Proceedings of the Joint Congress SIBV-SIGA
Pisa, Italy – 19/22 September, 2017
ISBN **978-88-904570-7-4**

Poster Communication Abstract – 6.30

A SPONTANEOUS MUTATION THAT CONFERS HYGROMYCIN RESISTANCE TO ARABIDOPSIS

LOMBARDI L.*, VAN VEEN H.**, LICAUSI F.*

*) Department of Biology, University of Pisa,Via Ghini 10, 56126 Pisa (Italy)
**) Institute of Environmental Biology, Utrecht University, Utrecht (Netherlands)

hygromycin, antibiotic resistance, Arabidopsis thaliana, shoot apical meristem

Resistance to the translation-inhibitor hygromycin is widely used as a selectable marker to identify plants carrying a transgene of interest. Usually, this trait is conferred through phosphorylation of the antibiotic by a bacterial kinase *Hygromycin phosphotransferase* (*Hpt*), which abolishes hygromycin binding to the of 18S rRNA.

On hygromycin-containing medium, arabidopsis seedlings expressing *Hpt* show severe growth inhibition with no or strongly delayed emission of leaves. The shoot apical meristem (SAM) is present but remains inactive, as demonstrated by the unaltered expression of SAM marker genes and the very low levels of mRNAs involved in leaf primordia and lateral organ emission. SAM activity is repressed until seedlings are transferred to soil, when the shoot starts to develop from the apex or from activated lateral shoot meristems.

We recently identified an arabidopsis mutant that shows natural resistance to hygromycin. This spontaneous Col-0 mutant, called *HR*, does not contain the bacterial marker gene *HPT* but achieves hygromycin resistance through a mechanism which is still to be elucidated. The mutation is dominant and does not confer resistance to other antibiotics with a similar site of action. Moreover, SAM activity in HR plants follows a normal pattern with leaf primordia being produced also in the presence of hygromycin. Genetic, molecular and metabolic analyses are currently aimed at the identification of the HR mutation since this can be considered a very useful tool to study the mechanisms underlying SAM development and the role of hygromycin repressing its activity.