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Editorial:

N-acetylaspartic acid monitors oxidative stress

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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

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

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