Chapter 21

Regulation of Photosynthetic Gene Expression by the Environment: From Seedling De-etiolation to Leaf Senescence

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Summary

Both endogenous and exogenous factors are involved in modulating and coordinating gene expression during plant development. Among them are light and plant hormones such as ethylene, cytokinins, abscisic acid, gibberellins, and brassinosteroids. Light and brassinosteroids have received particular attention because of their obvious and pronounced effects on early plant development following seed germination. By contrast, much less is known about the terminal stage of plant development referred to as senescence and the factors controlling this cell death program. Plant hormones such as ethylene and jasmonic acid have, however, been implicated in the initiation and progression of leaf senescence.

At all stages of their life cycle, plants are prone to various forms of oxidative stress. Upon illumination, excited tetrapyrroles such as chlorophyll, heme, and their precursors as well as degradation products can transfer their excitation energy onto oxygen, leading to the formation of highly reactive singlet oxygen. Angiosperms being the most highly evolved group of plants must have evolved efficient strategies to prevent the accumulation of such potentially harmful compounds. It is the aim of this chapter to summarize current concepts on the regulation of plant gene expression by light and the plant hormone jasmonic acid, with particular emphasis on the mechanisms by which higher plants prevent photooxidative self-poisoning. Special reference is made to the plastid compartment, which is the major site of tetrapyrrole metabolism and a source of signals that coordinate nuclear gene expression in response to light.

I. Introduction

As an environmental cue, light influences plant growth and development. Direction, periodicity, quality, and/or quantity of light regulate physiological responses such as phototropism, circadian rhythms, morphogenesis, and leaf senescence (Kendrick and Kronenberg, 1994). To account for some or all of these different effects, light perception is mediated through the action of various photoreceptors, including phototropins, cryptochromes, and the phytochromes (Cashmore, 1999; Casal, 2000; Christie and Briggs, 2001; Nagy and Schaefer, 2002; Quail, 2002a). Expression of several thousand genes is regulated by light and multiple, highly coordinated signal transduction pathways exist which, in many cases, involve sets of different photoreceptors. In addition, plant hormones such as brassinosteroids regulate gene expression during

Abbreviations: ACD – accelerated cell death; 5-ALA – 5-aminolevulinic acid; bHLH – basic helix-loop-helix transcription factor; BIN – brassinosteroid-insensitive; BL – brassinolide; BR – brassinosteroid; BRI – brassinosteroid-insensitive; Chlase – chlorophyllase; Chl(ide) – chlorophyll(ide); COI – coronatine-insensitive; COP – constitutive photomorphogenic; CRY – cryptochrome; DET – de-etiolated; ELIP – early light-inducible protein; FAD – fatty acid desaturase; FLU – fluorescent; GUN – genome-uncoupled; HIR – high-irradiance response; HR – hypersensitive response; JA(-Me) – jasmonic acid (methyl ester); LAF – long after far red light; LHCII – light-harvesting chlorophyll *a*/*b* binding protein of photosystem II; LHPP – light-harvesting POR (NADPH:protochlorophyllide oxidoreductase) protochlorophyllide complex; LLS – lethal leaf spot; LSD – lesion-simulating disease; NCC – non-fluorescent chlorophyll catabolite; Pao – phaeophorbide *a* oxygenase; PCD – programmed cell death; PΦB – phytochromobilin; Pheide – phaeophorbide; PHY or phy – phytochrome; PIF3 – phytochrome-interacting factor 3; PKS1 – phytochrome kinase substrate 1; PLB – prolamellar body; POR – NADPH:protochlorophyllide oxidoreductase; PQ – plastoquinone; Pr/Pfr – red light/far red light-absorbing form of phytochrome; Proto (gen)IX – protoporphyrin(ogen) IX; PS – photosystem; RCC – red chlorophyll catabolite; RCD – radical-induced cell death; ROS – reactive oxygen species; SA – salicylic acid; SAG(s) – senescence-associated gene(s); SARK – senescence-associated receptor-like kinase; SIRK – senescence-induced receptor-like kinase; THI – thionin.