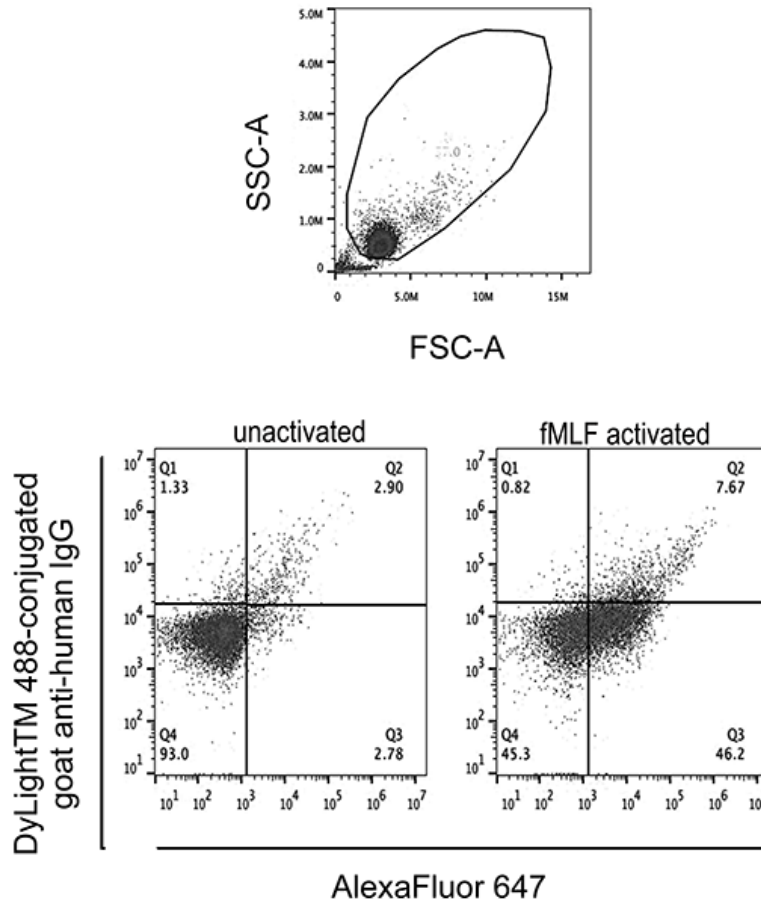
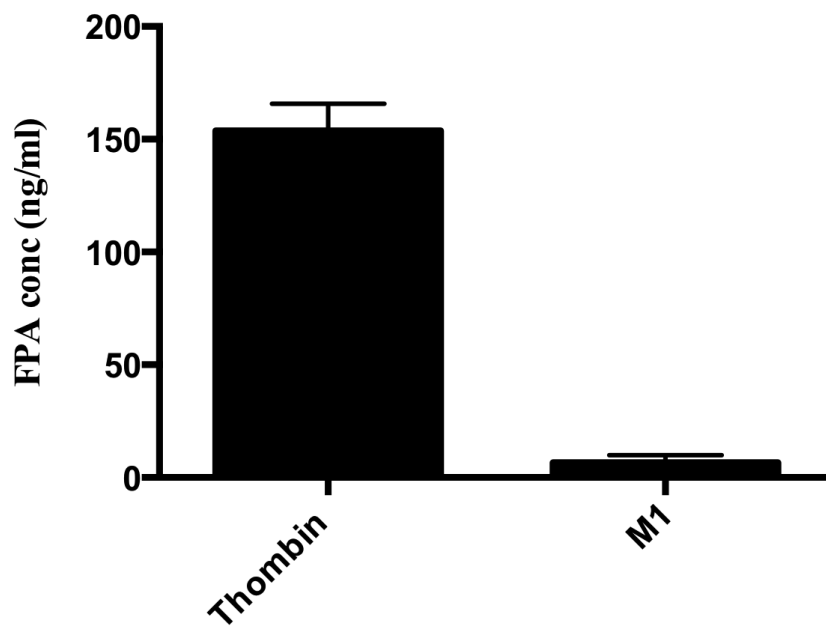


Supplementary Figure 1: ADP and Thrombin generate platelet activation while fMLF fails to do so. Citrated whole blood was incubated with buffer, ADP (10 μ M), Thrombin (0.5 U/ml + GPRP peptide), fMLF (1 μ M) or ADP + fMLF. Flow cytometry was used to determine platelet granule release (CD62P) (**A**) and platelet integrin activation (PAC-1) (**B**).



Supplementary Figure 2: Flow cytometry of neutrophil phagocytosis

Neutrophils were suspended in RPMI alone (unactivated), RPMI containing 1 μ M fMLF (activated). These neutrophils were incubated with serum opsonized Alexa 647 labelled bacteria before termination of phagocytosis on ice and staining of extracellular bacteria with goat anti human IgG Dylight 488. Flow cytometry was performed and representative dot plots are illustrated. Neutrophils were identified by typical forward scatter (FSC A) and side scatter (SSC A) characteristics (upper panel). Within the neutrophil population, cells that were positive for Alexa-fluor 647 (lower panel; Q2 and Q3) had intracellular and extracellular bacteria associated with them, while cells that are double positive for Alexa-fluor 647 and anti-human IgG 488 (lower panel; Q2) have extracellular bacteria.



Supplementary Figure 3. M1 protein does not generate fibrin. Citrated whole blood was incubated with Thrombin (0.5 U/ml + GPRP peptide), or M1 protein (5 μ g/ml) and Fibrinopeptide A generation was determined. Thrombin cleaved fibrinogen to release fibrinopeptide A and fibrin polymerization and clot formation was prevented by the GPRP peptide. M1 protein did not generate fibrinopeptide A and no clot formation was macroscopically evident.