**Effect of C5 inhibition on CC-induced inflammation.**

Human whole blood was added eculizumab or control CD20 antibody rituximab 5 minutes prior to 6 hours stimulation with CC (1.5x10^7 or 3x10^7 particles/ml) or PBS/HSA. (A) IL-1β, (B) TNF, (C) IL-6, (D) IL-8, (E) MIP-1α, (F) MIP-1β, (G) MCP-1 and (H) IL-1RA in plasma were quantified by multiplex analysis. Data represent mean (pg/ml) ± SD of one representative experiment of three.
**C3a and TNF as primers for CC-induced IL-1β release and effect of complement inhibition on phagocytosis of CC.**

Human PBMCs were primed for 2 hours in 10% heat inactivated human serum with PBS/HSA, C5a, C3a, TNF, a combination of C5a and TNF, or C3a and TNF prior to stimulation with CC. (A) IL-1β was detected by ELISA in supernatants 16 hours after stimulation with increasing concentrations of CC (0.15x10^7, 0.75x10^7, 1.5x10^7 particles/ml). One representative experiment of at least three performed is shown.

Human monocytes were incubated with CC (7.5x10^6 particles/ml) for 20 minutes or 60 minutes in the presence of DMSO control, control peptide, compstatin or Cytochalasin-D before phagocytosis was determined by confocal microscopy. (B) Cells that had phagocytosed CC were manually counted in eight images taken on different locations in each of three replicate wells, and plotted as the % of monocytes that had any phagocytosed CC for each group. Error bars: ± SEM. p<0.0001 and p<0.01 for compstatin induced reduction in phagocytic frequency compared to its control at 20 minutes and 60 minutes, respectively. One representative experiment of at least two performed is shown.