Aim of the study

The aim of this study was to assess preventive/therapeutic efficacy of CORM-A1 against experimentally induced Nonalcoholic steatohepatitis.

Objectives

1. ***To assess overall improvement in survival rate of acute hepatotoxic mice by CORM-A1 treatment.***

   Lethal dose of acetaminophen (APAP) was employed and possible improvement in survival rate in CORM-A1 treated group was studied herein.

2. ***To decipher the underlying mechanism of CORM-A1 mediated activation of Nrf2-ARE genes in experimentally induced acute hepatotoxicity.***

   t-BHP treated HepG2 cells and APAP treated swiss albino mice were used in this study as models of hepatotoxicity. Role of CORM-A1 in improving oxidative stress, inflammation and details of Nrf2-ARE pathway was studied herein.

3. ***To assess the potential of CORM-A1 in alleviating pathophysiology of NASH.***

   HepG2 and C57BL/6J mice were chosen as experimental models wherein, respective treatments with palmitic acid or high fructose high fat (HFHF) diet served as disease controls. Daily intraperitoneal injections of CORM-

A1 (from 9th to 16 weeks) were employed and possible improvement in inflammation, lipid profile, histopathology, liver function and expression of associated genes were studied herein.

4. ***To study the CORM-A1 mediated management of hepatic oxidative stress in NASH by Nrf2-ARE signaling.***

   In the above-mentioned experimental models, this objective focused on CORM-A1 mediated Nrf2 translocation and activation of ARE pathway leading to an improved cellular/organ antioxidant status studied through a series of carefully scripted protocols.

5. ***To decipher the role of CORM-A1 in improving mitochondrial biogenesis and function in NASH.***

   Role of CORM-A1 in improving mitochondrial respiration and biogenesis in palmitic acid treated t-BHP cells has been investigated in detail