



HAL
open science

The shade avoidance syndrome: A non-markovian stochastic growth model

Andrea Veglio

► **To cite this version:**

Andrea Veglio. The shade avoidance syndrome: A non-markovian stochastic growth model. Journal of Theoretical Biology, 2010, 264 (3), pp.722. 10.1016/j.jtbi.2010.02.039 . hal-00591230

HAL Id: hal-00591230

<https://hal.science/hal-00591230>

Submitted on 8 May 2011

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Author's Accepted Manuscript

The shade avoidance syndrome: A non-markovian stochastic growth model

Andrea Veglio

PII: S0022-5193(10)00114-1
DOI: doi:10.1016/j.jtbi.2010.02.039
Reference: YJTBI5892

To appear in: *Journal of Theoretical Biology*

Received date: 19 November 2008
Revised date: 13 January 2010
Accepted date: 23 February 2010

Cite this article as: Andrea Veglio, The shade avoidance syndrome: A non-markovian stochastic growth model, *Journal of Theoretical Biology*, doi:[10.1016/j.jtbi.2010.02.039](https://doi.org/10.1016/j.jtbi.2010.02.039)

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting galley proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.



www.elsevier.com/locate/jtbi

The shade avoidance syndrome: a non-markovian stochastic growth model

Andrea Veglio

Department of Oncological Sciences and Division of Vascular Biology, Institute for Cancer Research and Treatment, University of Torino, Str. Prov. 142 Km 3.95., 10060 Candiolo, Italy.

National Inter-University Consortium for the Physical Sciences of Matter (CNISM), Corso Duca degli Abruzzi 24, 10129 Torino, Italy.

Abstract

Plants at high population density compete for light, showing a series of physiological responses known as the shade avoidance syndrome. These responses are controlled by the synthesis of the hormone auxin, which is regulated by two signals, an environmental one and an internal one. Considering that the auxin signal induces plant growth after a time lag, this work shows that plant growth can be modelled in terms of an energy-like function extremization, provided that the Markov property is not applied. The simulated height distributions are bimodal and right skewed, as in real community of plants. In the case of isolated plants, theoretical growth dynamics and speed correctly fit *Arabidopsis thaliana* experimental data reported in literature. Moreover, the growth dynamics of this model is shown to be consistent with the biomass production function of an independent model. These results suggest that memory effects play a non-negligible role in plant growth processes.

1. Introduction

Ecologists have been concerned with competition in plants since the beginning of the last century. Clements (1905) published the first formal definition of competition in plants, defining it as “the relation between plants occupying the same area and dependent upon the same supply of physical factors”, and later (Clements et al., 1929) he considered that competition begins “when the immediate supply of a *single factor necessary* falls below the combined demand of the individual plant”.

Since the 50s a number of authors have systematically studied the effect of competition in plant systems. For example, Donald (1951) investigated in a seminal work the intra-specific light competition in pastures plants. More recently, Pacala et al. (1996) report experimental data providing little evidence of competition for nitrogen and water, and suggesting that the most important factor determining competition in sufficiently manured and watered plants is light. Accordingly, the present work will assume no stress due to insufficiency of nutrients or water (for models on dryland vegetation, see Kletter et al. (2009) and refs therein) and will study light competition in plants grown at high population density, i.e. experiencing density stress. In particular, it will be concerned with intra-specific (inter-plant) light competition. The series of physiological responses demonstrated by plants experiencing density stress is collectively known as the *shade avoidance syndrome* and comprises effects such as stem elongation at

expense of leaf and storage organ expansion, inhibition of branching, and acceleration of flowering (Tao et al., 2008).

There are two main ‘fingerprints’ characterizing the height distribution of a community of plants under density stress:

- i.* bimodality: bigger individuals grow more than smaller ones, thus become bigger and bigger, whilst smaller individuals grow much less (Ford and Diggle, 1981).
- ii.* hierarchy of exploitation (right skew): there is a large number of plants slightly smaller than the mean, and a small number of plants much greater than the mean, *i.e.* the distribution is right (positively) skewed (Harper, 1967).

The present work is based on the consideration that growth responses in plants at high population density are controlled by the synthesis of several phytohormones, the most important being auxin (Friml and Sauer, 2008; Teale et al., 2006). Auxin induces plant growth after some delay (Taiz and Zeiger, 2002), and recent experimental results (Tao et al., 2008; Stepanova et al., 2008) show that the auxin pathway is mainly modulated by two signals, an environmental one and an internal one (or, using the vocabulary of Guédon et al. (2007), ontogenetic). The environmental signal consists in the decrease in the red to far-red ratio of incoming light. In fact, plants grown at high population density perceive both absorption of red light by canopy leaves and reflection of far-red light from neighbouring plants (Tao et al., 2008). In other words, a plant ‘recognizes’ to be surrounded by other shading neighbours and responds producing auxin which induces growth after some time lag. On the other hand, the internal signal is mediated by the hormone ethylene (Stepanova et al.,

Email addresses: andrea.veglia@unito.it (Andrea Veglio)

2008), which stimulates the production of auxin even in isolated plants.

A countless number of models have been developed to study both inter- and intra-specific competition among plants. Reviewing the different spatial community dynamics models, Bolker et al. (2003) group them in three different frameworks, namely:

a. Interacting particle systems (IPS), which are stochastic, markovian (Neuhauser, 2001), continuous time models with discrete individuals located in the cells of a regular lattice (see, for example, Diggle (1976); Durrett and Levin (1994); Hendry et al. (1996)).

b. Stochastic point processes, which track discrete individuals that compete locally, but assume that they occupy a single point in continuous space (see, for example, Gandhi et al. (1998); Bolker and Pacala (1999); Dieckmann and Law (2000)).

c. Patch models, which track group of individuals, allowing (noncontiguous patch models) or not (metapopulation or patch-occupancy models) multiple individuals and multiple species per patch (see, for example, Levins and Culver (1971); Gilad et al. (2004, 2007)).

Strengths and weaknesses of each of the above frameworks have been exhaustively analyzed by Bolker et al. (2003).

In this paper, I propose a model that studies the old problem of the light competition in terms of the old method of the energy-like function extremization, but does not belong to any of the previous frameworks. In fact, even sharing some features with other IPS models, the model presented here is non-markovian.

Indeed, markovianity is a very nice property in stochastic systems. It assures that in a system the future state depends only on the present state and is independent of past states (van Kampen, 2007). In other words, the system has no memory and this considerably simplifies calculations. Unfortunately, auxin production induces plant growth after a time lag, and at the proper time scale this memory effect forbids modelling the system by exploiting the Markov property.

I will show that in the present model the non-markovianity is necessary:

- to reproduce plant height distributions that are bimodal;
- to correctly mimic experimental growth dynamics in isolated plants;
- to be consistent with the biomass production model by Cournède et al. (2008).

I will consider the total amount of auxin present in a plant, but not its spatial distribution in plant's body, i.e. the well characterized phenomenon of the auxin gradient (see Tanaka et al. (2006) and ref.s therein). For recent models on the auxin gradient the reader is referred to Jönsson et al. (2006); de Reuille et al. (2006); Smith et al. (2006); Newell et al. (2008).

2. The model

As mentioned in the introduction, auxin is the main regulator of plant growth. For plants growing at high population density, auxin synthesis is regulated by:

1) an environmental signal due to the absorption of red light by canopy leaves and reflection of far-red light from neighboring plants (Tao et al., 2008);

2) an internal signal, which makes plants growing also when they do not experience density stress (Stepanova et al., 2008).

Consider a community of plants placed on a 2-dimensional $N \times N$ square lattice. The lattice has N^2 nodes, each corresponding to a plant labelled as (i, j) , with $i, j = 1, \dots, N$. Each (i, j) -plant is associated to its own amount of auxin $s_{ij}(t)$ at time t . Each plant shades, i.e. interact with, its neighbours by which conversely it is shaded. I will consider only the interaction between first-nearest neighbouring plants. Indeed, trials with second-nearest neighbour interactions showed that the results do not change substantially.

The auxin amount of a plant is converted into biomass after a delay (Taiz and Zeiger, 2002) that I will call α : if a certain auxin amount is produced by a plant at time t , then at time $t + \alpha$ the plant shoots to grow proportionally to the produced quantity of auxin (in this paper I will take into account only vertical shooting, i.e. I will assume the height of each plant to be proportional to its weight.) Accordingly, I will define the *shade avoidance potential* $a_{ij}(t)$ as the function representing the predisposition at time t of the (i, j) -plant to avoid shade by shooting at time $t + \alpha$. Its definition needs some steps.

For simplicity, first consider an ideal subsystem of two plants, say the (i, j) - and the $(i + 1, j)$ -plant, subjected only to the environmental signal I .

The predisposition at time t of the (i, j) -plant to shoot at time $t + \alpha$ depends first of all on its own auxin amount $s_{ij}(t)$. In fact, the greater $s_{ij}(t)$ is, the more the (i, j) -plant has the possibility to grow at time $t + \alpha$.

Moreover, the (i, j) -plant perceives an environmental signal from the $(i + 1, j)$ -plant, that absorbs red light and reflects far-red light. This signal “warns” the (i, j) -plant of the presence of its $(i + 1, j)$ neighbour. The greater the $(i + 1, j)$ auxin amount $s_{i+1j}(t)$ is at time t , the more $(i + 1, j)$ will shade the (i, j) -plant at time $t + \alpha$. Because the (i, j) -plant “wants” to avoid the $(i + 1, j)$ shade, its predisposition at time t to shoot at time $t + \alpha$ has to depend also on the auxin amount $s_{i+1j}(t)$ of its neighbour. In fact, the greater $s_{i+1j}(t)$ is, the more the (i, j) -plant has to grow to avoid the shade of the $(i + 1, j)$ -plant at time $t + \alpha$.

Therefore, for a system of two plants subjected only to the environmental signal, the predisposition at time t of the (i, j) -plant to shoot at time $t + \alpha$, i.e. the shade avoidance potential $a_{ij}(t)$, reads

$$a_{ij}(t) = J s_{ij}(t) \cdot s_{i+1j}(t). \quad (1)$$

J is a non-negative constant that expresses how strong plants shade each other or, in other words, how strong they have to compete for light. Because in a generic system of N^2 elements the (i, j) -plant has more than one first-nearest neighbour (namely, two if (i, j) is at the corner of the lattice, three if it is at the border, four if inside), the (i, j) -plant shades and is shaded by all its first neighbours. Therefore, all the contributions from the first-nearest neighbour have to be taken into account in the shade avoidance potential, i.e.

$$a_{ij}(t) = J s_{ij}(t) \sum_{n.n.} s_{ij}(t), \quad (2)$$

where $\sum_{n.n.} s_{ij}(t) \equiv s_{i-1j}(t) + s_{i+1j}(t) + s_{ij-1}(t) + s_{ij+1}(t)$ is the sum over the first nearest neighbour of the (i, j) -plant.

Eq. (2) takes into account only the environmental signal 1) perceived by the (i, j) -plant; if it had no neighbours, its shade avoidance potential would be zero, i.e. it would have no chance to produce biomass. Since isolated plants grow, the internal signal 2) can be taken into account by adding a term proportional to the auxin amount in the plant, $h s_{ij}(t)$, where h is a non-negative constant expressing the strength of the signal, i.e. how high the plant would grow if isolated. Therefore, the complete function expressing the shade avoidance potential of the generic (i, j) -plant reads

$$a_{ij}(t) = h s_{ij}(t) + J s_{ij}(t) \sum_{n.n.} s_{ij}(t). \quad (3)$$

Notice that the greater the shade avoidance potential is at time t , the more the (i, j) -plant will be able to shoot at time $t + \alpha$, i.e. the more it will be able to intercept light and avoid shade.

Studying the system in the mean field approximation, it is worth considering the global shade avoidance potential $A(t) \equiv \sum_{i=1}^N \sum_{j=1}^N a_{ij}(t)$. Because all the plants of the community contribute with their mutual shading, $A(t)$ is a global quantity that represents the predisposition of the whole system to avoid shade by shooting after the time lag or, in other words, the mean shade avoidance potential up to a N^{-2} factor. $A(t)$ reads

$$A(t) = \sum_{ij} \left(s_{ij}(t) \left[h + J \sum_{n.n.} s_{ij}(t) \right] \right), \quad (4)$$

where the sum $\sum_{ij} \equiv \sum_{i=1}^N \sum_{j=1}^N$ runs over all the N^2 plants. In passing, notice that Eq. (4) is formally analogous to the Ising hamiltonian¹ (Huang, 1987).

Accordingly to what discussed above, it is reasonable to assume that on average plants try to maximize the shade avoidance potential: the greater is the global potential at time t , the maximal will be the light exposure of the community at time $t + \alpha$.

Therefore, at each time step t the auxin amount of a randomly selected (i, j) -plant may be increased only if the global shade avoidance potential is maximized, as follows:

$$s_{ij}(t+1) = s_{ij}(t) + \rho \xi(t) e^{-bt} \Theta[A(t) - A(t - \alpha)]. \quad (5)$$

$\xi(t) \in [-1, 1]$ is a randomly drawn number with flat distribution, $\rho > 0$ the maximum updating amplitude for the selected auxin amount, $1/b > 0$ the equilibrium time². $\Theta[\cdot]$ is the Heaviside step function, defined as $\Theta[x] = 0$ if $x \leq 0$ and $\Theta[x] = 1$ if $x > 0$. The exponential term e^{-bt} has been chosen to let the growing process dramatically slow down once reached the equilibrium time $1/b$. The positive discrete variable α represents the time lag introduced above and is calculated according to the flowchart in Fig. 1. Because at the very beginning of a plant life (dormant seed) the auxin amount is negligible, it is reasonable to choose as initial condition $s_{ij}(0) = 0$.

Eq. (5) states that, at each time step, the selected auxin amount s_{ij} can be updated by a randomly drawn quantity (which gets smaller and smaller as time goes by because of the exponential term) only if such an update implies the maximization ($\Theta[A(t) - A(t - \alpha)]$) of the shade avoidance potential (4), whose dynamics is defined as:

$$A(t+1) = A(t) + \rho \xi(t) e^{-bt} \left(h + J \sum_{n.n.} s_{ij}(t) \right), \quad (6)$$

with $A(0) = 0$.

$\xi(t)$ is the *same* random number selected in (5). The sum $\sum_{n.n.}$ runs over the nearest neighbours of the selected (i, j) -plant as above.

A mortality rate has been introduced in the following way: if at certain time the (i, j) -plant has reached an auxin amount smaller than a threshold d , s_{ij} is set to zero and no longer updated during the simulation. This corresponds to the death of the (i, j) -plant. For the sake of clarity, the mortality process has not been shown in the flowchart in Fig. 1.

Now, consider the function $q_{ij}(t)$, that shall represent the height of the (i, j) -plant at time t . As discussed before, the auxin amount of a plant is converted into biomass after a time lag α , and the biomass of a plant is assumed to be proportional to its height, $q_{ij}(t)$. Therefore, setting the proportionality constant equal to one, before the equilibrium is reached the plant height at time t corresponds to the auxin amount at time $t - \alpha$. Then, at the equilibrium, the system does not evolve appreciably and, therefore, the plant height at time t corresponds to the auxin amount at the same time. In formulae,

$$q_{ij}(t) = s_{ij}(t - \alpha) \quad \text{for } t < 1/b, \quad (7)$$

$$q_{ij}(t) = s_{ij}(t) \quad \text{for } t \gtrsim 1/b. \quad (8)$$

¹Actually, it is the opposite. In fact, here the global shade avoidance potential will be maximized, while in the Ising model the hamiltonian is minimized.

²In this work, for equilibrium I mean the condition when the increments Δs_{ij} become negligible. This happens at time $t \sim 1/b$.

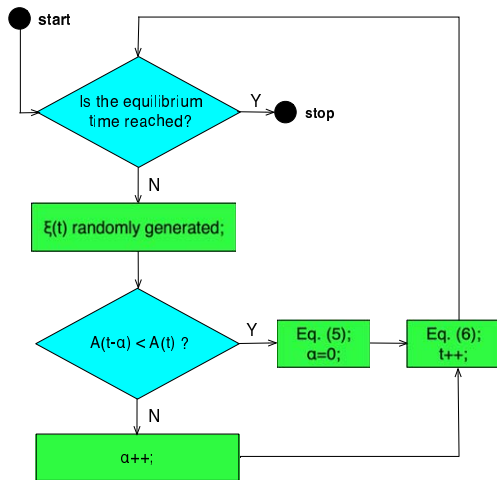


Figure 1: Flowchart of the system dynamics.

3. Numerical results

I performed numerical simulations of the dynamics (5-6) using the following parameter values: $N = 10$, $\rho = .2$, $b = 10^{-4}$, $J = 10^2$, $h = 1$. The mortality threshold has been set to $d = .3$, which corresponds to a rate of mortality of 70%, accordingly to experiments by Ford (1975).

Fig. 2 shows two height distributions at the equilibrium, i.e. at $t \gtrsim 1/b$, for initial communities of $N^2 = 100$ plants. Each histogram represents the height distribution of the $dN^2 = 30$ survived plants.

In Fig. 2a the simulation has been performed according to the model presented in the previous section: the system is non-markovian. The distribution results to be bimodal and right-skewed (see experiments by Ford (1975)). Moreover, plants at the lattice border are on average (see Appendix B) shorter than central ones.

On the contrary, in Fig. 2b, the time lag α has been set to be equal to zero, i.e. the system has been forced to be markovian. The height distribution loses bimodality, showing that in this model framework the non-markovianity is a fundamental feature to interpret data of plants competing for light.

The numerical simulations show that α varies with time. In particular, α can be consistently fitted by the simple saturating function

$$\alpha(t) = \frac{m t}{K + t}, \quad (9)$$

where m is the plateau value reached by α and $K \equiv 1/(2b)$.

4. Isolated plants

The model presented here is intended to mimic the behaviour of plants at high population density. Indeed, it may be extended to isolated plants.

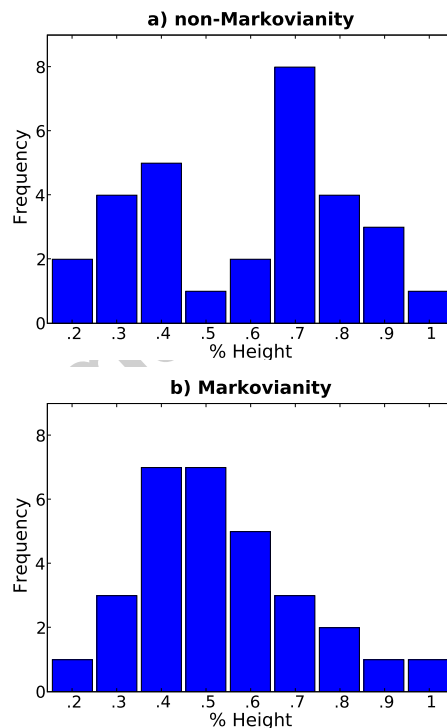


Figure 2: Height distribution at the equilibrium, $t \gtrsim 1/b$, for an initial community of $N^2 = 100$ plants. The rate of mortality have been set to 70%, accordingly to experiments by Ford (1975). The heights are reported in percentage relatively to the maximal height in each sample. a) Simulation of the system according to the model: the Markov property is relaxed. The distribution is bimodal and right-skewed, as in the experiments by Ford (1975). b) Simulation of the system setting the time lag $\alpha = 0$, i.e. forcing the system to be Markovian. The distribution loses bimodality.

In Appendix A, I derived the expression for the growth (height) dynamics $q(t)$ and the growth speed $\dot{q}(t)$ in isolated plants. They read

$$q(t) = \rho \Omega b^{-1} (1 - e^{-bf(t)}), \quad (10)$$

$$\dot{q}(t) = \rho \Omega \left(1 - \frac{mK}{(K+t)^2}\right) e^{-bf(t)}, \quad (11)$$

where Ω is defined as in (A3) and

$$f(t) = t - \alpha(t) = \frac{K - m + t}{K/t + 1} \quad (12)$$

This allows to directly check the model consistency with data available in literature.

Jouve et al. (1998) report experimental data for *Arabidopsis thaliana* floral stem elongation over time. They study circadian rhythms and report experimental data for Arabidopsis first inflorescence internode length and for first inflorescence extension rate as functions of time. The first inflorescence internode length can be assumed to be proportional to the plant growth dynamics $q(t)$, and the first inflorescence extension rate, up to a constant, to the growth speed $\dot{q}(t)$.

Fig. 3 shows $q(t)$ (10) fitted on Arabidopsis first inflorescence internode length data and Fig. 4 shows $\dot{q}(t)$ (11) fitted on Arabidopsis first inflorescence extension rate data.

The agreement are satisfactory in both cases; fluctuations in first inflorescence extension rate data are due to the circadian rhythm, which is not taken into account by this model and which can be easily averaged out.

Interestingly, Cournède et al. (2008) propose the following empirical formula for the biomass production $Q(t)$,

$$Q(t) = \beta \lambda(t) (1 - e^{-\gamma S(t)}), \quad (13)$$

where β and γ are characteristic parameters, $S(t)$ the leaf surface area of the plants, and $\lambda(t)$ a function of environmental conditions related to the evapotranspiration that I here assume to be constant.

Considering again the Arabidopsis case, experimental data from Fig.3a in Cookson et al. (2007) show that, in the time window of interest (0-8 days), Arabidopsis leaf surface area over time can be consistently fitted (reduced $\chi^2 \sim 1$) by $af(t)$, being a a proportionality constant.

This suggests that the growth dynamics $q(t)$ obtained here and the biomass production $Q(t)$ proposed by Cournède et al. (2008) are consistent. While parameter identification between the two models needs further investigation, it is worth observing that $f(t)$ can correctly reproduce experimental data for leaf surface area over time only in the case $m \neq 0$. In other terms, it is necessary the system to keep memory of the time lag α even in the case of isolated plants.

5. Sensitivity analysis

Focusing back to the general model ($J \geq 0$, $N \geq 1$), it is interesting to analyze its parameter sensitivity.

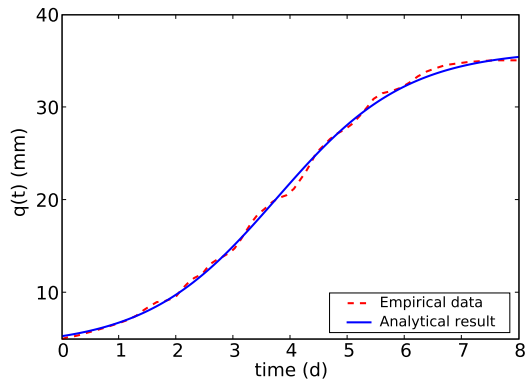


Figure 3: *Arabidopsis thaliana* growth dynamics. Eq. (11) (continuous line) and experimental data from Fig. 1, dataset A (12 hours of dark every 12 hours of light), in Jouve et al. (1998) (dashed line).

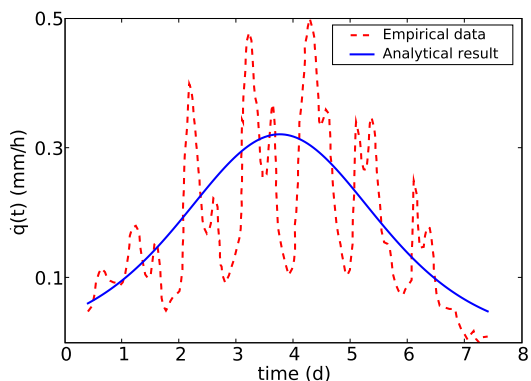


Figure 4: *Arabidopsis thaliana* growth speed. Eq. (10) (continuous line) and experimental data from Fig. 2A in Jouve et al. (1998) (dashed line).

I have performed a stochastic analysis of the model (Appendix B), whose main result is, according to (B13) and (B14),

$$\langle s_{ij} \rangle_{st} \simeq \text{function}(\rho, b, N). \quad (14)$$

This means that on average the auxin amount in a generic (i, j) -plant does not directly depend on J and h . Numerical simulations confirmed this result. Speculating, this suggests that plant height does not directly depend either on the R:FR ratio or on the hormone ethylene density. This observation is consistent with the fact that plant sensitivity both to light (Malooof et al., 2001) and to hormone ethylene (Chen and Bleecker, 1995) stimulations rapidly saturates.

6. Discussion

This paper proposes a simple model for the growth at high population density of plants competing for light. The model is based on the consideration that the plant growth is controlled by the auxin synthetic pathway, which is regulated by two signals, an internal one related to the hormone ethylene, and an environmental one, due to the mutual shading. Moreover, the auxin signal is converted into biomass only after a time lag, α , that varies on time. This memory effect forbids modelling the system markovianly at the natural time scale. In fact, a single plant has the predisposition to avoid the shade experienced at time t only by shooting at time $t + \alpha$. At the system level, this can be modelled by maximizing the global shade avoidance potential (4).

Consequently, this model cannot be considered to belong to any of the three frameworks of spatial community models drafted by Bolker et al. (2003), namely the Interacting Particle Systems, the Stochastic Point Processes, and the Patch Models. In fact, even sharing some features with IPSs, the model proposed here is non-markovian. Indeed, it can be considered closer to those models that describe in terms of energy-like function(al)s a broad range of life science phenomena, such as X chromosome inactivation (Nicodemi and Prisco, 2007), epithelial cell polarization (Veglio et al., 2009), and eukaryotic chemotaxis (Ferraro et al., 2008; Gamba et al., 2009).

The present work shows that the simulated plant height distributions are bimodal and right skewed, as in real communities of plants at high population density. On the contrary, if no delay is assumed between the auxin signal and the biomass production, i.e. if the time lag α is set to zero, simulations show that the distribution of plant heights is unimodal and cannot reproduce real distributions.

Extending the model to isolated plants, an analytical expression is obtained both for the growth dynamics (10) and the growth speed (11). They have been fitted to *Arabidopsis thaliana* first inflorescence length data and on first inflorescence internode extension rate data, respectively. Theoretical functions and experimental data show a good agreement.

Moreover, the obtained growth dynamics (10) is consistent with the biomass production function proposed by Cournède et al. (2008).

The bimodality in height distribution, the good match between experimental data and theoretical functions for isolated plants, and the consistency with an empirical model independently derived, can be considered the main results of this work. They hold as long as the time lag α is taken into account. If the model is forced to be markovian, $\alpha = 0$, real distributions and dynamics can no longer be mimicked. This suggests that memory effects play a key role in the plant growth process and that they cannot be neglected.

Interestingly, Hara (1984) studies the dynamics of plant populations with a stochastic model based on the Kolmogorov forward equation, which needs markovianity, and observes that the Markov property applied to plant growth is reasonable as a first approximation and needs further investigation. In fact, it seems not to be a case that his model does not reproduce bimodality in plant height distributions.

A further step in the comprehension of plant growth would be to experimentally check the effects of varying the time lag between the auxin production and its conversion in biomass (for example, along the lines of Tao et al. (2008)), both in high population density and isolated plants. Accordingly, from a theoretical point of view it would be interesting to study the bimodal-to-unimodal switch in height distribution due to the loss of markovianity, in particular in terms of statistical physics of phase transitions (Huang, 1987; Barabási and Stanley, 1995).

Appendices

A. Analysis of the Dynamics

To analyze the dynamics, it is worth switching from discrete to continuous time by substituting $t \rightarrow N^2 t$. This yields (5) to read

$$\dot{s}_{ij}(t) = \rho e^{-bN^2 t} \eta_{ij}(t), \quad (A1)$$

where

$$\eta_{ij}(t) \equiv \xi(t) \Theta[A(t) - A(t - \alpha)] \quad (A2)$$

is a random term. I will treat it as a gaussian non-zero-mean colored noise with time-independent mean, which I shall label as

$$\Omega_{ij} \equiv \langle \eta_{ij}(t) \rangle = \langle \xi(t) \Theta[A(t) - A(t - \alpha)] \rangle. \quad (A3)$$

Therefore, the average of (A1) over different system's realizations is simply

$$\langle \dot{s}_{ij}(t) \rangle = \rho \Omega_{ij} e^{-bN^2 t}, \quad (A4)$$

with the straightforward solution

$$\langle s_{ij}(t) \rangle = \frac{\rho \Omega_{ij}}{b N^2} (1 - e^{-bN^2 t}), \quad (A5)$$

which has the following steady state,

$$\langle s_{ij} \rangle_{st} \equiv \lim_{t \rightarrow \infty} \langle s_{ij}(t) \rangle = \frac{\rho \Omega_{ij}}{b N^2}. \quad (\text{A6})$$

The dynamics of an isolated plant height over time $q(t)$ may be defined as

$$q(t) \equiv \langle s(t) \rangle \Big|_{t=t-\alpha(t)}, \quad (\text{A7})$$

where $\langle \dot{s}(t) \rangle$ is the same as in (A4), but without position indexes because it is referred to an isolated plant. It is calculated at time $t - \alpha(t)$ to reflect that auxin is not immediately converted into biomass, as discussed in Section 2. (A7) is the same as (7) except for the average, which is intended to reduce fluctuations due to the isolated plant condition. The shade avoidance potential is ideally evaluated in the condition $J = 0$, i.e. no interaction, and $N = 1$.

Using (9), (A5) and (A7), $q(t)$ reads

$$\begin{aligned} q(t) &= \langle s(t) \rangle \Big|_{t=t-\alpha(t)} \\ &= \rho \Omega b^{-1} (1 - e^{-b(t-mt)/(K+t)}), \end{aligned} \quad (\text{A8})$$

and

$$\dot{q}(t) = \rho \Omega \left(1 - \frac{m K}{K+t}\right) e^{-b(t-mt)/(K+t)}. \quad (\text{A9})$$

The fact that α tends to the finite value m implies that at the equilibrium the delay gets negligible, letting $q(t) = \langle s(t) \rangle$ (A5) and $\dot{q}(t) = \langle \dot{s}(t) \rangle$ (A4). This is consistent with (8), that holds after the equilibrium.

B. Analysis of the Parameters

In order to explain the model dependence on the parameters, it is worth evaluating analytically the noise mean Ω_{ij} introduced in (A3). Its definition is general and valid for the whole system of plants.

(A1) is a Langevin-like equation with a non-zero-mean colored noise, no drift term and time dependent diffusion term. It is possible to perform some transformations to obtain a more standard Langevin-like equation. Defining

$$x_{ij}(t) \equiv s_{ij}(t) e^{bN^2 t}, \quad (\text{B1})$$

and inserting (A1) in the time derivative of (B1), I obtain

$$\dot{x}_{ij}(t) = F_{ij}(x) + \rho \tilde{\eta}_{ij}(t), \quad (\text{B2})$$

where

$$F_{ij}(x) \equiv bN^2 x_{ij}(t) + \rho \Omega_{ij}, \quad (\text{B3})$$

$$\tilde{\eta}_{ij}(t) \equiv \eta_{ij}(t) - \Omega_{ij}. \quad (\text{B4})$$

Consider that $x(t)$ and $s(t)$ have, at the steady state, the same pdf.

(B2) is a Langevin-like equation with time independent drift term, constant diffusion term and zero-mean colored noise.

Assuming that such a noise is a Ornstein-Uhlenbeck process (van Kampen, 2007), i.e.

$$\frac{d\tilde{\eta}_{ij}(t)}{dt} = -\frac{1}{\mu_{ij}} \tilde{\eta}_{ij}(t) + \zeta(t), \quad (\text{B5})$$

where μ_{ij} is the correlation time and $\zeta(t)$ is a white noise with zero mean and delta time-correlation, it is possible to write the Fokker-Planck equation corresponding to (B2), i.e.

$$\begin{aligned} \frac{\partial p_{ij}(x, \eta, t)}{\partial t} &= -bN^2 + \gamma p_{ij}(x, \eta, t) + \\ &+ \gamma \eta \frac{\partial p_{ij}(x, \eta, t)}{\partial \eta} + \frac{1}{2} \frac{\partial^2 p_{ij}(x, \eta, t)}{\partial \eta^2}. \end{aligned} \quad (\text{B6})$$

Even at the steady state, (B6) is not easy to be analytically solved. However, for a huge set of parameters (B2) lies in the validity range of the Unified Colored Noise Approximation, UCNA (Jung and Hänggi, 1987; Hänggi and Jung, 1995; Hasegawa, 2008). In fact, to apply the UCNA it is necessary (Hänggi and Jung, 1995) that

$$x_{ij} \gg \frac{\mu_{ij}}{b}. \quad (\text{B7})$$

According to (B1) and to the fact that $s_{ij} \sim 1$, constraint (B7) corresponds to

$$t \gg \frac{\log(\mu_{ij}/b)}{bN^2} \equiv t_u. \quad (\text{B8})$$

Therefore, since equilibrium time $1/b \gg t_u$, the UCNA can be applied for the parameter set used in the reported numerical simulations. Applying UCNA, the following effective Langevin equation holds

$$\dot{x}_{ij}(t) \simeq \sigma_{ij} F_{ij}(x) + \rho \sigma_{ij}^2 \zeta(t), \quad (\text{B9})$$

where $\zeta(t)$ is again a white noise with zero mean and delta time-correlation, while I defined σ_{ij} as

$$\sigma_{ij} \equiv \frac{1}{1 - \mu_{ij} b N^2}. \quad (\text{B10})$$

I calculated the correlation time μ_{ij} by averaging the numerical values of α over all the lattice nodes and over different realizations of the system. As an example, labelling the site at the center of the lattice as cc , the sites at the center of the lattice borders as bb and the sites at the corners as aa , I obtained, in continuous time units, $\mu_{cc} \simeq 54$, $\mu_{bb} \simeq 50$ and $\mu_{aa} \simeq 47$.

In passing, notice that the drift term of (B9), $\sigma_{ij} F_{ij}(x)$, is linear in x_{ij} ; therefore this model lies in a class of universality different from the principal growth models³ (Barabási and Stanley, 1995). Then, it is possible to write the Fokker-Planck equation corresponding to (B9),

$$\begin{aligned} \frac{\partial p_{ij}(s, t)}{\partial t} &= -\sigma_{ij} \frac{\partial (F_{ij}(s) p_{ij}(s, t))}{\partial s} + \\ &+ \frac{1}{2} \rho^2 \sigma_{ij}^4 \frac{\partial^2 p_{ij}(s, t)}{\partial s^2}, \end{aligned} \quad (\text{B11})$$

³In particular, in the Kardar-Parisi-Zhang model (Kardar et al., 1986) the drift term is equal to $\nu \nabla^2 x_{ij} + \frac{\mu}{2} (\nabla x_{ij})^2$, where ν and μ are characteristic parameters of that model.

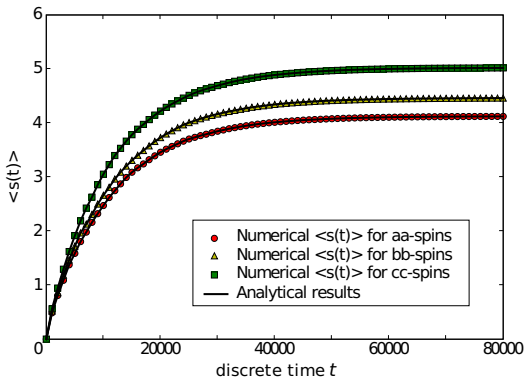


Figure 5: Eq. (A5) (lines) and numerical results from the simulation of the dynamics (5-6) (symbols). Arbitrary units.

and consequently to calculate the probability distribution function at the steady state $p_{ij}(s) \equiv \lim_{t \rightarrow \infty} p_{ij}(s, t)$ (van Kampen, 2007)

$$p_{ij}(s) = \frac{\exp[-\rho^{-2}\sigma_{ij}^{-3}(bN^2s^2 + 2\rho\Omega_{ij}s)]}{\int_0^1 dy \exp[-\rho^{-2}\sigma_{ij}^{-3}(bN^2y^2 + 2\rho\Omega_{ij}y)]}. \quad (\text{B12})$$

Therefore,

$$\langle s_{ij} \rangle_{st} \simeq \int_0^\infty ds s p_{ij}(s) \equiv g(\Omega_{ij}, \dots), \quad (\text{B13})$$

where the last term is to express that the analytical solution of the integral in (B13), that I do not report for reasons of shortness, is a function of the noise mean Ω_{ij} . Comparing (B13) to (A6) the following implicit equation in Ω_{ij} holds

$$\rho\Omega_{ij} \simeq bN^2g(\Omega_{ij}, \dots), \quad (\text{B14})$$

which has solutions, for plants in different position of the lattice labelled as above, $\Omega_{cc} \simeq .23$, $\Omega_{bb} \simeq .20$ and $\Omega_{aa} \simeq .18$. In Fig. 5 analytical solution (A5) and numerical results are plotted.

Acknowledgements

The author wishes to thank Federico Bussolino, Michele Caselle, Antonio Celani, and Marta Lucchini for useful discussions and comments, and Guido Serini for carefully reading the manuscript.

References

Barabási, A.-L., Stanley, H., 1995. Fractal Concepts in Surface Growth. Cambridge University Press.

Bolker, B. M., Pacala, S. W., 1999. Spatial moment equations for plant competition: Understanding spatial strategies and the advantages of short dispersal. *The American Naturalist* 153 (6), 575–602.

Bolker, B. M., Pacala, S. W., Neuhauser, C., 2003. Spatial dynamics in model plant communities: what do we really know? *Am Nat* 162 (2), 135–148.

Chen, Q. G., Bleecker, A. B., 1995. Analysis of ethylene signal-transduction kinetics associated with seedling-growth response and chitinase induction in wild-type and mutant arabidopsis. *Plant Physiol* 108 (2), 597–607.

Clements, F. E., 1905. Research methods in ecology. University Pub. Co.

Clements, F. E., Weaver, J. E., Hanson, H. C., 1929. Plant competition: an analysis of community functions. Carnegie Institution of Washington Publication.

Cookson, S. J., Chenu, K., Granier, C., 2007. Day length affects the dynamics of leaf expansion and cellular development in arabidopsis thaliana partially through floral transition timing. *Ann Bot* 99 (4), 703–711.

Cournède, P.-H., Mathieu, A., Houllier, F., Barthélémy, D., de Reffye, P., 2008. Computing competition for light in the green-lab model of plant growth: a contribution to the study of the effects of density on resource acquisition and architectural development. *Ann Bot* 101 (8), 1207–1219.

de Reuille, P. B., Bohn-Courseau, I., Ljung, K., Morin, H., Carraro, N., Godin, C., Traas, J., 2006. Computer simulations reveal properties of the cell-cell signaling network at the shoot apex in arabidopsis. *Proc Natl Acad Sci U S A* 103 (5), 1627–1632.

Dieckmann, U., Law, R., 2000. The Geometry of Ecological Interactions: Simplifying Spatial Complexity. Cambridge University Press., Ch. Relaxation Projections and the Method of Moments., pp. 412–455.

Diggle, P. J., 1976. A spatial stochastic model of inter-plant competition. *Journal of Applied Probability* 13 (4), 662–671.

Donald, C. M., 1951. Competition among pasture plants. i. intraspecific competition among annual pasture plants. *Australian Journal of Agricultural Research* 2 (4), 355–376.

Durrett, R., Levin, S. A., 1994. Stochastic spatial models: A user's guide to ecological applications. *Philosophical Transactions: Biological Sciences* 343 (1305), 329–350.

Ferraro, T., de Candia, A., Gamba, A., Coniglio, A., 2008. Spatial signal amplification in cell biology: a lattice-gas model for self-tuned phase ordering. *Europh. Lett.* 83, 50009.

Ford, E. D., 1975. Competition and stand structure in some even-aged plant monocultures. *Journal of Ecology* 63 (1), 311–333.

Ford, E. D., Diggle, P. J., 1981. Competition for light in a plant monoculture modelled as a spatial stochastic process. *Ann Bot* 48 (4), 481–500.

Friml, J., Sauer, M., 2008. Plant biology: in their neighbour's shadow. *Nature* 453 (7193), 298–299.

Gamba, A., Kolokolov, I., Lebedev, V., Ortenzi, G., 2009. Universal features of cell polarization processes. *Journal of Statistical Mechanics: Theory and Experiment* 2009 (02), P02019.

Gandhi, A., Levin, S., Orszag, S., 1998. "critical slowing down" in time-to-extinction: an example of critical phenomena in ecology. *J Theor Biol* 192 (3), 363–376.

Gilad, E., von Hardenberg, J., Provenzale, A., Shachak, M., Meron, E., 2004. Ecosystem engineers: from pattern formation to habitat creation. *Phys Rev Lett* 93 (9), 098105.

Gilad, E., von Hardenberg, J., Provenzale, A., Shachak, M., Meron, E., 2007. A mathematical model of plants as ecosystem engineers. *J Theor Biol* 244 (4), 680–691.

Guédon, Y., Caraglio, Y., Heuret, P., Lebarbier, E., Meredieu, C., 2007. Analyzing growth components in trees. *J Theor Biol* 248 (3), 418–447.

Hänggi, P., Jung, P., 1995. Advances in Chemical Physics. Vol. 89. John Wiley & Sons, Ch. Colored Noise in Dynamical Systems, pp. 239–326.

Hara, T., 1984. A stochastic model and the moment dynamics of the growth and size distribution in plant populations. *J Theor Biol* 109 (2), 173 – 190.

Harper, J. L., 1967. A darwinian approach to plant ecology. *Journal of Ecology* 55 (2), 247–270.

Hasegawa, H., 2008. Dynamics of the langevin model subjected to

- colored noise: Functional-integral method. *Physica A: Statistical Mechanics and its Applications*, 387 (12), 2697–2718.
- Hendry, R. J., McGlade, J. M., Weiner, J., 1996. A coupled map lattice model of the growth of plant monocultures. *Ecological Modelling* 84 (1-3), 81 – 90.
- Huang, K., 1987. *Statistical Mechanics*, 2nd Edition. John Wiley & Sons.
- Jönsson, H., Heisler, M. G., Shapiro, B. E., Meyerowitz, E. M., Mjolsness, E., 2006. An auxin-driven polarized transport model for phyllotaxis. *Proc Natl Acad Sci U S A* 103 (5), 1633–1638.
- Jouve, L., Greppin, H., Agosti, R., 1998. Arabidopsis thaliana floral stem elongation: Evidence for an endogenous circadian rhythm. *Plant Physiology And Biochemistry* 36 (6), 469–472.
- Jung, P., Hänggi, P., 1987. Dynamical systems: A unified colored-noise approximation. *Phys. Rev. A* 35 (10), 4464–4466.
- Kardar, M., Parisi, G., Zhang, Y.-C., 1986. Dynamic scaling of growing interfaces. *Phys. Rev. Lett.* 56 (9), 889–892.
- Kletter, A. Y., von Hardenberg, J., Meron, E., Provenzale, A., 2009. Patterned vegetation and rainfall intermittency. *J Theor Biol* 256 (4), 574–583.
- Levins, R., Culver, D., 1971. Regional coexistence of species and competition between rare species. *Proc Natl Acad Sci U S A* 68 (6), 1246–1248.
- Maloof, J. N., Borevitz, J. O., Dabi, T., Lutes, J., Nehring, R. B., Redfern, J. L., Trainer, G. T., Wilson, J. M., Asami, T., Berry, C. C., Weigel, D., Chory, J., 2001. Natural variation in light sensitivity of arabidopsis. *Nat Genet* 29 (4), 441–446.
- Neuhauser, C., 2001. Mathematical challenges in spatial ecology. *Notices of the American Mathematical Society* 48 (11), 1304.
- Newell, A. C., Shipman, P. D., Sun, Z., 2008. Phyllotaxis: cooperation and competition between mechanical and biochemical processes. *J Theor Biol* 251 (3), 421–439.
- Nicodemi, M., Prisco, A., Nov 2007. Self-assembly and dna binding of the blocking factor in x chromosome inactivation. *PLoS Comput Biol* 3 (11), e210.
- Pacala, S. W., Canham, C. D., Saponara, J., Silander Jr., J. A., Kobe, R. K., Ribbens, E., 1996. Forest models defined by field measurements: Estimation, error analysis and dynamics. *Ecological Monographs* 66 (1), 1–43.
- Smith, R. S., Guyomarc'h, S., Mandel, T., Reinhardt, D., Kuhlemeier, C., Prusinkiewicz, P., 2006. A plausible model of phyllotaxis. *Proc Natl Acad Sci U S A* 103 (5), 1301–1306.
- Stepanova, A. N., Robertson-Hoyt, J., Yun, J., Benavente, L. M., Xie, D.-Y., Dolezal, K., Schlereth, A., Jürgens, G., Alonso, J. M., 2008. Taa1-mediated auxin biosynthesis is essential for hormone crosstalk and plant development. *Cell* 133 (1), 177–191.
- Taiz, L., Zeiger, E., 2002. *Plant Physiology*, 3rd Edition. Sinauer Associates, Ch. 19, Auxin: The Growth Hormone, pp. 423–460.
- Tanaka, H., Dhonukshe, P., Brewer, P. B., Friml, J., 2006. Spatiotemporal asymmetric auxin distribution: a means to coordinate plant development. *Cell Mol Life Sci* 63 (23), 2738–2754.
- Tao, Y., Ferrer, J.-L., Ljung, K., Pojer, F., Hong, F., Long, J. A., Li, L., Moreno, J. E., Bowman, M. E., Ivans, L. J., Cheng, Y., Lim, J., Zhao, Y., Ballaré, C. L., Sandberg, G., Noel, J. P., Chory, J., 2008. Rapid synthesis of auxin via a new tryptophan-dependent pathway is required for shade avoidance in plants. *Cell* 133 (1), 164–176.
- Teale, W. D., Paponov, I. A., Palme, K., 2006. Auxin in action: signalling, transport and the control of plant growth and development. *Nat Rev Mol Cell Biol* 7 (11), 847–859.
- van Kampen, N., 2007. *Stochastic Processes in Physics and Chemistry*, 3rd Edition. North-Holland.
- Veglio, A., Gamba, A., Nicodemi, M., Bussolino, F., Serini, G., 2009. Symmetry breaking mechanism for epithelial cell polarization. *Phys. Rev. E* 80 (3), 031919.