agonist glycine site, and has long been at the focus of neuroprotective trials. Unfortunately, KYNA is barely able to cross the blood-brain barrier. Accordingly, the development and synthesis of KYNA analogs which can readily cross the BBB have been at the focus of research interest with the aim of neuroprotection.

A novel KYNA analog, 2-(2-N,N-dimethylaminoethylamine-1-carbonyl)-1H-quinolin-4-one hydrochloride (Patent Application No: 104448-1998/Ky/me), recently proved to be neuroactive in several experimental paradigms. The analog effectively reduced c-fos and nNOS activation in an experimental animal model of migraine, effects interpreted as due to NMDA blockade. Moreover, in an *in vitro* comparative electrophysiological study, this compound was found to have the same neuromodulatory attributes as KYNA. NMDA antagonism was also acknowledged. 1 mmol of the analog administered i.p. effectively reduces the amplitudes of hippocampal population spikes. Regarding these properties, we estimated the neuroprotective capability of a novel kynurenic acid analog in transient global forebrain ischemia, measuring the rate of hippocampal CA1 pyramidal cell loss and the preservation of long-term potentiation at Schaffer collateral-CA1 synapses.

The neuroprotective potential was reflected by a significantly diminished hippocampal CA1 cell loss and preserved long-term potentiation expression. The neuroprotective effect was robust in the event of pretreatment, and also when the drug was administered at the time of reperfusion.

A detailed analysis of the behavioral effects of this new compound appeared to be extremely important, and we have therefore investigated it from several aspects.

In a preliminary investigation of the effects of the analog on mice, we performed open-field tests of the locomotor activity and exploratory drive. The influence of the analog on spatial orientation and learning was also assessed in the radial arm maze imprinting test. In the Morris water maze tests we examined its effects on the working memory and long-lasting reference memory of rats.

It emerged that there is a dose of this KYNA-amide which is neuroprotective, but does not worsen the cognitive function of the brain. This result is significant in that a putative neuroprotectant without adverse cognitive side-effects is of great benefit.

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Developmental regulation of brassinosteroid distribution in *Arabidopsis*

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Brassinosteroids (BRs; steroidal phytohormones) are essential regulators of plant growth and development. Unlike most other hormones, BRs are not subject to active transport, but exert their effects locally, in a paracrine manner. Local BR levels are efficiently controlled by the coordinated actions of biosynthetic and degradative gene functions, which ensure both homeostatic and differential regulation. While the transcriptional regulation of BR biosynthetic genes is known in great detail, its direct effects on the hormone production and accumulation are still to be clarified.

The aim of our study is to find out how castasterone and brassionolide, the two biologically active forms of BRs, are distributed in the model plant *Arabidopsis thaliana*. To observe developmental changes in the hormone accumulation, we generated transgenic plants expressing reporter genes under the control of an artificial BR-responsive promoter. The BR response constructs will be used for monitoring developmental BR adjustments during morphogenic events, such as germination and the differentiation of reproductive organs. Parallelly, we determine the bioactive BRs in all *Arabidopsis* organs via CG-MS analyses, in order to construct a comprehensive map of hormone distribution in the adult plant. In another approach, we initiated studies on the role of regulated hormone distribution during embryonic development. This line of research utilizes GFP and LUC reporter-tagged versions of the CYP85A2 enzyme that catalyzes the rate-limiting step of BR biosynthesis. The transgenic lines expressing these chimeric proteins will be helpful in elucidating the induction and spatial pattern of embryonic BR synthesis, and its correlation with the developmental auxin re-distribution that has been well characterized.

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The examination of the telomer protecting *Drosophila melanogaster* gene (*dtl*)

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In *Drosophila melanogaster* chromosome ends consist of retrotransposon arrays, the well-defined, short telomeric repeats, characteristic of human and other telomerase-containing organisms are absent. Consequently, in Drosophila there is no need for the sequence-specific,