Three specifically appearing membrane proteins were found in the membrane fractions of cells grown on sulfanilic acid. The aminotransferase is probably one component of the sulfanilic acid converting enzymes catalyzing the deamination of sulfanilic acid. It was shown to be poorly membrane associated, since it was also found in the soluble fractions. The second protein contained motifs of ATP-binding casettes indicating the energy-dependence of sulfanilic acid uptake. The third protein is a hypothetical TonB-dependent protein, which might play a role in many types of transport including iron uptake. The expression of the TonB-dependent protein is upregulated specifically by xenobiotics/aromatics and iron. Since, two enzymes of the degradation pathway are known to contain iron in their active center, it is plausible to assume, that the TonB dependent protein is involved in the iron transport to feed the extra iron demand of the enzymes taking part in the biodegradation.

From our data it is assumed that the uptake and conversion of sulfanilic acid is linked to a membrane protein complex and this association can function as a self-defending mechanism for the cell against the cytoplasmic occurrence of the toxic substrates. Furthermore, a potential link between the xenobiotics degradation and iron transport is suggested.

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Supervisor: Gábor Rákhely
E-mail: anna.gara@gmail.com

Role of salicylic acid pretreatment on the photosynthetic performance and the generation of reactive oxigen species and nitric oxide in tomato plants (*Solanum lycopersicum* Mill. L. cvar. Rio Fuego) under salt stress: acclimatization or programmed cell death

Katalin Gémes

Department of Plant Biology, University of Szeged, Szeged, Hungary

Salicylic acid (SA) has long been known as a signal molecule in the induction of defense mechanisms in plants (Raskin 1992) and it was shown to improve the acclimation to different abiotic stress factors, including high salinity (Szepesi et al. 2009). SA increased reactive oxygen species (ROS) production and therefore oxidative stress (Knörzer et al. 1999). SA was also shown to influence a number of physiological processes (Raskin 1992) e. g. inhibited the activity of Rubisco and thus the photosynthetic activity (Vernooij et al. 1994).

The aim of my work was to reveal, how ROS production ($O^{\cdot}$; H_2O_2) was modified by different concentrations of SA, how the SA-treated cells could acclimate to oxidative stress or why other tissues became committed to programmed cell death (PCD).

On the basis of the inhibity effect of SA on stomatal conductance and photosynthetic performance, which has been documented in several papers, it was presumed, that the ROS produced after SA pretreatment may be derived from an inhibited photosynthetic electron transport. This may also reduce the plants capacity to synthesise compatible osmolytes, such as sugars, during pretreatment or salt stress.

That is why we measured the changes in photosynthetic activity (chl*a* fluorescence induction parameters, CO_2 fixation rate as function of PAR or Ci and stomatal conductance) during pretreatment.

Tomato plants were grown hydroponically in the presence of different SA (10^{-3} M, 10^{-4} M, 10^{-7} M). Seven-week-old plants were exposed to 100 mM NaCl for a week.

Short-term pretreatment of plants with 10^{-3} M SA resulted in a permanent decrease in the stomatal conductivity and the CO_2 fixation rate compared to the control and also decreased the viability of plants. In contrast, after a transient decline photosynthetic parameters of plants grown in 10^{-7} and 10^{-4} M SA were not significantly different from the untreated control at the end of the pretreatment period. Salt stress also inhibited the photosynthetic activity, which was significantly alleviated by 10^{-4} M SA. The improved photosynthetic performance and the accumulation of soluble sugars as compatible osmolytes resulted in a partial osmotic adjustment and contributed to successful acclimation to high salinity in 10^{-4} M SA pretreated plants.

The accumulation of putrescine in the leaves and those of spermidine and spermine in the roots are adaptive feature of some halophyte species. We found similar changes in the polyamine spectrum of plants grown in 10^{-4} M SA at the end of pre-treatment period. Moreover these tissues produced less ethylene, a PCD inducing plant hormone, which coincided with higher viability of root apical cells.

As it was expected a significant accumulation of H_2O_2 occurred in the leaves and roots of plants exposed to 10^{-4}-10^{-7} M SA, but after three weeks the differences disappeared in the root tissues and remained in the leaves. We prepared mesophyll protoplasts as model system to investigate the effects of the compounds that accumulated in plants during pre-treatments on ROS production and to compare the results with intact plants.

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Supervisor: Irma Tari
E-mail: gemeskatalin@freemail.hu

The role of Drosophila formin dDAAM in axon growth

Rita Gombos

Institute of Genetics, Biological Research Center, Hungarian Academy of Sciences, Szeged, Hungary

In the developing nervous system, the growth cones have an essential role in guiding the axons to their targets. Directed growth cone motility in response to extracellular cues is produced by the coordinated regulation of peripheral F-actin and central microtubule networks. The peripheral F-actin is organized into long bundled actin filaments in the finger-like filopodia and diffuse networks of shorter actin filaments contained in the veil-like lamellipodia. Previous work identified many immediate regulators of F-actin dynamics in growth cones, and for some of these, it has been demonstrated that they act downstream of signaling pathways involved in axonal growth regulation. Key regulators of actin dynamics are the so called nucleation factors, such as the Arp2/3 complex and formins, which use different mechanisms to seed new actin filaments (Pak CW et al. 2008).

The formin proteins are involved in actin polymerization and growth by associating with the fast-growing end (barbed end) of actin filaments. Formins contain two homology domains the FH1, and the FH2. The actin nucleation-promoting activity of formins has been localized to the FH1-FH2 domains. The FH1 domain is a proline-rich region, that can bind the G-actin monomer carrying profilin protein. The FH2 domain is necessary and sufficient to nucleate actin in vitro (Goode BL and Eck MJ 2007).

The principal field of research in our group is the functional analysis of the single *Drosophila DAAM* ortholog. We revealed that *dDAAM* is transcribed pan-neurally from stage 11 of embryogenesis in the area of central nervous system (CNS). The immunostaining experiments demonstrated that dDAAM protein is highly enriched in neurites, where it shows a strong colocalization with actin. Therefore we wanted to investigate if this protein plays a role in neurite growth.

To examine the function of *dDAAM* in the CNS, we first carried out a loss of function (LOF) analysis by examining mutant embryonic ventral nerve cord and primary neuronal cultures. The mutant embryos showed strong CNS phenotypes, displayed frequent breaks in the connectives and commissures. The *dDAAM* mutant neurons were able to develop axons of similar length as their wild-type counterparts, but the filopodia of mutant neurons were reduced in number and in length. Thus, the LOF experiments suggest that *dDAAM* regulates filopodium formation in neuronal growth cones.

Next we examined the effect of the constitutively active form of dDAAM on the embryonic CNS. We detected that the overexpression of this form in the embryonic CNS results in severe fasciculation defects and embryonic lethality. To collect additional evidences that activated dDAAM has the potential to increase neurite number, we examined embryonic nerve cord cultures overexpressing this form. The ventral nerve cords expressing activated dDAAM exhibited a much denser neuritic meshwork and growed more extended axons than their wild type counterparts. In cultured primary neurons, the overexpression of activated protein increased the number of filopodia. These observations suggest that dDAAM plays a major role in the regulation of axonal growth, presumably by promoting actin assembly in the growth cone (Matusek et al. 2008).

In addition, we detected strong dDAAM expression in adult brain as well. In LOF analysis we found several axonal projection defects indicating a role in the formation of the adult neuropil.

To determine proteins that may act together with dDAAM in the regulation of axonal growth, we carried out a genetic interaction analysis. We demonstrated that dDAAM shows an interaction with *Ena and profilin*. Moreover, we identified Rac as the most likely activator of dDAAM in the developing nervous system. (Matusek et al. 2008).

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Supervisor: József Mihály
E-mail: gombri@brc.hu