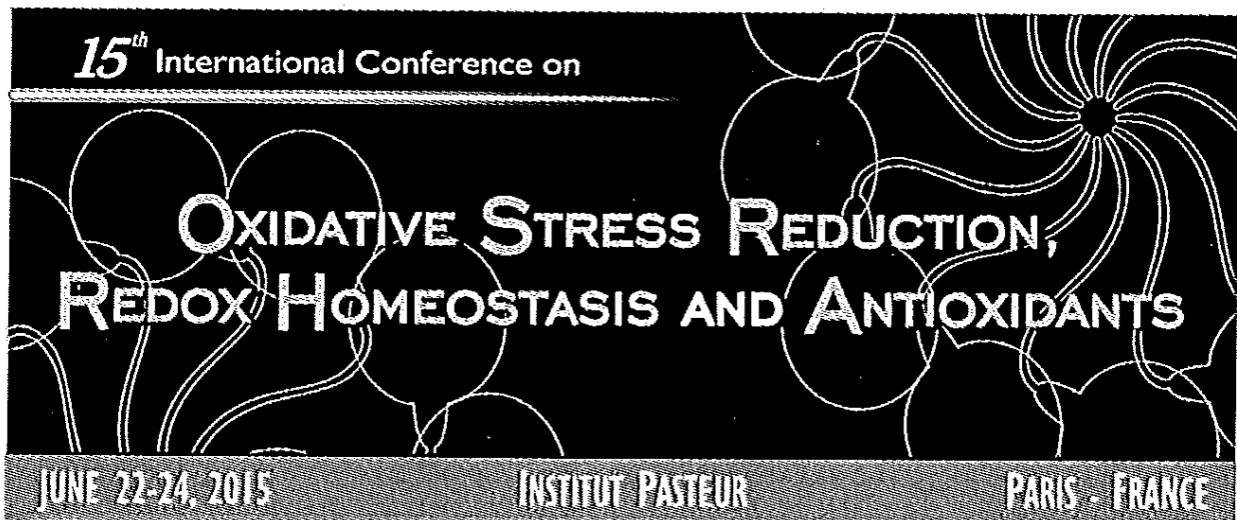


# *International Society of Antioxidants*

15<sup>th</sup> ISANH Conference on

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## Abstracts Book



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## ETHANOL-INDUCED OXIDATIVE STRESS IN MOUSE BRAIN: ROLE OF ACETALDEHYDE

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Introduction: Ethanol, one of the most widely consumed substances of abuse, can induce brain damage and neurodegeneration, although the particular mechanism of action at a central level has not been described in detail yet. Ethanol is centrally metabolized into acetaldehyde, which has been shown to be responsible for some of the neurophysiological and cellular effects elicited by ethanol (1). Moreover, centrally formed acetaldehyde seems to play a key role in the burst of ROS levels observed after acute ethanol administration (2). However, the particular mechanism by which ethanol or acetaldehyde contribute to oxidative stress is still unclear. Objectives: To ascertain the role of acetaldehyde on ethanol-induced generation of ROS in the brain. Methodology: Swiss mice were pretreated with the acetaldehyde scavenging agents D-penicillamine (75 mg/kg, i.p.) and alpha lipoic acid (100 mg/kg, i.p) at different times before ethanol (2.5 g/kg, i.p.) administration. Brains were immediately removed and glutathione peroxidase (GPx) activity and protein carbonyl groups (PCGs) were determined. Results: Ethanol administration increased the PCGs and enhanced GPx activity in a dose fashion. Pretreatment with these two acetaldehyde scavengers protected against these ethanol effects. Conclusions: These data point out the key role of acetaldehyde on the generation of brain ROS.

1. Tarragon E, Baliño P, Aragon CM. Centrally formed acetaldehyde mediates ethanol-induced brain PKA activation. *Neurosci Lett*. 2014 Sep 19;580:68-73.

2. Ledesma JC, Baliño P, Aragon CM. Reduction in central H<sub>2</sub>O<sub>2</sub> levels prevents voluntary ethanol intake in mice: a role for the brain catalase-H<sub>2</sub>O<sub>2</sub> system in alcohol binge drinking. *Alcohol Clin Exp Res*. 2014 Jan;38(1):60-7.