in older than in younger plants (but not in very young ones; Figs. 4c, d, f).

Additionally, the lack of an age-dependent decrease in photosynthesis rate in my model may be responsible for the low concentration of defensive substances at the very early stage. The inclusion of this feature would probably shift the maximum concentration of defensive substances towards the beginning of life.

Induced vs. constitutive defence

In a study of three plant species, van Dam *et al.* (1993) hypothesized that the differences in response among species are adaptations associated with the amount of herbivory. As the increase in the concentration of defensive substances in one of the species (*Cynoglossum officinale*) took place after the plants had been severely damaged, the authors attributed it to induced defence. In another study on the same species (van Dam and Vrieling, 1994), the authors concluded that the observed increase in alkaloid content was attributable to its transport within the plant (the entire amount in the plant did not change).

The problem of induced versus constitutive defence is a part of a broader theory of defence that also involves the immune response against pathogens in animals. Shudo and Iwasa (2001) examined the response of an animal organism to pathogens and parasites. They modelled explicitly the allocation to both of these kinds of defence, and examined when each of them is optimal. The criterion of optimality is the minimization of the total cost of defence (damage caused by pathogens and the cost caused by the defence response). In their model, the constitutive defence is adopted when it is more effective and less expensive than the induced defence, the time delay of the response is large, and the pressure of the pathogen is strong (attacks more frequent, its initial abundance is large, growth rate high, and damage severe). In my model, the criterion of optimality is the maximization of lifetime reproductive success. Contrary to their model, I do not define here explicitly whether the defence is induced or not; however, the model predicts results that are perceived as an induction of defensive substances. One possible explanation is a phenotypic plasticity of energy allocation to the defensive substances of a plant characterized by a determined intrinsic property to produce defensive substances (α). For example, a plant with moderate α does not produce defensive substances under moderate herbivory but does invest in such substances under high herbivory (compare Fig. 3d with Fig. 3c). We can easily imagine that herbivory is not maintained constant during life, and that optimal energy allocation is 'adjusted' to its changing levels at each moment of life. It is thus reasonable to assume that α could not change during life, because it is genetically fixed (e.g. Vrieling *et al.*, 1993; van Dam and Vrieling, 1994; Rousi *et al.*, 1996; Agrawal *et al.*, 1999). In other words, a plant with a constant intrinsic property to produce defensive substances may change the rate of production of such substances depending on herbivory pressure.

Obviously, the model can be modified in such a way that the intrinsic property to produce defensive substances is not genetically fixed, but rather the proportion of energy allocated to each physiological activity: this would resemble the constitutive defence. In this case, a plant would have the same allocation pattern independent of the levels of both herbivory and external mortality. Indeed, *Catharanthus roseus* in the study of van Dam *et al.* (1993) did not induce defensive substances after severe damage.

Resistance vs. non-resistance (tolerance)

Some cases of tolerance or non-resistance can be explained in terms of optimal energy allocation and phenotypic plasticity. The model predicts that selection is expected to favour non-resistance and elevated levels of damage under low herbivory, which is concordant with the results of Tiffin and Rausher (1999) for *Ipomea purpurea*. Under higher rates of herbivory, resistance will be favoured, even when it diverts energy from reproduction. That is because at higher α, the production of defensive substances (resistance) at moderate and high levels of herbivory results in higher lifetime reproductive success than non-resistance (Fig. 7c, the interval between arrows).

Interestingly, the problem of resistance and tolerance can be extended also to the immune response and the production of defensive structures in animals. Shudo and Iwasa (2004) state that the optimal control of an immune activity often does not result in complete elimination of pathogens from the host. When the population of pathogens is low, the specific immune cells are not produced (non-resistance). Also, when predation pressure is low or the cost of defence is high in molluscs, the optimal strategy is to have no shell (Irie and Iwasa, 2003, 2005). Both cases resemble the non-resistance in plants when herbivory pressure is moderate and the production of defensive substances is moderately costly (Fig. 3d).

Herbivory at the interspecific level

Different sets of parameters regarding the physiological intrinsic properties of the plant to respond against herbivory are treated here as different species. For example, a combination of high values of both α and g describes a plant that produces cheap and effective defensive substances, and a combination of low α and high g describes a plant that produces expensive and effective defensive substances. The results suggest that the production of defensive substances in plants can be explained in terms of lifetime optimal energy allocation. Some numerical results are non-intuitive. For example, more damaged plants or a higher number of non-resistant species are expected under low herbivory pressure. Analogously, lower vegetative tissue injury and larger numbers of resistant species are expected under higher herbivore pressure (Fig. 5). This is an effect of optimal energy allocation: moderate herbivory makes the allocation to defence non-optimal and vice versa.

The model predicts that there will be more species with similar lifetime reproductive success at moderate rather than at high levels of herbivory. Since an optimal plant species needs time to replace a sub-optimal species (e.g. Kozłowski and Janczur, 1994), variations in herbivory pressure will promote species with different sets of the parameters α and g at different levels of stress (Figs. 5c and d). Only a few species that produce cheap and non-effective defensive substances and have a high concentration of such substances will be found at high herbivory (Figs. 5a and c). With moderate herbivory, highly toxic plants are very unlikely (Fig. 5b). Selection will favour plants that produce cheap and effective defensive substances, since their lifetime reproductive success is much higher. Plants that produce cheap and non-effective defensive substances will have a lifetime reproductive success similar to that of plants that produce moderately cheap and effective defensive substances at both high and moderate herbivory pressure (Figs. 5c and d).

Implications for field studies

The numerical results of the model highlight some implications for future field studies. For example, shapes of the defence function should be established in the field: if they are negative-exponential, it can explain one of the possible causes of the simultaneous allocation of energy to growth and reproduction, as well as some instances of a decrease in the concentration of defensive substances during the vegetative season. If we find badly damaged plants in the field, it does not necessarily mean that the herbivory pressure is high there. And, if we find plants with very little damage, it does not necessarily mean that there is low herbivory pressure in the environment. Severe damage occurs presumably because herbivory pressure is low or the external mortality of entire plants is high. It is not optimal to allocate energy to defence in such cases, because minor losses in body tissue produce a lower fitness decline than diverting energy to the production of defensive substances under low herbivory rate (Figs. 6 and 7). On the other hand, allocation to defence under high mortality delays maturity, and a plant that tries to allocate energy to defensive substances could die before it reproduces, or its reproductive output will be lower due to a shorter reproductive period. The external mortality of a plant is a factor generally neglected in studies of plant–herbivore interactions. If we find cases where plants with a very high concentration of cheap and non-effective defensive substances are predominant, it probably means that some constraint exists that prevents plants producing cheap and effective defensive substances even when herbivory pressure is high.

Under high herbivory, it is optimal to produce defensive substances: a plant would lose too high a proportion of somatic tissues and thus its production rate would be very low (since it is size-dependent).

The model shows that emphasis should be placed on the quantification of defensive substances at the early stage of the plant's (leaf's) life: as such substances increase very quickly during growth, the plant's (leaf's) early dynamics is frequently omitted.

The results of the model are concordant with some empirical studies. The model draws attention to the fact that the energy costs of plant defence should be estimated in future work.

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