Obesity is a ubiquitous public health concern. Its effects on patients suffering from the acute respiratory distress syndrome (ARDS) have yet to be fully elucidated, although data from the NIH ARMA and ALVEOLI studies demonstrate that obesity may actually increase survival. These findings are surprising, especially given that obesity and ARDS are both systemic inflammatory conditions, yet obese patients with ARDS were shown to have *decreased* serum IL-6 and IL-8. We hypothesized that resistance to leptin (as develops in obesity) might in part explain obesity's 'protective' effect in ARDS. To elucidate the mechanism by which obesity and leptin-resistance might lead to an altered inflammatory response, we examined two mouse models of obesity: diet-induced obese (DIO) C57Bl/6 and leptin-resistant (DbDb) mice. Both groups (and controls) were exposed to nebulized lipopolysaccharide (LPS) to induce acute lung injury, and were subsequently euthanized 24h after exposure. Obese groups exhibited attenuated lung neutrophilia assessed by bronchoalveolar lavage. Next, we treated normal weight mice with pegylated leptin via aspiration prior to LPS exposure, and found increased lavage neutrophil counts at 24h. *In vitro* studies revealed a chemotactic effect of leptin on neutrophils isolated from lean mice, and this effect was attenuated in neutrophils of obese mice. Additionally, to see if leptin might mediate a change in cytokine release after LPS injury, we compared cytokines levels in bronchoalveolar lavage fluid from mice treated with LPS and leptin versus LPS/control-treated mice. This did not reveal any significant differences. Lastly, we looked at leptin's effects *in vivo* in the absence of LPS injury. Leptin treatment did not result in neutrophil changed counts or cytokine levels after 24h. These findings demonstrate leptin's effect on neutrophil trafficking and suggest that aberrant neutrophil functioning in the setting of leptin resistance may alter the pathophysiology of ARDS in obese patients.