Cystathionine beta synthase

Cystathionine β-synthase (CBS) is involved in homocysteine and hydrogen sulfide (H2S) metabolism. Both products have been implicated in the pathophysiology of cerebrovascular diseases.

Cystathionine beta synthase (CBS) catalyzes the condensation of serine with homocysteine to form cystathionine and occupies a crucial regulatory position between the methionine cycle and the biosynthesis of cysteine by transsulfuration. It was reported that CBS was a novel marker of both differentiation and proliferation for certain cell types, suggesting that CBS represents a survival-promoting protein. However, its expression and function in the central nervous system lesion are not well understood. To investigate changes of CBS after traumatic brain injury (TBI) and its possible role, mice TBI model was established by controlled cortical impact system, and the expression and cellular localization of CBS after TBI was investigated in the present study. Western blot analysis revealed that CBS was present in normal mice brain cortex. It gradually decreased, reached a valley at the third day after TBI, and then restored to basal level. Importantly, more CBS was colocalized with neuron. In addition, Western blot detection showed that the third day postinjury was also the apoptosis peak indicated by the elevated expression of caspase-3. Importantly, immunohistochemistry analysis revealed that injury-induced expression of CBS was colabeled by Bcl-2 and had no co-localization with caspase-3. These data suggested that CBS may be implicated in the apoptosis of neuron and involved in the pathophysiology of brain after TBI [1)].

The insertion allele of the 844ins68 CBS insertion polymorphism was independently associated with aSAH while the GG genotype of rs234706 was associated with an unfavorable outcome both at discharge and last follow-up. Increased CBS activity may exert its neuroprotective effects through alteration of H2S levels, and independent of clinical vasospasm and DCI [2)].

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2)
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