oxidase activity and abscisic acid accumulation, and increases Na^+ content in leaves without toxicity symptoms in Solanum Lycopersicum L. J Plant Physiol 166(9):914-925.
Vernooij B, Friedrich L, Morse A, Reist R, Kolditz-Jawhar R, Ward E, Uknes S, Kessmann H, Ryals J (1994) Salicylic acid is not the translocated signal responsible for inducing systemic acquired resistance but is required in signal transduction. Plant Cell 6:959-965.

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).

Goode BL, Eck MJ (2007) Mechanism and function of formins in the control of actin assembly. Annu Rev Biochem 76:593-627.
Matusek T, Gombos R, Szécsényi A, Sánchez-Soriano N, Czibula A, Pataki C, Gedai A, Prokop A, Raskó I, Mihály J (2008) Formin proteins of the DAAM subfamily play a role during axon growth. J Neurosci 28(49):13310-13319.
Pak CW, Flynn KC, Bamburg JR (2008) Actin-binding proteins take the reins in growth cones. Nat Rev Neurosci 9(2):136-147.

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