Supplementary Data

Figure S1. Lipid extracts from EtOH + thermal burn injury-treated HaCaT cells does not affect IL-8 levels in PAF-R-negative KBM cells.

Figure S2. EtOH + thermal burn injury does not affect PAF-AH enzymatic activity in HaCaT cells.

Figure S3. TSI-01 pretreatment blocks augmentation of EtOH on PAF-R agonist formation in response to thermal burn injury in HaCaT cells.

Table SI Glycerophosphocholine species generated in response to EtOH or thermal burn alone, and EtOH plus thermal burn treatment of HaCaT cells.

Table SII. Direct incorporation of EtOH into PAF.

Table SIII. Ethanol + thermal burn injury modulates serum cytokine levels.

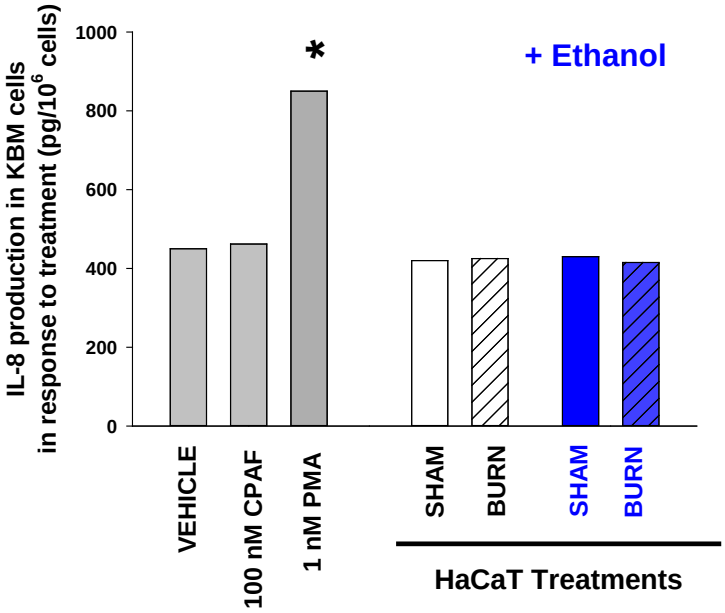

Figure S1. Lipid extracts from EtOH + thermal burn injury-treated HaCaT cells does not affect IL-8 levels in PAF-R-negative KBM cells.

HaCaT cells were subjected to 90^0C water bath x 2 min, or sham injury either under normal conditions or following a 30 min pre-incubation with 1% EtOH (BLUE). 5 min following injury treatment, the lipids were extracted. Lipid extracts from 5 x 10^6 HaCaT cells previously treated with thermal injury ± ethanol or 100nM CPAF, or 1 nM PMA as positive control were incubated with KBM cells and 6h later IL-8 measured in the supernatants. The data are the Mean ± SD IL-8 production in KBM cells (pg/10^6 KBM cells) from a single experiment representative of three separate experiments. *Denotes statistically significant (*P<0.05*) changes in levels of IL-8 from sham control values.

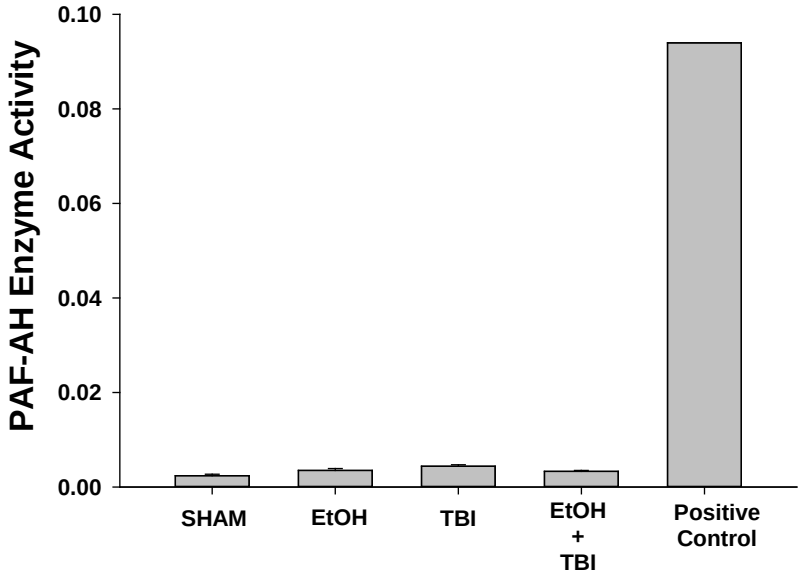

Figure S2. EtOH + thermal burn injury does not affect PAF-AH enzymatic activity in HaCaT cells. HaCaT cells were treated with 1% EtOH for 30 min. Cells were then subjected to thermal burn injury (TBI), and 5 min following injury the PAF-acetylhydrolase enzymatic activity was assessed. The data represent Mean ± SE PAF-AH activity (μmole/min/ml) from three separate experiments.

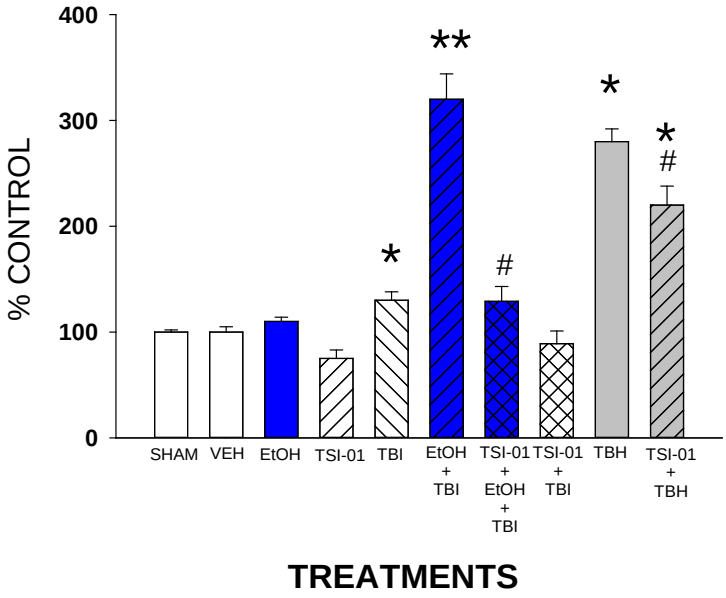

Figure S2. TSI-01 pretreatment blocks augmentation of EtOH on PAF-R agonist formation in response to thermal burn injury in HaCaT cells. HaCaT cells were untreated (SHAM), or pretreated with 60 μM TSI-01, or 0.1% DMSO vehicle (VEH) for 30 min before treatment with 1% EtOH (BLUE) for an additional 30 min. Cells were then subjected to thermal burn injury (TBI), or 100 μM tert-butyl hydroperoxide (TBH) and 5 min following injury the lipids were extracted and PAF-R agonistic activity determined as measurement of IL-8 released in KBP cells. The data are the Mean ± SD % control PAF-R agonists measured from IL-8 production in KBP cells (pg/10^6 KBP cells) from a single experiment representative of three separate experiments. ** Denotes statistically significant (P<0.01) changes in comparison to burn injury alone; *Denotes statistically significant (P<0.05) changes in comparison to SHAM treatment; # Denotes statistically significant (P<0.05) changes induced by TSI-01 treatment.