Plant Science 177 (2009) 523–531

Contents lists available at ScienceDirect

Plant Science

journal homepage: www.elsevier.com/locate/plantsci

Review

Common mechanisms regulate flowering and dormancy

David Horvath

United States Department of Agriculture - Agricultural Research Service, Bioscience Research Lab, 1605 Albrecht Blvd, Fargo, ND 58105, United States

ARTICLE INFO

Article history:
Received 18 June 2009
Received in revised form 4 September 2009
Accepted 7 September 2009
Available online 12 September 2009

Keywords:
Endodormancy
Flowering
MADS-box
Circadian regulation
Microarrays

ABSTRACT

In 1960, Chouard hypothesized that there might be a connection between vernalization (facilitation of floral competence) and release from endodormancy. In 2003 we reiterated this hypothesis and suggested potential mechanisms involving chromatin remodeling. Since then, there have been several papers from various laboratories working on several different perennial species that suggest common signaling components regulate flowering and the growth cessation that must precede endodormancy induction. This work has directly implicated *FT*, *CENL1*, and MADS-box transcription factor genes similar to *SVP* and/or *AGL24* in growth cessation and endodormancy development. Numerous studies in other model systems have demonstrated the function and regulation of similar genes in floral regulation. Combined, these studies allow the development of a paradigm for future investigations designed to understand the nature and function of the regulatory mechanisms that control induction and release of endodormancy in perennial plants. Dicots, monocots, and gymnosperms have similar floral regulatory genes, suggesting that general regulation of flowering is highly conserved among perennial species. Likewise, reports of differential expression of particular MADS-box genes and putative *FT* orthologues during endodormancy transitions across multiple plant species suggest a conserved role for these genes in responses to endodormancy induction and maintenance. The limited but tantalizing linkage between floral regulatory machinery and seasonal growth cessation and bud set through regulation of *FT* and other *FT*-like genes suggest a general model for endodormancy regulation.

Published by Elsevier Ireland Ltd.

Contents

1. Introduction

In most temperate perennial plants, light and temperature regulate flowering and dormancy. Based on this simple observation, several groups have hypothesized that similar mechanisms regulate both processes [1–3]. Likewise, several excellent reviews have directly or indirectly discussed possible mechanisms that relate to this hypothesis [4–6]. This review will discuss recent findings on the regulation of bud dormancy and flowering that are beginning to provide mechanistic support of this hypothesis.

1.1. Brief overview of floral regulation

Due to its importance in plant reproduction, extensive research has been conducted which has identified many of the environ-

E-mail address: david.horvath@ars.usda.gov.

0168-9452/$ – see front matter. Published by Elsevier Ireland Ltd.
doi:10.1016/j.plantsci.2009.09.002

mental controls and genes involved in regulating flowering. Indeed there are numerous in-depth reviews on this process in both annual dicot and monocot plants [7–11], as well as in perennial trees [12,13]. Flowering occurs when meristems receive developmental and or environmental signals that cause the meristem to develop into flowers. These meristems may originally be predestined to flower upon growth, or they may initially be actively growing vegetative meristems that transition to floral meristems. In either case, induction of two key genes appear to initiate a cascade of events that alters the development of organ primordia within the meristem so that sepals, petals, pistils, and stamen are produced rather than leaves and maintenance of an undifferentiated core of cells at the center of the meristem. In the well-studied systems of rice (*Oryza sativa*), poplar (*Populus* ssp.), *Citrus* ssp., and arabidopsis (*Arabidopsis thaliana*), very similar genes and signaling networks appear to regulate flowering although arguably most of the research has been done on the winter annual arabidopsis, and thus unless noted otherwise, generalizations will refer to floral regulation in this plant.

There are many genes and signals that regulate flowering, most of which converge on *FLOWERING LOCUS T* (*FT*) (Fig. 1). FT has been touted as an essential component of the graft transmissible florigen whose existence was long hypothesized [8]. *FT* is mostly expressed in mature leaf tissue in response to floral promoting environmental conditions; however there is evidence for its expression in young leaves in the shoot apices, and in dormant bud tissue [14,15]. Leaf expressed FT is known to be phloem-transmissible and is transported to the meristem where it initiates floral morphogenesis.

The genes that initiate the developmental cascade towards flowering are *APETELA1* (*AP1*) and *LEAFY* (*LFY*) [16,17]. *AP1* is directly induced by *FT* [18], and *LFY* is directly induced by *SUPPRESOR OF OVEREXPRESSION OF CONSTANS 1* (*SOC1*) [19]. In turn, both *FT* and *SOC1* are positively regulated by CONSTANS (CO) [20], and both are negatively regulated by the MADS-box transcription factor FLOWERING LOCUS C (FLC) [21–23]. *CO* is in turn regulated by light through the various genes encoding components that make up the circadian clock and by PHYTOCHROME A (PHYA) [24].

Environmental and developmental signals that include aspects of chromatin remodeling and response to extended cold temperatures needed for vernalization (the process through which seeds and sometimes buds "remember" winter conditions and become competent to flower in the following spring) also regulate *FT* [25,26]. *FT* expression is also suppressed by another MADs-box transcription factor called SHORT VEGETATIVE PHASE (SVP) [27]. Like FLC, SVP binds to various regulatory sequences within the *FT* gene and inhibits its expression. However, *SVP* is primarily involved in ambient temperature regulation of *FT* [27] whereas *FLC* plays a more prominent role in the vernalization response.

With the exception of *FLC*, all plants appear to have functional homologues to these floral regulatory genes. In the two best characterized perennial model species, poplar and leafy spurge, there are homologues to genes related to *FLC*, specifically, *MADS AFFECTING FLOWERING 2* (*MAF2*). It is also noteworthy that the *FT* gene family is expanded in perennials such as poplar [28]. However, there are differences such as altered responses of *FT* to *CO* in short day flowering rice relative to long day flowering

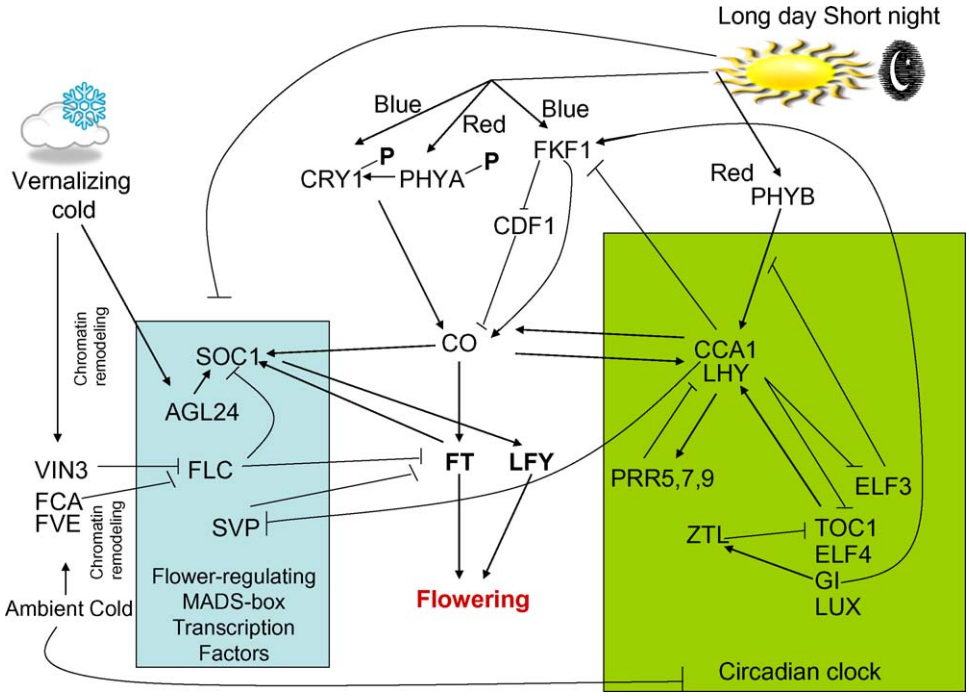

Fig. 1. A simplified schematic showing some of the floral regulatory genes that control flowering through regulation of *FLOWERING LOCUS T* (*FT*). Long day and/or short night regulated CONSTANS (CO) through the activation of PHYTOCHROME A (PHYA) and CRYPTOCHROME 1 (CRY1) and through PHYOCHROME B (PHYB) via the many components of the circadian clock [CIRCADIAN CLOCK ASSOCIATED 1 (CCA1), LATE ELONGATED HYPOCOTYL (LHY), PSEUDO-RESPONSE REGULATORS 5,7,9 (PPR5,7,9), EARLY FLOWERING 3 and 4 (ELF3,4), TIMING OF CAB EXPRESSION 1 (TOC), GIGANTEA (GI), LUX ARRHYTHMO (LUX), ZEITLUPE (ZTL)] which eventually result in inhibition of FLAVIN-BINDING, KELCH REPEAT, F BOX 1 (FKF1). FKF1 and GIGANTEA (GI) interact to both block the CO inhibiting CYCLING DOF FACTOR 1 (CDF1) during evening hours and increase expression of *CO* and *FT* in the morning. Long day and cold temperatures result in altered expression of various floral promoting and inhibiting MADS-box transcription factors such as SUPPRESOR OF OVEREXPRESSION OF CONSTANS 1 (SOC1), AGAMOUS-LIKE 24 (AGL24), FLOWERING LOCUS C (FLC), and SHORT VEGETATIVE PHASE (SVP) resulting in altered flowering responses. Cold temperatures also impact expression of FLC through chromatin remodeling via VERNALIZATION INSENSITIVE 3, and other chromatin remodeling genes. Chromatin remodeling also appears to play a role in ambient temperature responses through several autonomous pathway genes such as MULTICOPY SUPPRESSOR OF IRA1 4 (FVE), FCA (no other name), and others. Likewise, ambient cold temperatures also repress cycling of the circadian clock by constitutively inducing several key clock regulatory genes.