Fat Embolization Syndrome (FES) occurs in many clinical settings such as long bone fractures, liposuction and CPB. Embolized fat is hydrolyzed by lipase into free fatty acids which has been shown to be toxic to the lung. Exposure of the lung to free fatty acids can cause the clinical picture of ARDS.

An animal model for FES has been developed utilizing intravenous triolein as a surrogate for FES exposure. We, therefore, hypothesized that intravenous triolein will produce histological changes that are similar to that seen in human FES.

Gross histopathological changes were seen in the lungs of the triolein group, although heart, liver, kidneys and spleen were normal and weight gain equaled the controls at 3 weeks.

Pulmonary histological examination revealed diffuse intra-alveolar hemorrhages and edema with peri-bronchial inflammation.

Vasculitis was more prominent in the peri-bronchial areas.

Collagen development in the vascular, perivascular, and bronchial regions was evident in both the 3 and 6 week groups treated with triolein.

Pulmonary arteries revealed significant medial thickening as compared with the control groups with arterial lumen patency significantly reduced in triolein groups vs controls (p=0.004).

Adventitia/media ratio was not statistically significant.

Pure triolein (0.2 mL) was injected into the caudal vein of unanaesthetized Sprague Dawley rats (n=13), while control animals received 0.2 mL of saline (n=12).

Weights were recorded until necropsy at 3 wks and 6 weeks.

Tissues were stained using H&E, trichrome, and Oil Red O.

Tissues of the lung were examined using 400x magnification.

Morphometric measurements were made on arterial vessels from both H&E and trichrome-stained lung tissues, triolein and control, at 3 and 6 weeks.

Arterial lumen patency = ratio of vessel lumen over external medial diameter

Media/adventitia ratio = outer medial diameter over outer adventitia diameter

Data were analyzed as a function of time and treatment (StatSoft, Tulsa Ok) using analysis of variance and post-tests or t-tests, with p<0.05 as statistically significant.

This study is a continuation of earlier work that showed severe pulmonary damage within 3-6 hours following triolein induced fat embolism in the rat model, reaching a peak at 96 hours post injection.

The present work shows that, despite recovery of general condition, body weight and partial reopening of the pulmonary arteries, collagen and vasculitis persisted up to 6 weeks.

Further studies are needed to verify the eventual recovery or organ evolution toward chronic fibrosis.