Toxicity and oxidative stress of acrylonitrile in rat primary glial cells: Preventive effects of N-acetylcysteine

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Received 7 February 2007; received in revised form 3 May 2007; accepted 4 May 2007
Available online 18 May 2007

Abstract

Brain is a target organ for acrylonitrile (ACN) toxicity. The objective of the current work was to investigate ACN cytotoxicity in rat primary glial cells, using N-acetyl-l-cysteine (NAC) as a potential protective agent. Cells were exposed in vitro to different concentrations of ACN for different time intervals. Cell membrane integrity was assessed by trypan blue exclusion and lactate dehydrogenase (LDH) leakage. Approximately 50% membrane damage was observed in the incubations containing 1.0 mM ACN for 3 h. Therefore, these experimental conditions were used in subsequent studies. ACN enhanced lipid peroxidation, as indicated by malondialdehyde (MDA) accumulation, and depleted reduced glutathione (GSH) level with no change in total glutathione. Also, ACN was activated to cyanide (CN\textsuperscript{-}) with dramatic decrease in ATP level. Cell treatment with NAC prior to exposure to ACN afforded some protection, as indicated by reducing MDA level and elevating level of both reduced and total glutathione. Further, pretreatment with NAC inhibited CN\textsuperscript{-} formation and caused an increase in ATP level. Our results indicate that ACN is toxic to rat primary glial cells as evidenced by induction of oxidative stress and generation of CN\textsuperscript{-} with subsequent energy depletion. NAC can play an important role against ACN-induced oxidative damage.

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Keywords: Acrylonitrile; N-Acetyl-l-cysteine; Oxidative stress; Glial cells

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0378-4274/\$ – see front matter © 2007 Elsevier Ireland Ltd. All rights reserved.
doi:10.1016/j.toxlet.2007.05.001