SUPPLEMENTAL DATA

Figure S1. HNE-induced ROS generation in mitochondria. THP-1 cells were labeled with mitochondrial staining dye, Mitotracker (200 nM) for 30 min, and then loaded with 5 µM of H2DCFDA for 5 min at 37 °C. The cells were then washed and treated with HNE (20 µM) for 20 min at 37°C. Following HNE treatment, cells were washed and analyzed for their fluorescence by confocal microscopy. The images shown here are merged images of mitochondrial (red) and ROS (green) staining. Orange and yellow colors indicate overlap between mitochondrial staining and ROS control.
Figure S2. HNE-induced increase in FVIIa activation of FX in THP-1: TF dependency. THP-1 cells ($2 \times 10^5$ cells/ml) were incubated with ± monospecific polyclonal anti-TF IgG (10 µg/ml) for 1 h, and then treated with a control vehicle or HNE (20 µM) for 4 h. Following the treatment, FVIIa (10 nM) and FX (175 nM) were added to the cells and FX activation was measured in a chromogenic assay. ***, p < 0.001.
Figure S3. HNE treatment does not alter TF antigen levels in THP-1 cells. THP-1 cells (2 \times 10^5 cells/ml) were treated with a control vehicle or HNE (20 µM) for 4 h. Following the treatment, cell lysates were made and subjected to western blot analysis to probe for TF.
**Figure S4. Effect of electron transport chain inhibitors on ATP production.** THP-1 cells were treated with various electron chain transport inhibitors for 1 h and then treated with a control vehicle or HNE (20 µM) for 4 h. Cellular ATP content was measured using ATP detection assay kit (Cayman Chemicals). The inhibitors used were: rotenone, 2.5 µM; THF, 0.5mM; antimycin, 10 µM; sodium azide, 10 mM; oligomycin, 5 µM. **, p < 0.01; ***, p <0.001 compared to their respective controls (no inhibitor).
Figure S5. The combined effects of the inhibition of ROS generation by oligomycin and p38 MAPK activation by SB203580 on HNE-induced TF decryption. THP-1 cells (2 x 10^5 cells/ml) were treated with a control vehicle, SB203580 (20 µM), oligomycin (5 µM) or SB203580 plus oligomycin for 1 h. After that, cells were stimulated with HNE (20 µM) for 4 h, and cell surface TF activity was measured in FX activation assay. *, p <0.05; **, p <0.01.
Figure S6. Effect of SB203580 on HNE-induced p38 MAPK activation in THP-1 cells. THP-1 cells (2 x 10^5 cell/ml) were treated with a control vehicle or SB 203580 (20 µM) for 1 h followed by HNE (20 µM) for 15 min. Cell lysates were subjected to western blot analysis and probed for phospho or total p38 MAPK. Densitometry analysis showed 70% reduction in HNE-induced phospho p38 MAPK levels (normalized to total MAPK) in cells treated with the SB compound prior to HNE treatment.
Figure S7. Inhibition of p38 MAPK activation blocks HNE-induced increase in cell surface phosphatidylserine levels. THP-1 cells (2 x 10^5 cells/ml) were treated with a control vehicle or p38 MAPK inhibitor SB 203850 (20 µM) for 1 h, followed by HNE (20 µM) for 4 h. Following HNE treatment, cells were processed for measuring cell surface prothrombinase activity. **, P < 0.01
**Figure S8. Effect of phenylarsine oxide (PAO) on HNE-induced p38 MAPK activation.** THP-1 cells were treated with a control vehicle or PAO (10 µM) for 15 min before they were stimulated with HNE (20 µM) for 15 min. Cells were subjected to immuno blot analysis to probe phospho- and total p38 MAPK.