Abstract
Acute respiratory distress syndrome is a lethal condition of acute bilateral lung disease associated with trauma, sepsis, and shock that occurs as a result of fluid build up in the alveoli. Prior work in the field suggests that ARDS and its pathophysiology may be mediated by epigenetically controlled factors, such as DNA methylation, and that the syndrome itself may incur genome-wide DNA methylation changes.

In this investigation, we compare DNA methylation data from blood collected from 39 adults with ARDS, 75 ICU controls, and 30 healthy individuals made available by Szilagyi et al (1).

Our analysis reveals significant demethylation in the promoter regions of 7 histone deacetylase genes, suggesting up-regulation of HDAC transcription.

These results indicate that patients with ARDS have increased HDAC expression, potentially leading to closed chromatin structure and gene repression, suggesting a potential mechanism for increased HDAC expression in patients with ARDS.

Background
ARDS is a lethal condition of acute bilateral lung disease affecting 5% of adult patients on ventilators with mortality estimated at 40%.

Previous studies have demonstrated that ARDS pathophysiology is likely mediated, in part, by epigenetic mechanisms (2-4).

Histone deacetylases (HDACs) are known to regulate gene expression. In general, HDAC inhibitors cause an overall increase in gene expression.

HDAC inhibitors repress expression of inflammatory cytokines (5) and have been shown to attenuate lipopolysaccharide-induced acute lung injury in mice (6).