Journal of Clinical Respiratory Open Access

Extended Abstract
Vol. 1, Iss. 2
2019

COPD 2016: Effects of hyperoxia exposure on free radicals accumulation in relation to ultrastructural pathological changes of diaphragm_Al-Said A Haffor_Dar Al Uloom University, KSA

Al-Said A Haffor

Dar Al Uloom University, KSA

COPD are related with an expanded burden on the stomach prompting gathering of responsive oxygen species (ROS) and the resulting cell harms and passing. Th e neurotic adjustments enlisted by ROS in the stomach during oxygen breathing are not known. Th e reason for the current examination was to analyze the impacts of hyperoxia presentation (HP) on free radicals (FR) aggregation corresponding to the ultra basic neurotic changes in the stomach. Twenty grown-up male rodents were haphazardly doled out to two gatherings; control (C) and hyperoxia (HP). Creatures of the HP were breathing 100% O2 for 72 hours consistently.

Both serum and stomach tissue supernatant investigation indicated essentially higher ($p<0.05$) FR in HP bunch as contrasted and control gathering. Ultra-structure assessments indicated that HP brought about assortment of neurotic adjustments in the mitochondria and endoplasmic reticulum that were related with disarrangement of myofibrils, loss of I-banding for myosin, central myolysis of the myofilaments, complete discontinuity of myosin, tearing of myofilaments from Z plates and tearing of the endothelial cell of the interstitial blood vessels. In view of the consequences of the current investigation, it very well may be presumed that hyperoxia initiated speeding up ROS development harmed the contractile devices of the stomach and related endomembrane proteins that could include intracellular calcium channels proteins

Discussion

Hyperoxia is accepted to create receptive oxygen species (ROS) and restrain cell reinforcements safeguard. O2 harmfulness is accepted to happen when the body's cancer prevention agent guards are overpowered by expanded creation of ROS. Remembered for ROS list are superoxide, hydrogen peroxide, hydroxyl radicals, and peroxynitrite at elevated levels of PtiO2 (Demchenko et al., 2001; Demchenko et al., 2003; Torbati et al., 1992). In this, the current investigation indicated that natural cell reinforcement potential (BAP) in the lungs was overpowered following 48 h of ceaseless hyperoxia presentation.

Plainly useless mitochondria bring about discharging its substance, for example, oxidative compounds and hydrogen peroxides to the cytoplasm, in endeavor to forestall growing. At the point when the pace of arrival of mitochondrial substance surpasses end rate by cell reinforcements resistance framework, ROS gather and FR creations rise. Past work demonstrated that hyperoxia drafted assortment of obsessive changes in the inward mitochondrial layer (Haffor, 2004) that gave basic mitochondrial occasions mindful to oxidative pressure interceded cell passing known as harmful oxidative pressure (TOS). Thus the current examination demonstrated that hyperoxia presentation for 48 h brought about an expansion in free radicals creation related with decrease in natural cancer prevention agent potential (BAP).

The seriousness of hyperoxic-actuated cell injury is time-and portion subordinate (Hayatdavoudi et al., 1981; Barry and Crapo, 1985; Crapo et al., 1994). The impacts of hyperoxia and related danger of bronchopulmonary dysplasia in newborn children or in grown-up respiratory pain condition (ARDS) in grown-ups starts with presentation period more than 8 h In sound grown-up hazard starts after 48 h (Comroe et al., 1945). Be that as it may, intense introduction to hyperbaric-hyperoxia causes tissue and cell harms in the mind as ahead of schedule as 16 h of presentation (Huang et al., 2000; Gerstner et al., 2006). Other than lung epithelial ROS age, fine endothelial cells were recognized as the wellspring of hyperoxia-incited ROS creation. In this, aftereffects of the current examination indicated a consistent ascent in ROS age, past the beginning of lung tissue injury as announced in these investigations, which mirrored extra phagocytes barrier system of the alveolar macrophages which thus contributed additively to ROS age.

Biography

Al-Said Haffor obtained his PhD (1985) degree in Applied Physiology from the Ohio State University, Columbus, OH, USA. Began his career as an Assistant Professor of Applied physiology at Mississippi State University, USA. Three years later being Promoted to Associate Professor of clinical epidemiology at MSQA, CSU-DH, besides being a physiologist and supervisor in Internal Medicine and Critical, in the Pulmonary Physiology Department at LAC+USC Medical Center (1987 - 2000). Since 2001 he has been Professor of Physiology in King Saud University (in Riyadh, Alkharj & Wadi Aldwasir) and Dar AlUloom University, College of Medicine (2014 - present). His research had been funded by SABEC, as well as by the Deanships for Research & Graduate studies at King Saud University and Salman Bin Abdul Aziz University. He conducted more than 30 presentations in regional and international conferences, while in the USA and KSA. He published more than 40 Original Articles and Reviews in peer reviewed journals.

saidhaffor@yahoo.com

This abstract is partly presented at 3rd International Conference on Chronic Obstructive Pulmonary Disease on July 11-12, 2016 held at Brisbane, Australia