Editorial:

N-acetylaspartic acid monitors oxidative stress

Sankar Surendran

1 UNT Health Science center, Fort Worth, Texas, USA; Email: sankar_surendran@yahoo.com; Tel: +1 504 568 5481

N-acetylaspartic acid (NAA) was discovered by Tallan et al., in 1956. It is synthesized from acetyl coenzyme A and aspartate by a mitochondrial enzyme, L-aspartate N-acetyltransferase (Goldstein, 1969). NAA is mainly found in the gray matter of the brain and also present at lower levels in the astroglia, white matter, superior cervical ganglion, splenic nerve, peripheral nervous tissue of spleen, lung, liver, kidney, muscle, ovary, thymus, stomach, heart, adrenal medulla and retina of fishes to mammals (see review, Surendran et al., 2011). Normal level of NAA is important in the maintenance of potential antioxidants. N-acetylaspartic acid level is altered in many diseases including alcoholic brain (Schweinsburg et al., 2001), brain oedema (Demougeot et al., 2001), HIV-related dementia (Meyerhoff et al., 1993; Sacktor et al., 2005), HIV positive alcoholism (Pfefferbaum et al., 2005), Canavan disease (see review, Surendran et al., 2011), Parkinson’s disease (Surendran and Rajasankar, 2010), type 2 diabetes (Surendran et al., 2006) and spinocerebellar ataxia type 1 (Oz et al., 2010). Altered levels of NAA changes nitric oxide and potential antioxidant levels to cause disease pathophysiology (Surendran, 2009; Surendran and Rajasankar, 2010), suggesting NAA monitors oxidative stress by regulating antioxidant levels.

Aspartoacylase deacetylates N-acetylaspartic acid into aspartate and acetate (Birnbaum et al., 1952). While aspartoacylase activity is very mild or no activity in normal astrocytes, the activity is increased in inflammatory conditions suggesting aspartoacylase contribution in reactive astrocytes (Surendran, 2007; Surendran et al., 2011). In Table 1, the key message of recently published studies on NAA effect on oxidative stress has been summarized.

Table 1: Studies in N-acetylaspartic acid (NAA) resulting oxidative stress

<table>
<thead>
<tr>
<th>Key message</th>
<th>Reference</th>
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<tr>
<td>NAA induced nitric oxide toxicity and alters proteins associated with inflammation, transcription and contractility to cause pathophysiology</td>
<td>Surendran, 2009</td>
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<tr>
<td>NAA induced nitric oxide toxicity contribute to neurodegeneration</td>
<td>Surendran, 2008</td>
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<td>NAA induced nitric oxide toxicity to cause Canavan disease pathophysiology</td>
<td>Surendran, 2010</td>
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<td>Altered levels of nitric oxide cause contractile abnormality</td>
<td>Surendran and Kondapaka, 2005</td>
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<td>NAA contributes in Parkinson’s disease</td>
<td>Surendran and Rajasankar, 2010</td>
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<td>NAA induced oxidative stress to contribute in disease pathophysiology</td>
<td>Surendran and Bhatnagar, 2011</td>
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<tr>
<td>NAA induced nitric oxide toxicity contributes in Canavan disease pathophysiology</td>
<td>Surendran et al., 2011</td>
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<td>NAA reduced glucose 6-phosphate dehydrogenase and enhanced protein carbonyl content and superoxide dismutase</td>
<td>Pederzolli et al., 2009</td>
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<td>NAA reduced catalase and glutathione peroxidase and induced hydrogen peroxide</td>
<td>Pederzolli et al., 2010</td>
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References


