ENERGETIC HOMEOSTASIS AND SUGAR SIGNALING:
DIVERSIFICATION OF THE MOLECULAR MECHANISMS INVOLVED IN
THE CONTROL OF THE ENERGETIC BALANCE IN ANGIOSPERMS

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To optimize their growth and development, plants, as sessile organisms, have developed a range of efficient mechanisms to sense and respond adequately to ever changing environmental conditions. The production of sugar through photosynthesis primarily relies on light accessibility. These photosynthetically-derived sugars represent important signals, which, by interacting with the circadian clock, and in combination with developmental and environmental cues, such as mineral nutrition, water availability or pathogens attacks, influence the use of energy resources to ensure survival and propagation. Interaction between developmental, hormonal and sugar regulatory signals is deeply involved in growth control and ultimately in biomass production. The molecular mechanisms responsible for the cross talk between these different signaling pathways and their diversification in plants still need to be further elucidated to better understand plant growth patterns and biomass production. The present proposal aims at unraveling new mechanistic aspects of sugar signal transduction in plants and more broadly at how energetic homeostasis is controlled. We anticipate that the data will improve our view of sugar signaling and energy homeostasis control in plants and the results will be integrated into databases that could feed projects related to biomass and bioenergy research.

Figure 1. Putative AtbZIP63 target genes. The expression of ASN1 (At3g47340) and SEN1 (At4g35770) is misregulated in two AtbZIP63 T-DNA insertion mutants after dark-induced energy starvation. A, Schematic representation of T-DNA insertion sites in atbzip63-1 and atbzip63-2 mutants. LB, T-DNA left border; RB, T-DNA right border. Primers used to locate T-DNA insertion and detect chimeric transcripts are indicated by the arrows. B, PCR amplification from genomic DNA of the T-DNA insertion region and RT-PCR after DNase treatment showing chimeric transcripts between AtbZIP63 and T-DNA in both atbzip63-1 and atbzip63-2 mutants. Size differences between amplification products from genomic DNA (1,200 bp) and cDNA (810 bp) in atbzip63-1 are due to introns that are absent in spliced AtbZIP63 mRNA. C, ASN1, SEN1, and DIN10 transcript accumulation in atbzip63-1 (Col-0 ecotype) and atbzip63-2 (Ws ecotype) 6-d-old seedlings after 24 h of dark treatment. Significant differences related to seedlings of the same genotype without dark treatment (light) are represented by the letter a (n = 3; P < 0.05) and those between equally treated mutants and their respective wild-type genotype are represented by the letter b (n = 3; P < 0.05). (adapted from Matioli and al., 2011)
SUMMARY OF RESULTS TO DATE AND PERSPECTIVES

The Arabidopsis thaliana (A. thaliana) Transcriptional Regulatory Factor (TF) of the basic leucine type AtbZIP63 is a key regulatory node that integrates energetic status, abiotic and biotic signals to adjust growth and development in phase with the diurnal cycle. Comparative RNA profiles analysis and Chromatin Immuno Precipitation approaches revealed that AtbZIP63 interacts with the circadian clock to control starch degradation and is a key intermediate of SNF1 Related kinases 1(SnRK1)-mediates energetic responses (Figure 1). Deregulated starch usage at night in atbz63 mutant lines resulted in severe growth defects involving reduction of cell wall expansion. We are in the process of unraveling new aspects of the regulatory networks in which AtbZIP63 is involved and we mainly are focusing on its role in mediating the interaction between the two functionally antagonist and evolutionary conserved pathways that control growth in response to the available energy: the Target of Rapamycin (TOR)-related pathway and the SnRK1-related pathway.

We found that the control of AtbZIP63 expression is under a complex set of regulatory mechanisms including, transcriptional, mRNA decay control and protein degradation. The stress-related hormone Abscisic Acid (ABA) and Glucose interact to promote AtbZIP63 mRNA degradation. The underlying mechanisms are being investigated. This data prompted us to analyze at genomic scale the extent of mRNA decay regulation mediated by ABA and/or Glucose. We found that ABA possibly negatively feed-back regulates its own signaling pathway probably as a way to reset the signalization by promoting destabilization of mRNA of ABA receptors and ABA-activated TFs. The mechanisms involved are being investigated.

We obtained clear evidences for the existence of a mannose-specific signaling pathway and the details of the signaling process are being revealed including the identification of a potential Mannose receptor. Our working hypothesis is that Mannose represents a Cell wall-derived Damage Associated Molecular Pattern.

We identified a de novo originated Arabidopsis thaliana gene called QQS which is involved in the control of starch metabolism. QQS is prone to epigenetic switches that impact its expression in a manner independent of genetic variation. This gene represents a new tool to obtain new insights into the role of epigenetic variation in adaptation/evolution and how epialleles arise (Figure 2). We found more recently that the epigenetic state of QQS and developmental signal interacts to define the expression pattern of this gene during development and obtained evidences that the demethylase ROS1 is also involved. Remarkably, QQS is specifically demethylated in the male germ line cells and we propose that this event is important step in QQS evolution.

MAIN PUBLICATIONS


Figure 2. QQS is embedded in a repeat-rich region. (A) Genomic structure of the QQS locus (30 kb window) in the Col-0 accession. Dark grey boxes represent TE sequences. (B) Magnified view of the QQS gene and upstream sequences, showing tandem repeats (TR), methylation of cytosine residues (5 mC) at the three types of sites (CG, CHG and CHH, H = A, T or C) and locus-specific sense and antisense siRNAs (numbers referring to copy number). (adapted from Siveira et al., 2013)

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