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taxonomical groups were selected, and used for the molecular studies. All species used were documented with light and scanning electron microscope images. Distance and likelihood based methods were used in combination with our own and GenBank sequences to determine the genetic similarity between oribatid mites at family level.

Our results suggest that the similarity of 28S ribosomal DNA sequences support the monophyly of lower, but not higher oribatid groups. Cytochrome-oxidase (Cox1) sequences are known to be useful at taxonomical level of genera. We introduced Cox1 sequence in the identification of larva stages.

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Kynurenines: neuroactive compounds in the central nervous system: An *in vitro* study

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Kynurenic acid (KYNA) is a neuroprotective endogenous tryptophan metabolite produced by astrocytes and neurons via the kynurenine pathway in both humans and rodents. At non-physiological concentrations, KYNA is an excitatory amino acid receptor antagonist that can partially act at both the α-amino-3-hydroxy-5-methylisoxazole-4-propionic acid and N-methyl-D-aspartate subunits of the glutamate receptors (Stone 1993).

In the brain, KYNA is synthesized in astrocytes from its bioprecursor L-kynurenine (KYN) and is then rapidly released into the extracellular compartment. Previous studies have indicated that rat cortical slices have also the ability to synthesize KYNA from exogenously added KYN (Turski et al. 1989). The synthesis of KYNA from KYN is catalysed by kynurenine aminotransferase I and II (KAT I and II) (Schwarcz et al. 2002).

The use of KYNA as a neuroprotective agent is rather restricted, however, because KYNA has only a very limited ability to cross the blood–brain barrier (Fukui et al. 1991). In contrast, KYN and different synthetic KYNA derivatives cross this barrier more readily (Giles et al. 2003).

In the course of the experiments on rat brain slices, the Schaffer collaterals were stimulated and field excitatory postsynaptic potentials (fEPSPs) were recorded in the pyramidal layer of the hippocampal CA1 region. To test the effects of KYNA, we used an *in vitro* pentylenetetrazole (PTZ) model. PTZ, a chemical convulsant frequently utilized in the study of seizures (Yudkoff et al. 2006), exerts its effects by binding to the picrotoxin binding site of the post-synaptic GABA-A receptor (Macdonald et al. 1977). PTZ administered *in vitro* at 1mM induced a considerable increase in the amplitude of the fEPSPs recorded from the hippocampal CA1 region. When applied locally in an extremely high concentration (20 mM), PTZ resulted in characteristic wavelets. However, KYNA administration not only decreased the amplitude of the hippocampal CA1 responses evoked by Schaffer collateral stimulation, but also afforded protection from the PTZ-induced response enhancement.

The KYNA precursor KYN also blocked the development of the PTZ-induced high increase in amplitude. To prove that the KYN→KYNA conversion did take place in our experiments and that it was KYNA which afforded the protection against the effects of PTZ, we applied *N*-omega-nitro-l-arginine, an inhibitor of KAT I and II (Rozsa et al. 2008).

SZR-72, a synthetic kynurenic acid derivative, applied *in vitro*, prooved to be also effective in preventing the high increase in fEPSP amplitudes, generated by PTZ.

These findings show that treatment with KYN, or the synthetic kynurenic acid derivative, SZR-72, even at very low concentration, has an effect on enhanced neural excitability and thus support the hypothesis that manipulations of the kynurenine pathway might be a rewarding target in different neuronal disorders affected by neuronal hyperexcitation.

Additionally we have shown, that KYNA in submicromolar concentration range has a positive neuromodulatory effect. In nM concentrations, kynurenic acid does not give rise to inhibition, but in fact facilitates the field excitatory postsynaptic potentials, recorded from the hippocampal CA1 region (Rozsa et al. 2008).

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Examination of the hydrogen-metabolism in *Methylococcus capsulatus* (Bath)

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Methylococcus capsulatus (Bath) is a Gram-negative, methylotrophic bacterium, which oxidizes methane to carbon dioxide for energy generation. The enzyme complexes methane monooxygenases (MMOs) oxidize methane to methanol and co-oxidize a wide variety of aliphatic, aromatic and halogenated hydrocarbons, therefore they are extremely versatile enzymes for biocatalysis and bioremediation. The in vivo electron donor of the MMOs is NADH, which must be regenerated. Since biodegradation processes using MMO are co-oxidation processes, alternative ways of supplying reducing power are needed. Possible candidate could be H_2 for NADH + H^+ generation.

Hydrogenases are metalloenzymes catalyzing the reversible oxidation of H_2. *M. capsulatus* (Bath) contains a soluble (Hox) hydrogenase - which is able to reduce NAD^+ using H_2 - , and a membrane-bound nickel-iron Hup hydrogenase - which plays an important role in the recycling of hydrogen, and maybe donates the electrons to the quinone pool. Another enzyme – nitrogenase - produces H_2 as a byproduct under nitrogen fixing condition.

Δ*hupSL* and Δ*hoxH* deletion mutants were generated (Csáki et al. 2001). H_2-driven MMO activities of these mutants and wild type were measured to obtain information about the *in vivo* function of the hydrogenases (Hanczár et al. 2002). The deletion mutants revealed unexpected behavior: the Δ*hupSL* mutant did not show H_2-driven MMO activity, while the Δ*hoxH* mutant showed. The Hup hydrogenase - which is unable to reduce NAD^+ directly - is required for the H_2-driven activity of MMO. To understand the role of Hup hydrogenase in H_2-metabolism the first step is to find all genes coding for proteins, which has any affect on Hup hydrogenase activity.

Several Hup^- phenotype mutants were isolated from a *M. capsulatus* random mutant library, which was generated by transposon mutagenesis. The transposon was found in a structural gene (*hupL*), in an accessory gene (*hupD*) of Hup hydrogenase, and in other genes: TonB-dependent receptor-like putative protein coding gene (*tonB*) and conserved hypothetical protein for NADH ubiquinone/plastoquinone complex coding gene (*nupX*).

The *in vivo* H_2 production capacities of the wild type and the mutant strains were compared. The Hup hydrogenase of the wild type consumed a lot of H_2 from the gas phase, while the mutants had lower H_2 consumption activity both under nitrogen fixing and nitrogenase repressed conditions. Hup hydrogenase structural proteins were detected both in wild type, $HupD^-$, $TonB^-$ and $NupX^-$ transposon mutants with HupL antibody by Western Blot assay, in contrast to the Δ*hupSL* deletion and $HulpL^-$ transposon mutants.

The results show the presence of the matured Hup hydrogenase in $TonB^-$ and $NupX^-$ transposon mutants, but hydrogen-metabolism of these mutants is damaged, therefore they have Hup^- phenotype. According to the *in silico* analysis and global protein alignment the proteins of the *nupX* containing operon are similar to the NuoM, NuoL and NuoN proteins of the NUO (NADH ubiquinon-plastoquinon oxydoreductase) complex. In our hypothesis the proteins of the examined nuo-like operon maybe play a role in the energy conversion of the bacterium, while the examined TonB-dependent receptor-like putative protein perhaps takes part in the mechanism of TonB-catalyzed iron transport through the bacterial cell envelope, indirectly contributing to the assembly of the membrane-bound nickel-iron Hup hydrogenase.

To determine the role of the mutant genes further investigations are needed.

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