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Light-dependent kinetic model for microalgae experiencing photoacclimation, photodamage, and photodamage repair

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ABSTRACT

Microalgae naturally are exposed to changing light conditions. While a higher light intensity can promote a faster growth rate, it also can cause photodamage that leads to a temporary or semi-permanent decline in growth rate. We developed a model of photosynthetic growth including photoacclimation, reversible photodamage to photosystem II (PSII), and more severe photodamage to photosystem I (PSI). Phototrophic biomass optimizes its photosynthetic machinery to the light intensity it is experiencing; this is captured in the model by photoacclimation, in which photodamage to PSII caused by absorbed light is balanced by repair. However, repair of PSII photodamage can be overwhelmed by increases of light outside the photoacclimated condition, and this leads to severe PSII photodamage that slows the cells' specific growth rate. Furthermore, very large increases in light intensity can lead to photodamage to PSI, which is semi-permanent in that it can take days to weeks to repair. Our model captures all these phenomena. Example model outputs demonstrate the importance of each phenomenon for increases and decreases in light intensity from the photoacclimated state.

1. Introduction

In natural conditions, cyanobacteria and single-celled algae (collectively referred to as microalgae) are exposed to constantly changing light conditions due to diurnal and seasonal light patterns, variations in incident light intensity (LI) over time, and mixing in the water column. Because microalgae are photosynthetic, exposing them to greater LI should lead to higher growth rates; however, changing LI also can lead to more complex phenomena, namely photoacclimation, photodamage, and photodamage repair [1–3]. Capturing these phenomena in a mathematical model can improve predictions of photosynthetic activity and give further insight to bioreactor design for microalgae cultivation [2].

Photoacclimation is a set of changes in macromolecular composition (cell morphology, pigment concentration, and enzymes associated with photosynthesis and respiration) in response to differing light conditions [3]. Photoacclimation allows microalgae to optimize photosynthetic activity for a given LI. This includes increasing the capacity for non-photochemical quenching (i.e., harmless quenching of excitation energy) under intense light, or increasing light absorption under dim light [4,5]. Sudden increases in LI from a photoacclimated state, however, leave biomass susceptible to photoinhibition that alters the capacity of microalgae to harvest light and leads to a decrease in the rate of photosynthesis [6]. As we document in a companion paper with extensive experimental results ([7]; select examples are present in supplementary material) and has been seen previously [8,9], a large and sudden step from low LI to high LI gives an initial spike in the rate of photosynthetic growth, but soon the rate declines to a value below the eventual steady-state growth rate of the new LI. The initial spike in growth is due to rapid accumulation of carbohydrates, and the slow down after the spike arises from near complete reduction of the plastoquinone pool, which leads to photodamage (sometimes called photoinactivation) [8].

The literature describes two types of photodamage – to photosystem I (PSI) and to photosystem II (PSII) – with damage to PSII occurring far more frequently [10]. It is believed that the primary mechanism of PSII photodamage occurs when antenna complexes enter triplet states during light absorption and create reactive oxygen species (ROS) that damage the photosynthetic machinery [11,12]. The main target of these ROS is the D1 protein, the primary electron-accepting protein from the oxygen evolving complex [13]. The D1 protein has damage and repair mechanisms that are active under all illuminated conditions, but photoinhibition occurs when the rate of damage exceeds the rate of repair, such as after a sudden change of light intensity [14,15]. While PSII photodamage is thought to be proportional to light intensity, the loss of this balance is primarily caused by an inactivation of the repair

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function [10,16]. Repair of PSII photodamage is a complex process involving disassembly of the damaged component, reassembly of a working unit, and its insertion into a PSII complex [17–19].

PSI is more protected than PSII, but damage still occurs when the flow of electrons from PSII exceeds the capacity of the electron acceptors in PSI [11,20,21]. Because the source of photodamage is electrons from PSII, photodamage to PSII effectively protects PSI under normal fluctuations in LI; however, intense LI can lead to photodamage to PSI [20,21]. Repair to PSI is very slow, on the order of days to weeks, and inhibited PSI can lead to substantial photodamage to PSII because of a lack of electron acceptors from PSI [21,22]. In our companion paper, we demonstrated a semi-permanent (i.e., for tests lasting 4 days) decrease in the specific growth rate of *Synechocystis* sp. PCC 6803 (*Synechocystis* from here) due to extreme high light [7]. To our knowledge, this phenomenon has not been explained for microalgae; however, long-term PS1 photodamage has been documented for plants [23], and we believe it is a likely cause of the observed decrease in specific growth rate with extreme high light.

Using *Synechocystis*, [5] suggested that light extinction due to its absorption (ε_{abs}) was a suitable metric of photoacclimation. Here, we expand upon the ε_{abs} concept by introducing three new state variables: ε_{nf}, representing PSII photodamage; ζ, representing PSII repair inhibition; and δ, representing PSI photodamage. We develop and illustrate a kinetic model accounting for photoacclimation, PSII photodamage, PSII photodamage repair, and PSI photodamage. PSI photodamage is considered semi-permanent and, therefore, we do not address its repair. While a number of light-dependent models of photosynthesis can be found in the literature (reviewed by [24]), few account for photoacclimation, photodamage, and photodamage repair, and ours is the first to address a semi-permanent reduction in growth addressed as PSI photodamage. Thus, our model is the first comprehensive representation of how photoacclimation, photodamage, and photodamage repair control the growth rate of microalgae experiencing changes in light intensity.

In a companion paper, we evaluate our model experimentally using *Synechocystis* and find that our model describes well the effects of sudden light-intensity changes on the specific growth rate [7]. Here, we present a set of modeling experiments that demonstrate the features of the model and why modeling without photoacclimation and photodamage can seriously overestimate the rate of photosynthetic growth during changes in light. These modeling results lay the foundation for understanding the experimental results of our companion paper [7].

2. Modeling growth with photoinhibition phenomena

The steps in photosynthetic growth are the absorption of light, electron transport, CO_2 fixation, and cell division. Light absorption takes on the order of 10^{-15} to 10^{-9} s, electron transport is on the order of 10^{-9} to 10^{-4} s, CO_2 fixation is on the order of 10^{-4} to 1 s, and cell division takes 1 to 10^3 s [25]. With rapid mixing or flashing light, these energy intermediates become important to capturing trends in growth. In this model, we, therefore, include a pool of absorbed light energy (LI_p; μmol (g biomass)$^{-1}$ (simply μmol g^{-1} from here)) to capture the pooling of light energy under rapidly changing light intensities. The timescale of LI_p is related to the interface between CO_2 fixation and cell division such that LI_p is absorbed light energy that leads to the synthesis of additional biomass. We describe the accumulation of LI_p with the following relationship:

$$\frac{dLI_p}{dt} = \left(\varepsilon_{abs} LI - \varepsilon_{nf} LI - \left(\frac{k_{LI} LI_p}{k_{LI} + LI_p} + \frac{LI_p^2}{k_{LI} + LI_p}\right) k_{LIp}\right) \quad (1)$$

where ε_{abs} (m^2 g^{-1}) is the specific light absorption, LI (μmol m^{-2} s^{-1}) is the light intensity, k_{LI} (μmol g^{-1}) is the half-maximum-rate light absorption, ε_{nf} (m^2 g^{-1}) is *PSII* photodamage, and k_{LIp} (s^{-1}) is the rate constant of light-pool dissipation. From left to right, Eq. (1) includes terms for light absorption, non-photochemical quenching (NPQ) from damaged biomass, photochemical light quenching (i.e., for photosynthetic growth), and NPQ from other pigments. The third and fourth terms (for photochemical quenching and NPQ) are separate to articulate their separate derivations, but they mathematically collapse to -$LI_p k_{LIp}$, which shows that all absorbed light is either photochemically or non-photochemically quenched. In Eq. (1), dt is in units of seconds, whereas dt in the other rate equations is in days. Therefore, a conversion factor of 86,400 s d^{-1} should be applied for unit consistency when using Eq. (1). Eq. (1) has its greatest importance in situations of rapidly changing LI, such as flashing light or rapid mixing. Within 10 s of a change in light intensity, LI_p reaches a steady-state of ($\varepsilon_{abs} - \varepsilon_{nf}$) LI/$k_{LIp}$. Therefore, when changes to LI are more gradual (e.g., light changes > 1 min apart or < 10 μmol m^{-2} s^{-1}), LI_p can be simplified to $LI_p = (\varepsilon_{abs} - \varepsilon_{nf})$ LI/k_{LIp} with minimal effect on overall growth rate. As we demonstrate later, photoacclimation and components of photodamage occur on the order of hours to days.

To capture all the phenomena associated with photoinhibition, our model uses four biomass state-variables: photoacclimation (represented by ε_{abs}), PSII photodamage (ε_{nf}), the reduction in PSII repair or repair inhibition (ζ; m^2 g^{-1}), and PSI photodamage (δ; m^2 g^{-1}). Fig. 1 is a schematic of the interactions of these state variables and how they contribute to phototrophic growth. All four state variables depend on LI, and they sequentially affect each other. Ultimately, the interdependent effects are captured by ε_{nf}, and the specific growth rate (μ, d^{-1}) is given by:

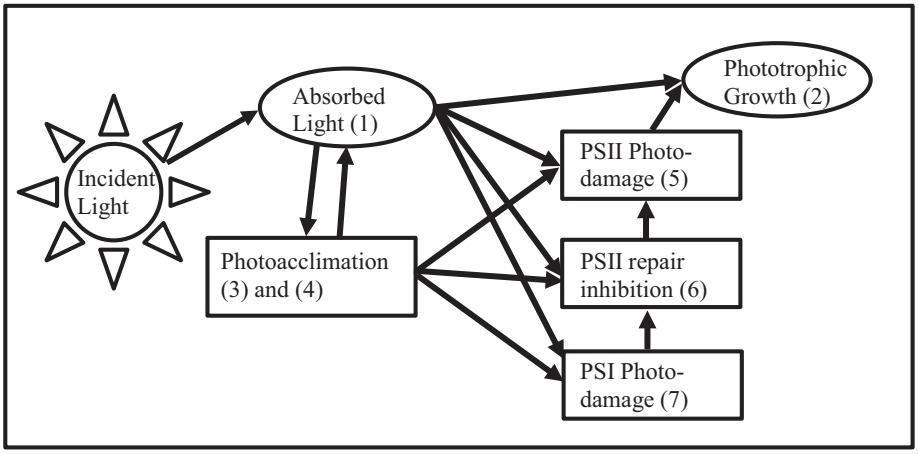

Fig. 1. Schematic depicting the structure of the model. Oval shapes indicate key processes, and rectangles indicate the state-variables of the biomass with associated equations in parenthesis. The arrows lead from a state variable or process to another state-variable or process that is affected by the originating state variable or process.