

Crosstalks between Myo-Inositol Metabolism, Programmed Cell Death and Basal Immunity in *Arabidopsis*

Ping Hong Meng^{1,9}, Cécile Raynaud^{1,9*}, Guillaume Tcherkez², Sophie Blanchet¹, Kamal Massoud¹, Séverine Domenichini¹, Yves Henry¹, Ludivine Soubigou-Taconnat³, Caroline Lelarge-Trouverie², Patrick Saindrenan¹, Jean Pierre Renou³, Catherine Bergounioux¹

1 Institut de Biotechnologie des Plantes, UMR CNRS 8618, Université Paris-Sud XI, bât 630, Plateau de Moulon, Orsay, France, **2** Plateforme Métabolisme Métabolisme IFR87, Institut de Biotechnologie des Plantes, Université Paris-Sud XI, bât 630, Plateau de Moulon, Orsay, France, **3** Unité de Recherche en Génétique Végétale, 2, CP5708, Evry, France

Abstract

Background: Although it is a crucial cellular process required for both normal development and to face stress conditions, the control of programmed cell death in plants is not fully understood. We recently reported the isolation of ATXR5 and ATXR6, two PCNA-binding proteins that could be involved in the regulation of cell cycle or cell death. A yeast two-hybrid screen using ATXR5 as bait captured AtIPS1, an enzyme which catalyses the committed step of myo-inositol (MI) biosynthesis. *atips1* mutants form sensitive lesions on leaves, raising the possibility that MI metabolism may play a role in the control of PCD in plants. In this work, we have characterised *atips1* mutants to gain insight regarding the role of MI in PCD regulation.

Methodology/Principal Findings: Lesion formation in *atips1* mutants depends of light intensity, is due to PCD as evidenced by TUNEL labelling of nuclei, and is regulated by phytohormones such as salicylic acid - MI and galactinol are the only metabolites whose accumulation is significantly reduced in the mutant, and supplementation of the mutant with these compounds is sufficient to revert PCD - the transcriptional profile of the mutant is extremely similar to that of lesion mimicking mutants such as *cpr5*, or wild-type plants infected with pathogen.

Conclusion/Significance: Taken together, our results provide strong evidence for the role of MI or MI derivatives in the regulation of PCD. Interestingly, there are three isoforms of IPS in *Arabidopsis*, but AtIPS1 is the only one harboring a nuclear localization sequence, suggesting that nuclear isoforms may play a specific role in PCD regulation and opening new research prospects regarding the role of MI in the reventive tumorigenesis. Nevertheless, the significance of the interaction between AtIPS1 and ATXR5 remains to be established.

Citation: Meng PH, Raynaud C, Tcherkez G, Blanchet S, Massoud K, et al. (2009) Crosstalks between Myo-Inositol Metabolism, Programmed Cell Death and Basal Immunity in *Arabidopsis*. PLoS ONE 4(10): e7364. doi:10.1371/journal.pone.0007364

Editor: Mohammed Bendahmane, Ecole Normale Supérieure, France

Received: February 19, 2009; **Accepted:** September 16, 2009; **Published:** October 8, 2009

Copyright: © 2009 Meng et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Funding: P.H. Meng was supported by a fellowship from the Chinese Government. This work was supported by the IFR 87 "L'Analyse et le Sens de l'Environnement" (www.ifr87.cnrs-gif.fr). The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Competing Interests: The authors have declared that no competing interests exist.

* E-mail: cecile.raynaud@u-psud.fr

† Current address: Institute of Horticulture, Guizhou Academy of Agricultural Sciences, Guiyang, Guizhou Province, People's Republic of China

‡ These authors contributed equally to this work.

Introduction

Plants are sessile organisms and are thus exposed to a wide range of environmental stresses. In response to these stresses, plants have evolved a complex signaling network that involves the perception of the stress, the transduction of the signal, and the activation of defense responses. One of the most important defense responses is programmed cell death (PCD), which is a highly regulated process that leads to the death of cells. PCD is essential for the development of plants and for their survival under stress conditions. In plants, PCD is controlled by a complex network of signaling molecules, including hormones and secondary metabolites. Myo-inositol (MI) is a secondary metabolite that is involved in the regulation of PCD in plants. MI is a polyhydroxylated compound that is synthesized from glucose and inositol phosphate. MI has been shown to be involved in the regulation of PCD in plants, and its accumulation is significantly reduced in mutants that are deficient in MI biosynthesis [1].

MI is a polyhydroxylated compound that is synthesized from glucose and inositol phosphate. MI has been shown to be involved in the regulation of PCD in plants, and its accumulation is significantly reduced in mutants that are deficient in MI biosynthesis [1]. MI is a secondary metabolite that is involved in the regulation of PCD in plants. MI is a polyhydroxylated compound that is synthesized from glucose and inositol phosphate. MI has been shown to be involved in the regulation of PCD in plants, and its accumulation is significantly reduced in mutants that are deficient in MI biosynthesis [1].

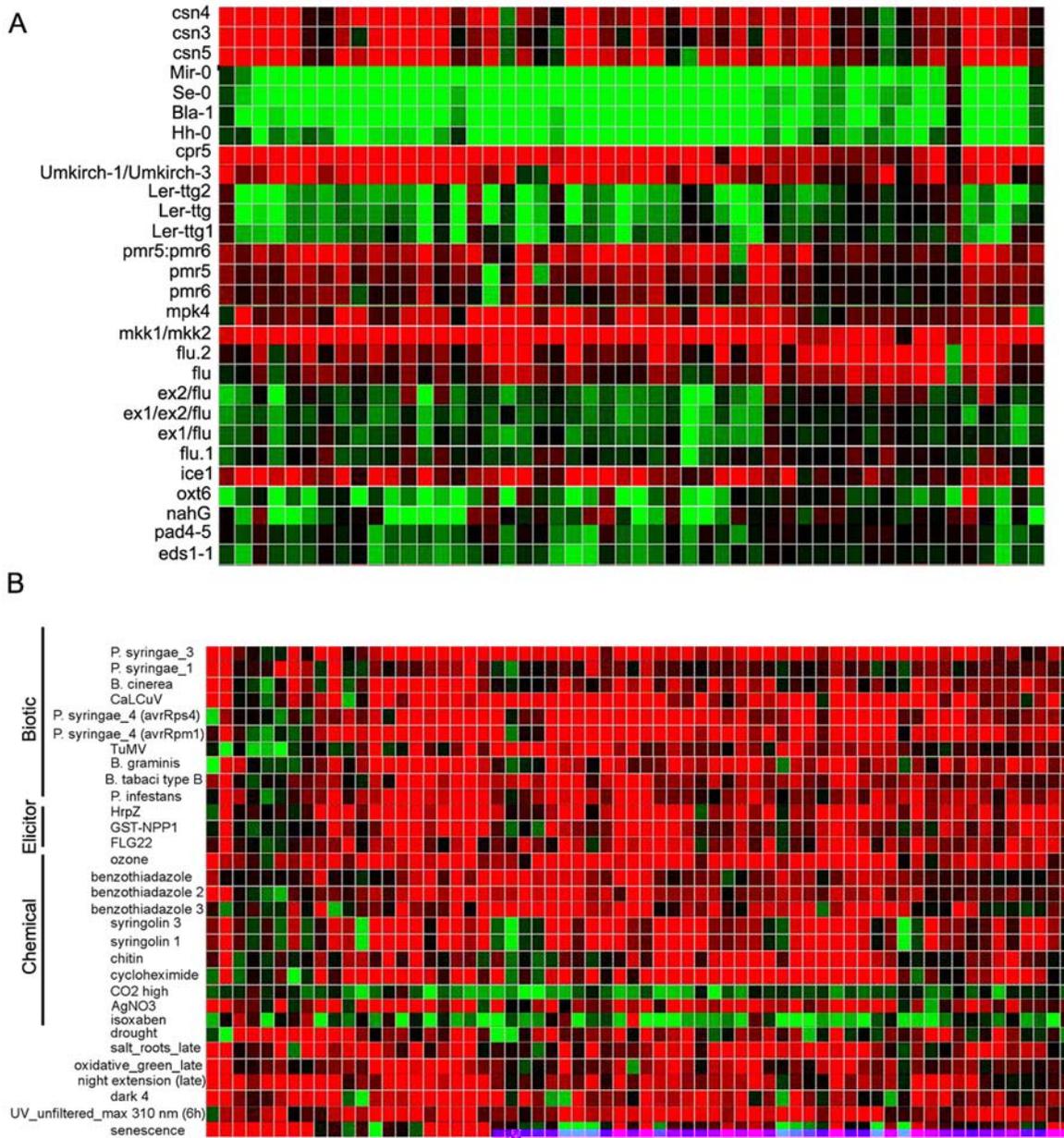


Figure 6. The transcriptome of *atips1-1* is similar to that of several LMM mutants or plants infected by pathogens. Hierarchical clustering was performed using 150 transcripts across the different SD/LD conditions. Each vertical line displays the expression data for one gene. List of genetic backgrounds or treatments are displayed horizontally. Red and green indicate up- and down-regulation in mutants (A) or treated plants (B) compared to wild-type or untreated plants, respectively. Intensity of the colours is proportional to the absolute value of the fold difference. Images presented here correspond to a representative region of the global image which was too wide to be reproduced integrally. doi:10.1371/journal.pone.0007364.g006

M... et al... *atips1-1*... *AtIPSI*... *atips1* is a LMM displaying enhanced basal defence... *AtIPSI*... *atips1*... *SAG12*...

8. **NYG S, KV6Heh** (2009) **AK d i AK n BE7 m** **h q 6**
bioRxiv 6: 763768.

9. **AV, DV983** **h h h h**
Arabid 4: 137461.

10. **AV, W2000** **h h h h** 50:
 149.

11. **BHSDhR** (1995) **A p o i n**
e h7: 10994111.

12. **hVA, BA, hCpRch**
 (1996) **h h h h**
h h 9: 537548.

13. **hVA** (1993) **A h h h h**
3- h h h h h h
h h h *Spirodela polyrrhiza*. **h** 4: 279293.

14. **hCS** (1997) **h h h h h**
h h h h h h h h 3:
 811820.

15. **h** (2003) **h h, 2,3,4,5,6- h h** 4:
 10334043.

16. **hVP hK, hDSch** (2000)
h h h h h -1 **h h** 2-1.
h h 24: 355368.

17. **hAC hC hA h** **h h** (2006)
h h h h h h h h
(GmMPSI) **h h h h h h h**
h h 24: 125432.

18. **hSA, hV1**
h h h h h h h
h h h h h 16: 403410.

19. **hV1** (1995) **1 h h 1- h h h**
Arabidopsis thaliana. **h** 07: 613619.

20. **hV1 hA, hW2008** **A h h**
h h h h h h h
h h 56: 638652.

21. **hV1 h** (1997) **h h h**
h h h h h 6: 277282.

22. **hV1 h** **h h h h h h** (1997)
h h h h h
h h 305: 295305.

23. **hV1 h** (2000) **h h h** **h h**
h h h h h h h 97:
 978994.

24. **hV1 h** (1999) **h h**
h h h h h
h h 85: 15794582.

25. **hV1 h** **h h h h** (1999)
GIGANTEA: a h h h h h h
h h h h h
h h 8: 46794688.

26. **hV1 h** **h h h h** (1995)
h h h h h h h h
h h h h: 863870.

27. **hV1 h** (2003) **h h h h h**
h h *Pseudomonas syringae pv. phaseolicola* **h h h h**
h h 33: 733742.

28. **hV1 h** **h h** (1999) **h h h h**
h h h h 1: 13934404.

29. **hV1 h** (2001) **h h**
h h h h h 14: 562565.

30. **hV1 h** **h h** (2008) **h h h**
h h h h h 3: 589595.

31. **hV1 h** **h h h h** (2002) **A**
h h h h h
h h 31: 142.

32. **hV1 h** **h h h** (2007)
A h h h h h h h h
h h h h h
h h 1: 161474.

33. **hV1 h** (2008) **h h h**
h h h h h h h 147:
 12514263.

34. **hV1 h** **h h h h** (2004)
h h h h h
h h 36: 26212632.

35. **hV1 h** **h h h h** (2007)
A h h h h h h h
h h h h 55: 19624972.

36. **hV1 h** (1997) **h h**
h h h h h
h h 9: 15734584.

37. **hV1 h** **h h h h** (2008) **A h h**
h h h h h h
h h h h h
 148: 212222.

38. **hV1 h** **h h h h** (2000)
A h h h h h
h h 103: 11114120.

39. **hV1 h** **h h h h** (1996)
h h *Peronospora parasitica* **h h h h**
h h 20332046. **RPP g h h**:

40. **hV1 h** **h h h h** (1994) **A**
h h h h h h 8: 12474250.

41. **hV1 h** (2003) **h h h h**
h h h h 32: 22402247.

42. **hV1 h** **h h h h** (2003) **h h**
h h h h h 36: 629642.

43. **hV1 h** **h h h**
h h h h h (2004)
h h h h h 36: 28182830.

44. **hV1 h** (2005) **h h**
h h h h h h 4: 683690. **GIGANTEA g h h**

45. **hV1 h** (1998) **h h**
h h h h h h h
h h 14: 759764.

46. **hV1 h** **h h**
h h h h h h h h
h h h h 8: 65660. **R h h h h** (2008) **R**

47. **hV1 h** (2008) **h h h**
h h h h h 59: 43544.

48. **hV1 h** **h h h h** (2007) **h**
h h h h h h
h h h h h 51:
 941954.

49. **hV1 h** **h h h h h h h h**
h h h h (2008) **h h h h**
h h h h h
h h 20: 23392356.

50. **hV1 h** **h h h h** (2006) **h h**
h h h h h h h 18:
 11214133.

51. **hV1 h** (1993) **h h h h**
h h *CAB d RBCS g h h*
h h 4: 787799.

52. **hV1 h** (2005) **h h h h**
h h h h
lesion initiation 1 h h h:
 19294934.

53. **hV1 h** (2008) **h h h h**
h h h h h 15: 33344.

54. **hV1 h** (2005) **h h h**
h h h h
Alternaria alternata h h
h h 8: 8999.

55. **hV1 h** **h h h h** (2003) **h h**
h h h h 7: 26362641.

56. **hV1 h** **h h h h** (2002) **h h**
h h *ACCELERATED-CELL-DEATH11 h h*
h h 6:
 490502.

57. **hV1 h** **h h h h** (2008) **A h**
h h h *h h h h h h* 20:
 31633179. **h h h h h h**

58. **hV1 h** **h h h**
h h h h (2007) **h h h h**
h h h h h h h
h h *cat2 h h h h h h*
h h O_2 -**h h h** 52: 640657.

59. **hV1 h** **h h h h** (2008) **h h**
h h h h h
Pseudomonas
chlororaphis **h h h h** 21: 16434653.

60. **hV1 h** **h h h h** (2009)
h h h h h h h
h h 1, **h h h h h h**
h h

61. **hV1 h** **h h h h** (2003) **h h**
h h h
Arabidopsis thaliana. **h** 01: 653657.

62. **hV1 h** (1989) **h h h**
h h h

63. **hV1 h** (1999) **h h**
h h h h h h
h h h h h 12: 118190. **arcA3**

64. **hV1 h** **h h h h** (2002) **h h h h** **h h h h**
h h h h h

65. [doi:10.1371/journal.pone.0157392](#).
[doi:10.1371/journal.pone.0157392](#).
65. [doi:10.1371/journal.pone.0157392](#).
66. [doi:10.1371/journal.pone.0157392](#).
67. [doi:10.1371/journal.pone.0157392](#).
68. [doi:10.1371/journal.pone.0157392](#).
69. [doi:10.1371/journal.pone.0157392](#).