J Clinic Toxicol 2016, 6:5(Suppl)
http://dx.doi.org/10.4172/2161-0495.C1.019

Effect of oral exposure to crude oil on the total body weight of male and female albino wistar rats

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Crude oil composition varies slightly by its source but contains many chemicals with toxic properties that are fairly in animals and human. Male and female albino rats weighing between 120kg and 200kg were grouped separately into test groups and control groups. Each group had five rats and was administered 60mg/kg body weight of crude oil orally for 28 days except for the control groups. The total body weights were taken at 7days interval for 28 days. Results showed a progressive decrease in total body weight of rats and a significant ($p<0.05$) decrease in average body weight of both male and female albino rats compared with respective control groups. Additionally, all test animals exhibited adverse reactions like itching watery eyes, sleepiness, shortening of breath and release of mucous. Also, there was a marked sign of dehydration and impaired digestion in week four of administration.

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Novel progress in the mechanism of phosgene-induced acute lung injury

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The present study was designed to investigate the effect of diosgenin (DSG) on metabolic dysfunction and to elucidate the possible molecular mechanisms. High fat (HF) diet-fed mice and 3T3-L1 preadipocytes was used to evaluate the effect of DSG. We showed that DSG attenuated metabolic dysfunction in HF diet-fed mice, as evidenced by reduction of blood glucose level and improvement of glucose and insulin intolerance. DSG ameliorated oxidative stress, reduced body weight, fat pads, and systematic lipid profiles and attenuated lipid accumulation. DSG inhibited 3T3-L1 adipocyte differentiation and reduced adipocyte size through regulating key factors. DSG inhibited PPARγ and its target gene expression both in differentiated 3T3-L1 adipocytes and fat tissues in HF diet-fed mice. Overexpression of PPARγ suppressed DSG-inhibited adipocyte differentiation. DSG significantly increased nuclear expression of ERβ. Inhibition of ERβ significantly suppressed DSG-exerted suppression of adipocyte differentiation and PPARγ expression. In response to DSG stimulation, ERβ bound with RXRα and dissociated RXRα from PPARγ, leading to the reduction of transcriptional activity of PPARγ. These data provide new insight into the mechanisms underlying the inhibitory effect of DSG on adipocyte differentiation and demonstrate that ERβ-exerted regulation of PPARγ expression and activity is critical for DSG-inhibited adipocyte differentiation.

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