fungus was sprayed directly onto a bunch of ears in full flowering stage. Afterwards 48 hours polyethylene bag coverage was maintained to allow moisture condition for growing fungus. Head blight symptoms were evaluated visually as the percentage of scabby spikelets from 10 days after inoculation (dai) on every 4th day till the 26 dai. Inoculated ears are harvested and trashed so as to evaluate the percentage of Fusarium damaged kernels (FDK) and deoxynivalenol content (DON) by HPLC. Flowering time, plant height, ear type, present of awns, thousand kernel weight (TKW) was evaluated also during the experiment. Analysis of the data was made by two-way ANOVA and Pearson's-correlation.

Genotyping was carried out with microsatellite (SSR) markers, linked to FHB resistance QTLs, collected from literature. At the present time four QTL regions, located on the 2D, 3BS, 5A and the 6B chromosomes was mapped with 40 SSR markers. Since in resistant genotypes Sumai3 alleles dominated the tested regions, alleles were scored as derived from Sumai3 and others non-Sumai3. Linkage analysis and QTL mapping were done with JoinMap and MapQTL. Reduced hegiht (Rht) genes Rht-B1b, Rht-D1b and Rht8 were also detected.

Significant (P = 0,1%) positive correlations were found between FHB, FDK, DON, and TKW data. Plant height was in significant (P = 10%) negative correlation with disease traits. This means that taller plant and plants with lower TKW had lower disease level. Highly effective QTL, originated from Sumai3, found on the 3BS chromosome, explained the 60% of the phenotypic variance (p.v.) of FHB with 21 LOD value and the 45% of p.v. of FDK with 14 LOD value. It is flanked by 3 SSR markers on a 3 cM distance. A Sumai3 originated medium effective QTL found on the 5A (LOD 6-8), and small or medium effective QTL on 2D (LOD 3-7) and 6B (LOD 4-6) chromosomes. The mapped regions had significant effect on TKW also. Sumai3 alleles are linked to lower TKW mainly on 5A and 6B chromosomes which is a negative effect in breeding. The distribution of QTLs according to head type showed significant differences. The ratio of QTL carrying genotypes in the group of tapered and spindle headed plants was larger than in the group of square or semi-butt headed plants.

Fusarium resistance QTLs are identified and their effectiveness and agronomic relation are investigated in this study. However this research program wasn't finished, members of this population with good agronomic characters and Fusarium resistance (supported by known QTLs) are able to start to do marker assisted selection.

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Supervisor: Ákos Mesterházy
E-mail: lehoczkisz@gabonakutato.hu

The role of the *Drosophila* DAAM in the development of the Indirect Flight Muscle

Imre Molnár

Biological Research Center, Department of Genetics, Szeged, Hungary

In the past few years our group has working on to study of a new *Drosophila* formin protein called DAAM (Dishevelled associated activator of morphogenesis). This protein has many interesting tissue-specific functions. For instance, it regulates the tracheal cuticle pattern (Matusek et al. 2006), plays a role during axon growth (Matusek and Gombos et al. 2008), and participates in the process of genitalia rotation.

In collaboration with a research group from the University of York (UK) we showed that the *Drosophila* DAAM (dDAAM) is localized in the Indirect Flight Muscle (IFM) of both the pupae and adult flies.

In non-muscle cells, generally there are two major actin nucleating-polymerizing systems, the formins and the Arp2/3 complex. Formins are producing long straight actin filaments and the Arp2/3 complex is producing branched actin network (Pollard 2007; Chhabra and Higgs 2007).

Because unbranched straight actin filament is the major form in striated muscle cells, it is possible that a formin family protein serves as the key regulator of actin dynamics in myofibrils (Taniguchi et al. 2009). These straight actin filaments are organized into the contractile unit called sarcomere but little is known about the regulation of actin assembly in muscle cells.

Our aim was to reveal the function of *Drosophila* DAAM insight the striated muscle's sarcomere.

With the use of different approaches, like muscle functional tests, immunohistochemistry, biophysical essays, we confirmed that dDAAM plays role in the nucleation of actin filaments and sarcomere assembly.

In the 6% of the dDAAM loss of function hypomorph mutants, called Ex1, about 16% reduction in the sarcomere length was observed compared to the wild type. These flightless mutants carrying the full length protein construct could rescue the phenotype at 100% both in sarcomere level and functionally as well.

With the overexpression of the tagged full length protein, we found that it is partially localized in the expected region, namely in the M-line of the sarcomere.

The RNA interference-mediated depletion of dDAAM resulted in a marked reduction in sarcomere length and disruption of the sarcomeric structure.

These findings suggested that actin dynamics regulated by dDAAM are critical for sarcomere organization in striated muscle cells.

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

Some neuroprotective approaches in focal and global ischaemia on *in vivo* and *in vitro* rat models

Dávid Nagy

Department of Physiology, Anatomy and Neuroscience, University of Szeged, Szeged, Hungary

Glutamate (Glu) is the major excitatory amino acid neurotransmitter in the central nervous system. It mediates a number of physiological processes, but it is involved in the pathological processes of excitotoxicity too. Traumatic brain injury, focal brain lesion or global hypoperfusion are followed by acute excitotoxicity caused by the presence of abnormally high Glu levels in the cerebrospinal and interstitial fluids.

It has recently been demonstrated that this excess Glu in the brain can be eliminated by the intravenous administration of oxaloacetate (OxAc), which, by scavenging the blood Glu, induces an enhanced and neuroprotective brain-to-blood Glu efflux.

In this study, we subjected rats to a photothrombotic lesion and treated them after the illumination with a single 30-min long administration of OxAc (1.2 mg/100 g, i.v.). Following induction of the lesion, we measured the infarct size by Fluoro-Jade B (FJB)-staining. FJB binds sensitively and specifically to damaged neurons, with increased contrast during acute neuronal stress. Coronal sections (30μm) were cut with a freezing microtome and the sections were stained with FJB. The sections were subsequently analyzed with a fluorescent microscope. The volume of the hemispheric lesion and the number of FJB-positive cells were calculated for each animal. The administration of OxAc resulted in a reduction in the volume of the ischemia-induced cortical damage.

We also examined the functional consequences of the photothrombotic lesion by measuring the amplitudes of the somatosensory evoked potentials (SEPs). SEPs were induced in the contralateral primary somatosensory cortex by electrical stimulation of the right whisker pad and were transcranially recorded. The photothrombotic lesion resulted in appreciably decreased amplitudes of SEPs, but OxAc administration significantly attenuated this reduction.

We suggest that the neuroprotective effects of OxAc are due to its blood Glu scavenging activity, which, by increasing the brain-to-blood Glu efflux, reduces the excess Glu in the brain. This limits the size of the penumbra, improves the tissue perfusion and oxygenation and reduces the ischemia-related functional damage.

Ischemic postconditioning is referred to preventing ischaemia/reperfusion injury in both myocardial and cerebral infarction. The next study was undertaken to evaluate possible neuroprotective effects of kainate postconditioning against delayed neuronal death in hippocampal CA1 neurons if applied two days after hypoperfusion.

Transient global hypoperfusion was induced in male Wistar rats by two-vessel occlusion (2VO) for 30 min. 2VO causes inhibition of protein synthesis in selectively vulnerable brain regions such as CA1 and leads to the decrease of dendritic spine number and resulted in an impaired long-term potentiation (LTP) function in the hippocampal CA1 region.

In order to determine the number of apical dendritic spines we used Golgi-Cox staining. When the impregnation was ready coronal brain sections were cut by vibratome. The clear Golgi sections have been evaluated by light microscopical stereology.

For electrophysiological recordings we prepared coronal slices from the middle part of hippocampi. Field excitatory postsynaptic potencials (fEPSPs) were monitored and after a control period, LTP of the Schaffer collateral-CA1 synaptic response was induced by high-frequency stimulation (HFS). After the HFS the fEPSPs were recorded for at least a further 60 min-long period. If we apply the kainate (5 mg/kg) 48 hours after the 2VO, the loss of hippocampal dendritic spines and dysfunction of LTP could be significantly averted.

These results suggest that a sublethal second post-ischaemic event can be considered as a trigger for the start of protein synthesis activity in post-ischaemic cells. Postconditioning probably causes a re-modulation of protective protein (hsp70, hsp72, Bcl-2) synthesis leading to a switch from pro-apoptotic to anti-apoptotic pathways.

Supervisor: Tamás Farkas, Zsolt Kis
E-mail: david.nagy01@gmail.com