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FLOWERING NEWSLETTER

My favourite flowering image: the role of cytokinin as a flowering signal

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Abstract

My favourite flowering image shows a section in a shoot apical meristem of *Sinapis alba* undertaking the very first step of its transition to flowering. This step is the activation of the *SaSOC1* gene, the *Sinapis* orthologue of Arabidopsis *SUPPRESSOR OF OVEREXPRESSION OF CO1* (*SOC1*), in a few central cells of the meristem. Hidden behind this picture is my long quest of physiological signals controlling flowering. Milestones of this story are briefly recounted here and this gives me an opportunity to raise a number of questions about the nature and function of florigen.

Key words: Cytokinin, florigen, *FLOWERING LOCUS T* (*FT*), mitotic activation, organizing centre, shoot apical meristem (SAM), *Sinapis alba* (*Sa*), *SUPPRESSOR OF OVEREXPRESSION OF CO1* (*SOC1*), WUSCHEL (*WUS*).

Introduction

My interest in the flowering process spans half a century. It started in the early 1960s at a time when a group of physiologists at CalTech (Pasadena), led by James Bonner, was discovering that nucleic acid and protein biosyntheses were involved in the floral induction of photoperiodic plants (Salisbury, 1963). For young flowering scientists of those days this created great excitement. Since then, we have been progressively engulfed in a world in which the control of flowering time is explained in terms of an increasingly complex network of genetic pathways (Kobayashi and Weigel, 2007; Turck *et al.*, 2008; Amasino, 2010).

Among the numerous flowering images accumulated in my mind during these decades, I selected one (Fig. 1A) which is a longisection through a shoot apical meristem (SAM) of the long-day (LD) plant *Sinapis alba* showing initial expression of the *SaSOC1* gene*,* the *Sinapis* orthologue of Arabidopsis *SUPPRESSOR OF OVEREXPRESSION OF CO1* (*SOC1*). This image has a special significance for me for several reasons. First, activation of *SOC1* is currently viewed as the earliest critical event caused in the SAM by photoperiodic floral induction (see below). In my favourite image, however, activation of *SaSOC1* was obtained in non-inductive short days (SDs) by the application of a hormone of the cytokinin family. Secondly, the experimental frame in which this picture was obtained is actually the outcome of a story that goes back to my early studies on flowering. How my research put me on the track of cytokinins is summarized below.

Plant material

I selected *Sinapis*, the white mustard, for flowering work in 1960. This choice was unintentionally fortunate since this member of the Brassicaceae family is taxonomically related to Arabidopsis, the reference plant of today. Both species belong to the group of LD plants and recent work has shown that the gene activation cascade induced successively in leaves and SAM by a LD exposure is quite similar in the two species (D'Aloia *et al.*, 2009). In our growing conditions, *Sinapis* plants will stay vegetative for several months when grown in continuous 8 h SDs. When 2 months old in SDs, a single LD of 20–22 h was found to be sufficient to induce flowering in all plants (Bernier, 1969).

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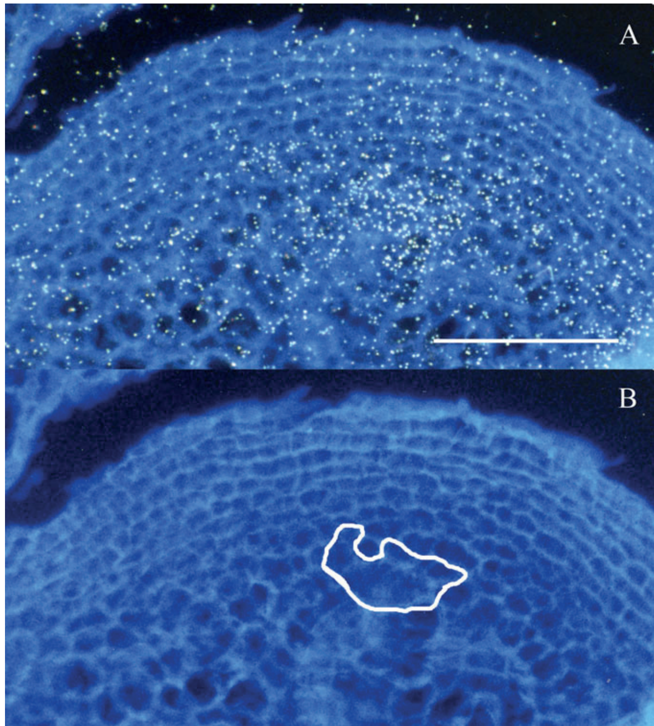

Fig. 1. (A) *In situ* hybridization showing initial up-regulation of the *SaSOC1* gene in a shoot apical meristem of the long-day plant *Sinapis alba*. This plant was continuously grown in non-inductive short days. When 2 months old, a single application of 5×10^{-5} M N^6-benzylaminopurine, a cytokinin, was made on to the plant apex at midnight and this apex was collected 8 h later. Bar=50 μm. (B) The area of cells expressing *SaSOC1* is delineated. This unpublished figure is from the PhD thesis of my graduate student Françoise Bonhomme (2002). I am very grateful to Françoise for her permission to use this figure.

The SAM response to LD induction is incredibly fast

The response of the *Sinapis* SAM to a LD induction has been of major interest to me since the early years (Bernier, 1962). A marked stimulation of the mitotic index was detected at 22–26 h after the start of the single inductive LD, i.e. at the end of the photoperiodic treatment (Bernier *et al.*, 1967). Such an early stimulation was entirely unexpected since the first floral primordia were initiated only about two days later. Undoubtedly, these extra mitoses were my first impressive flowering image. I observed that they started in the peripheral zone, before expanding rapidly to the central and rib-meristem zones. They were later shown to result from an increase in the rate of cell division (Gonthier *et al.*, 1987).

A similar early stimulation of cell division was uncovered in a variety of photoperiodic and day-neutral plants (see references in Bernier *et al.*, 1981; Bernier, 1988; Lyndon, 1998), including LD-induced *Arabidopsis* (Jacqmard *et al.*, 2003). It is thus, apparently, of general occurrence. For a long time, I had the feeling that this event was needed for, or at least facilitated, the further reprogramming of the SAM at floral transition, i.e. the necessary switches in gene expression (Bernier, 1971, 1988). Surprisingly, this problem has not attracted much interest in recent times despite the likely implication of several important flowering-time genes in the control of other cell-division related developmental processes, like potato tuberization (Rodríguez-Falcón *et al.*, 2006) and establishment of bud endodormancy (Ruonala *et al.*, 2008; Horvath, 2009).

The LD-induced up-regulation of *SaSOC1* in the SAM is concomitant with early mitotic activation

In *Arabidopsis*, the *SOC1* gene, which encodes a MADS-box transcription factor, is commonly regarded as the earliest gene whose expression is up-regulated in the SAM following floral induction by LDs (Giakountis and Coupland, 2008; Jang *et al.*, 2009; Lee and Lee, 2010). A similar situation exists in *Sinapis* for *SaSOC1*. Expression of this gene, formerly called *SaMADS A*, was not detected in the SAM of SD-grown (non-induced) plants (Menzel *et al.*, 1996) and was first up-regulated 24 h after the start of the single inductive LD (Bonhomme *et al.*, 2000), again well before the activation of genes required for floral meristem identity, such as *SaLEAFY* (*SaLFY)* and *SaAPETALA1* (*SaAP1*) (D'Aloia *et al.*, 2009). Up-regulation of *SaSOC1* was initially observed in central L3 (corpus) cells, just above the rib meristem. Expression spread later to all L3 cells (peripheral, central, and rib-meristem zones), but not to L1 and L2 (tunica) cells. These L1+L2 layers expressed *SaSOC1* only when plants were induced by more than one LD (Bernier and Bonhomme, 2001).

A trigger of both early mitoses and *SaSOC1* up-regulation is a leaf-generated cytokinin

In photoperiodic plants, the events of the floral transition at the SAM are known to be set in motion by the arrival of a leaf-generated signal called 'florigen' (Lang, 1965). In the early days, I wondered whether this assertion held for all events observed during this transition, in particular for the early mitotic activation. In *Sinapis*, using sequential defoliations of induced leaves, it was found that leaf removal, within the time interval of 8–16 h following the start of LD, suppressed mitotic activation in the SAM, whereas later defoliations did not (Bernier *et al.*, 1974). The results showed that the trigger of extra mitoses was of leaf origin and defined the time window of its movement.

Next, I attempted to identify this trigger and used two approaches: (i) exogenous applications to identify mitotic substance(s) that would mimic the inductive LD when applied once to SD-grown (non-induced) *Sinapis* plants, and (ii) exudate analyses to identify the mitotic substance(s) exported from induced leaves. Applications were tested directly on the apex in order to avoid translocation problems between the leaves and the SAM and at midnight,

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i.e. at a time corresponding to start of the mitogen movement in induced plants. To my delight, it turned out that, in these conditions, only a cytokinin, either zeatin or N^6-benzylaminopurine (BAP), was able to cause the early mitotic activation in the SAM in the absence of LD induction (Bernier *et al.*, 1977). Other hormones, like gibberellins, or sucrose had no such effect. Remarkably, the cytokinin treatment perfectly mimicked the LD effect, causing mitotic activation in the same SAM zones and with the same amplitude and kinetics.

The cytokinin application, however, did not induce flowering, i.e. it was unable to cause initiation of floral primordia. On the other hand, with this experimental design in hand, the stage was set to disclose other SAM events that are observed during the floral transition induced by a LD and could be mimicked by this hormone. For example, vacuole fragmentation (Havelange *et al.*, 1986) and increased frequency of secondary plasmodesmata (Ormenese *et al.*, 2006) were observed. However, the most exciting event induced by a cytokinin application at midnight to non-induced plants was the up-regulation of the *SaSOC1* gene (Bonhomme *et al.*, 2000), as shown in my favourite flowering image. What impressed me was that, although the time of treatment was chosen according to the kinetics of mitogen movement, the applied cytokinin did mimic perfectly in time and space the initial effect of the LD on *SaSOC1* expression.

In parallel, the exudate analyses demonstrated that a cytokinin of the isopentenyladenine (iP) type was present in greater amounts in the phloem sap collected at either the leaf exit or apex entry in plants of *Sinapis* induced by one LD, when compared with non-induced plants (Lejeune *et al.*, 1988, 1994). This enrichment occurred during the period corresponding to the time of movement of the mitotic trigger. As a result, the iP content of the SAM itself increased markedly (Jacqmard *et al.*, 2002).

These data conclusively showed that, in *Sinapis*, a cytokinin of leaf origin was a trigger of several SAM events which are normally observed early during the floral transition, e.g. extra mitoses and *SaSOC1* up-regulation (Bernier *et al.*, 1993). These events, however, were not sufficient to reach floral primordium initiation and it was later confirmed that the exogenous cytokinin was unable to up-regulate genes acting downstream of *SaSOC1*, like *SaLFY* (D Bonhomme, unpublished data) and *SaAP1* (Bonhomme, 2002).

How is the work dispatched between the cytokinin and SaFT, the presumptive *Sinapis* florigen, at the SAM floral transition?

The provocative side of the above conclusion is that the cytokinin is viewed as performing, in *Sinapis*, part of the work (i.e. essentially *SaSOC1* up-regulation) usually attributed to florigen.

The protein FLOWERING LOCUS T (FT) was recently identified as the *Arabidopsis* florigen (Corbesier *et al.*, 2007) or at least as its 'center' piece (Turck *et al.*, 2008) and indeed this protein is generally regarded as the trigger of *SOC1* in LD-induced *Arabidopsis* plants (Giakountis and Coupland, 2008; Lee and Lee, 2010). In *Sinapis*, *SaFT* was recently found to be up-regulated in the leaves exposed to the inductive LD during the 16–32 h time interval (D'Aloia *et al.*, 2009). Long ago, the time of movement of the LD florigen, also called the 'floral stimulus', from leaves to SAM was determined in *Sinapis* using the same sequential defoliation technique as above, but judging its effect on floral primordium initiation rather than mitotic activation. It was found that the florigen moved during the 16–28 h time interval (Kinet *et al.*, 1971). There is thus a remarkable correspondence in timing between recent data on *SaFT* expression and the results on florigen movement obtained during 'pregenomic' times. This correspondence further indicates that movement of the presumptive florigen, SaFT, starts soon after activation in leaves of the corresponding gene and occurs at the same time as movement of the cytokinin, iP (see above). The two signals thus appeared to be transported to the SAM side by side. At this stage, one may wonder what function(s) are fulfilled by each signal there. Based on current knowledge in *Arabidopsis*, the most popular answer would certainly be that, in *Sinapis*, SaFT is responsible for the whole gene activation cascade occurring in the SAM at floral transition, including *SaSOC1* activation. Accordingly, the cytokinin would be regarded as a SaFT travel companion having little or no importance, except perhaps in the early mitotic activation. The inability of cytokinin to cause flowering reinforces this view.

An alternative view, which is in line with my long-held idea of a multifactorial florigen (Bernier *et al.*, 1981; Bernier, 1988; Bernier and Périlleux, 2005), would be that the cytokinin and SaFT co-operate at the SAM, the cytokinin being responsible for *SaSOC1* up-regulation and mitotic activation and SaFT triggering critical processes downstream of *SaSOC1*.

That this last view could also be valid for *Arabidopsis* is supported by two sets of data. First, my group showed that the cytokinin contents of leaves, phloem sap, and SAM increased in response to LD induction in *Arabidopsis*, just as in *Sinapis* (Corbesier *et al.*, 2003). This increase in the phloem sap was concomitant with movement of the *Arabidopsis* florigen, as determined in defoliation experiments (Corbesier *et al.*, 1996).

Second, using hydroponically-grown *Arabidopsis* plants, the group of Claire Périlleux in Liège recently discovered that an 8 h supply of BAP to roots was sufficient to induce flowering in SDs (D'Aloia *et al.*, 2011). In this case, the exogenous cytokinin seemingly acts alone but, in fact, it was found that its florigenic effect, which bypasses *FT,* absolutely requires activity of its paralogue *TWIN SISTER OF FT* (*TSF*). As in *Sinapis*, the flowering process requires the presence of both a cytokinin and a partner, TSF, capable of substituting for FT (Yamaguchi *et al.*, 2005); but, unlike in *Sinapis*, the cytokinin itself activates its necessary partner in leaves. Interestingly, the cytokinin also up-regulates *SOC1* in the SAM, as it did in *Sinapis.* The

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exact function of TSF in this system has not yet been determined.

These two sets of data collected in *Arabidopsis* reinforce the idea that cytokinin acts in the floral transition process as a long-distance signal.

Is the initial expression of *SaSOC1* in the SAM organizing centre fortuitous?

Another striking feature of my favourite flowering image is that the initial activation of *SaSOC1* by cytokinin in *Sinapis* occurred in a zone of the SAM (Fig. 1B), which perfectly matches what is called the 'organizing centre' (Haecker and Laux, 2001). To my knowledge, this feature has never been reported before, leading me to wonder whether it was a fortuitous observation or not. Let us suppose it was not and then consider the possible significance of what we see in Fig. 1A. The organizing centre is defined as a small central subapical zone of the Arabidopsis SAM whose cells specifically express the homeodomain transcription factor *WUSCHEL* (*WUS*). *WUS* participates with the *CLAVATA* (*CLV*) genes in a feedback regulatory loop that plays a central role in maintenance of the apical pool of pluripotent stem cells and, consequently, in maintenance and growth of the SAM itself (Wang and Li, 2008).

Initial expression of *SaSOC1* in the presumptive *Sinapis* organizing centre raises the possibility that this expression requires activity of *SaWUS*. Such a situation has already been reported in the case of *AGAMOUS* (*AG*), an *Arabidopsis* gene which specifies stamen and carpel identities and encodes, like *SaSOC1*, a MADS-box transcription factor (Meyerowitz, 1998). *AG* up-regulation started in the centre of floral meristems at stage 3 and, although it occurred in a larger zone than *WUS*, it was shown that it required *WUS* activity (Lohmann *et al.*, 2001). Accordingly, *WUS* was regarded by these authors as providing spatial specificity to *AG* expression. Does *SaWUS* play the same role for *SaSOC1*?

More generally, the situation disclosed in Fig. 1A opens the question of the relationships between the increased cytokinin supply to SAM at floral transition, *SaSOC1* activation, and SAM homeostasis. Recent work shows that cytokinin, as the *CLV/WUS* circuit, is involved in SAM maintenance and growth. Cytokinin receptors, at least some of them, were found to be spatially restricted in the *Arabidopsis* SAM, with higher expression in an area corresponding to the organizing centre (Gordon *et al.*, 2009). On the other hand, external cytokinin activates *WUS* expression through both *CLV*-dependent and *CLV*-independent pathways (Lindsay *et al.*, 2006; Gordon *et al.*, 2009), and *WUS* is known to repress the type A *ARR* (*ARABIDOPSIS RESPONSE REGULATOR*) genes, which act as negative feedback regulators of cytokinin signalling (Leibfried *et al.*, 2005). Thus, the cytokinin signal and *WUS* reinforce each other through positive feedback loops and these loops essentially take place where cytokinin receptors are concentrated, i.e. in the organizing centre. Initial activation of *SaSOC1* in this centre at floral transition is quite possibly related to this localized interplay of cytokinin and *WUS*.

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