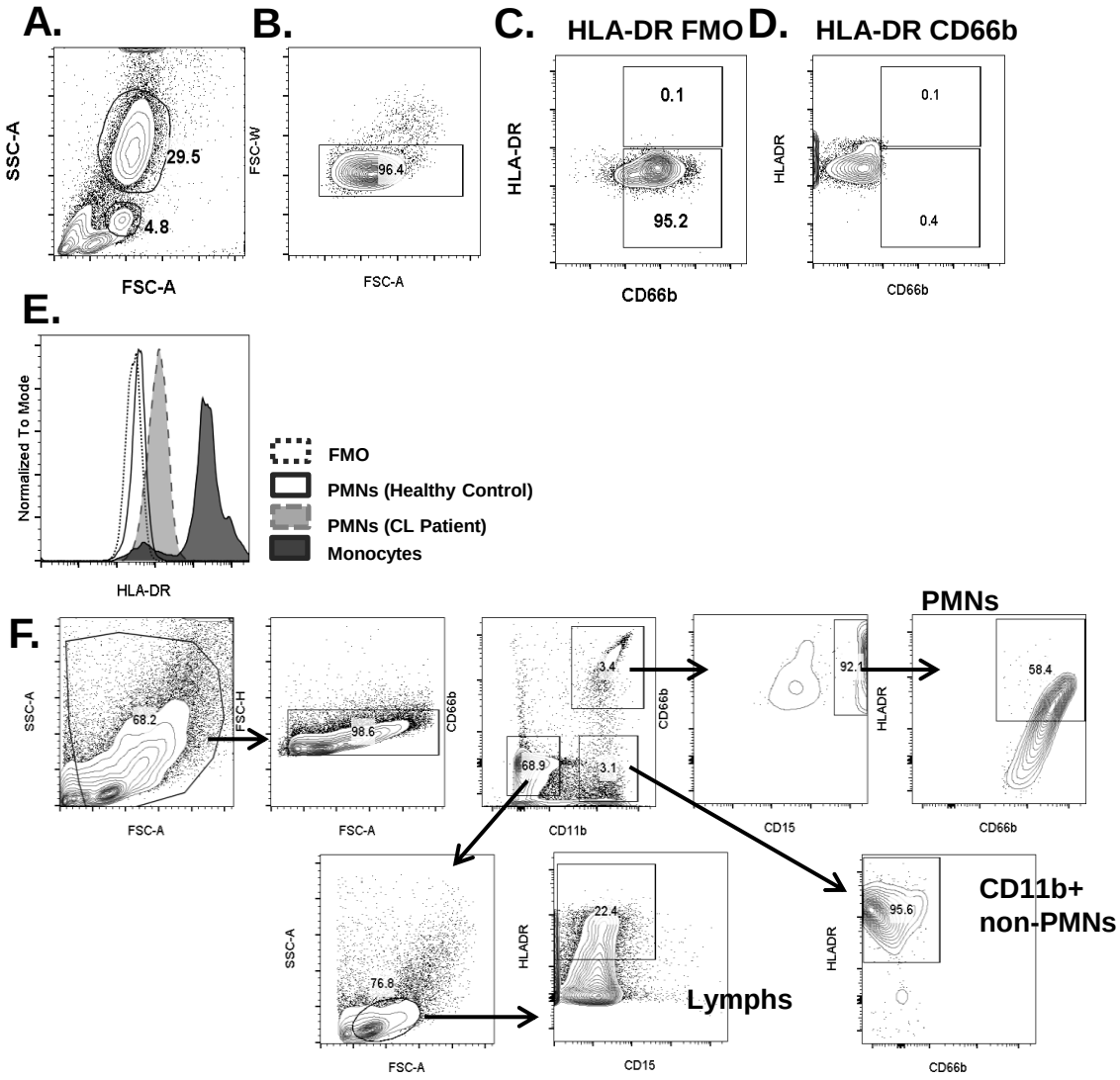
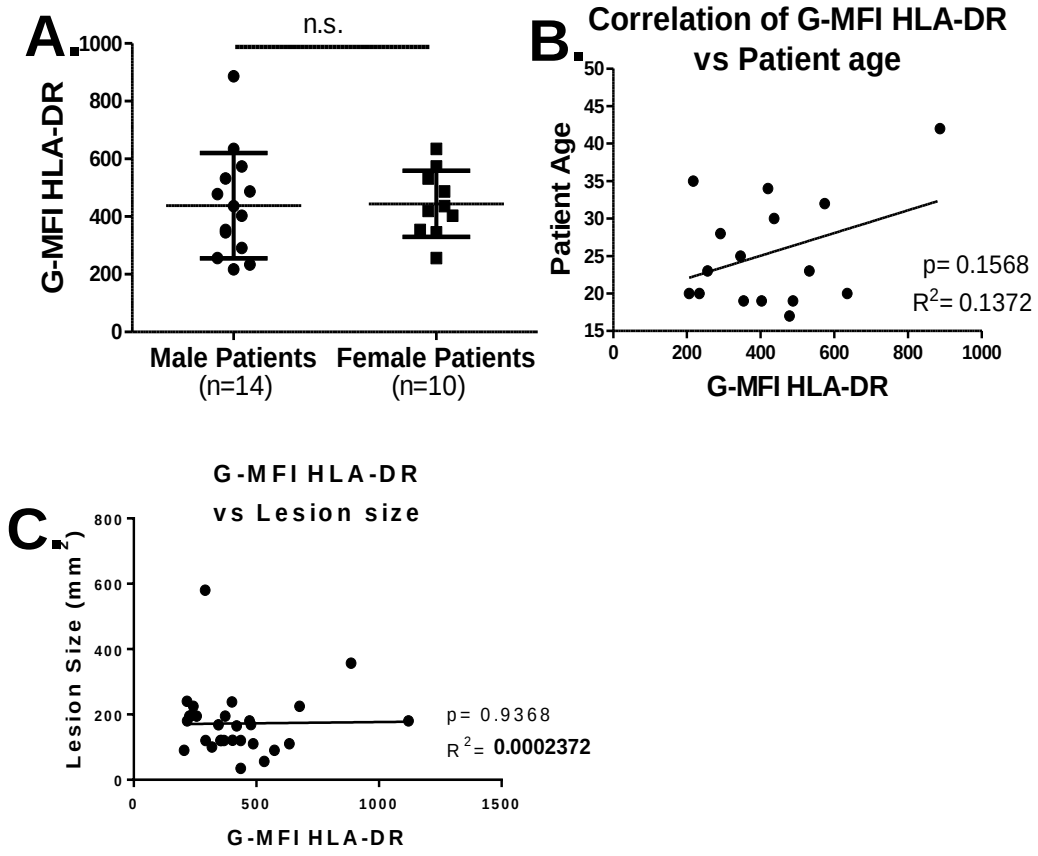
Supplemental Figure 1: Gating strategy for detection of CD66b+ HLA-DR+ PMNs in circulation and lesions



Supplemental Figure 1: Gating Strategy for detection of CD66bb+ HLADR+ PMNs. A. Gating on granulocytes and monocytes from stained and fixed CL patient whole blood. B. Gating on single cell subset. C. Fluorescence minus one (FMO) control used for setting cut off gates, showing gating on granulocytes without HLA-DR-antibody. D. FMO control showing gating on granulocytes without CD66b antibody. E. Histogram comparing expression of HLA-DR on FMO control, PMNs from healthy subject, PMNs from CL patient, and monocytes from a CL patient, demonstrating robust staining using HLA-DR antibody. F. Gating strategy for lesion-derived cells, showing PMNs (defined as CD11b+ CD66b+ and CD15+), CD11b+ non PMNs (CD11b+, CD66b-) and lymphocytes (CD11b- FSC/SSC, CD15-)

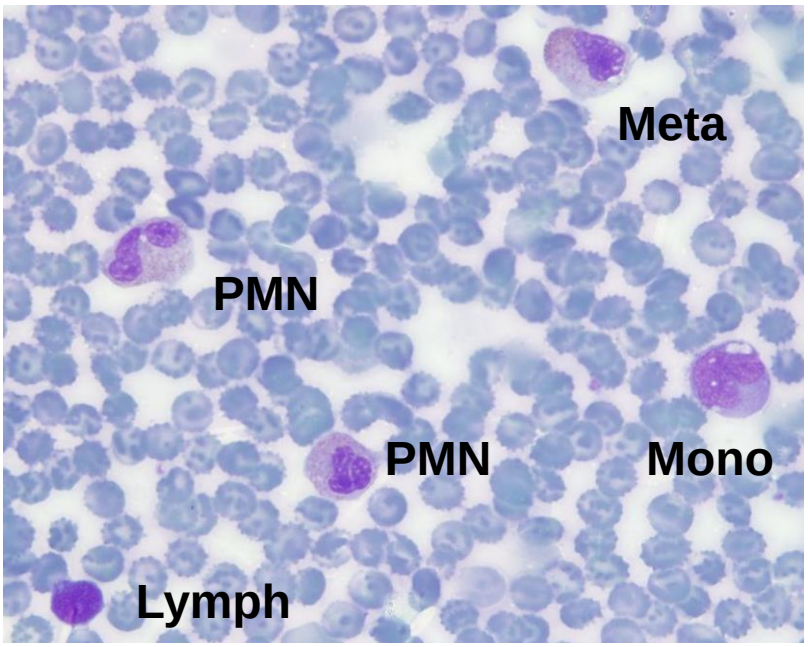
Supplemental figure 2: Patient sex, age, or lesion size do not correlate to expression of HLA-DR on CD66b+ neutrophils



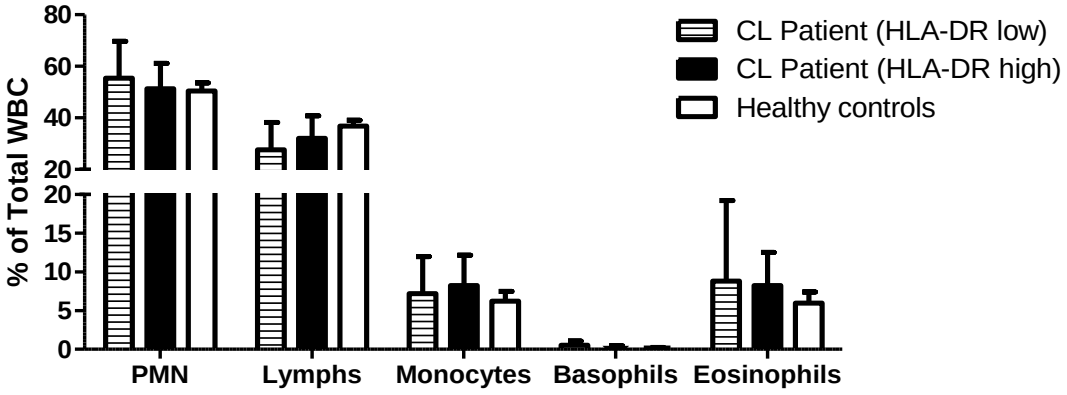
Supplemental figure 2: Patient sex, age, or lesion size do not correlate to expression of HLA-DR on CD66b+ neutrophils. (A) Male and female patients had equal expression of HLA-DR on CD66b+ neutrophils. (B-C) There was no significant correlation between patient age, panel B, or lesion size , panel C, and expression of HLA-DR on CD66b+ PMN**.**

Supplemental figure 3: CL Patient and Healthy Control Complete Whole Blood Differential

A.

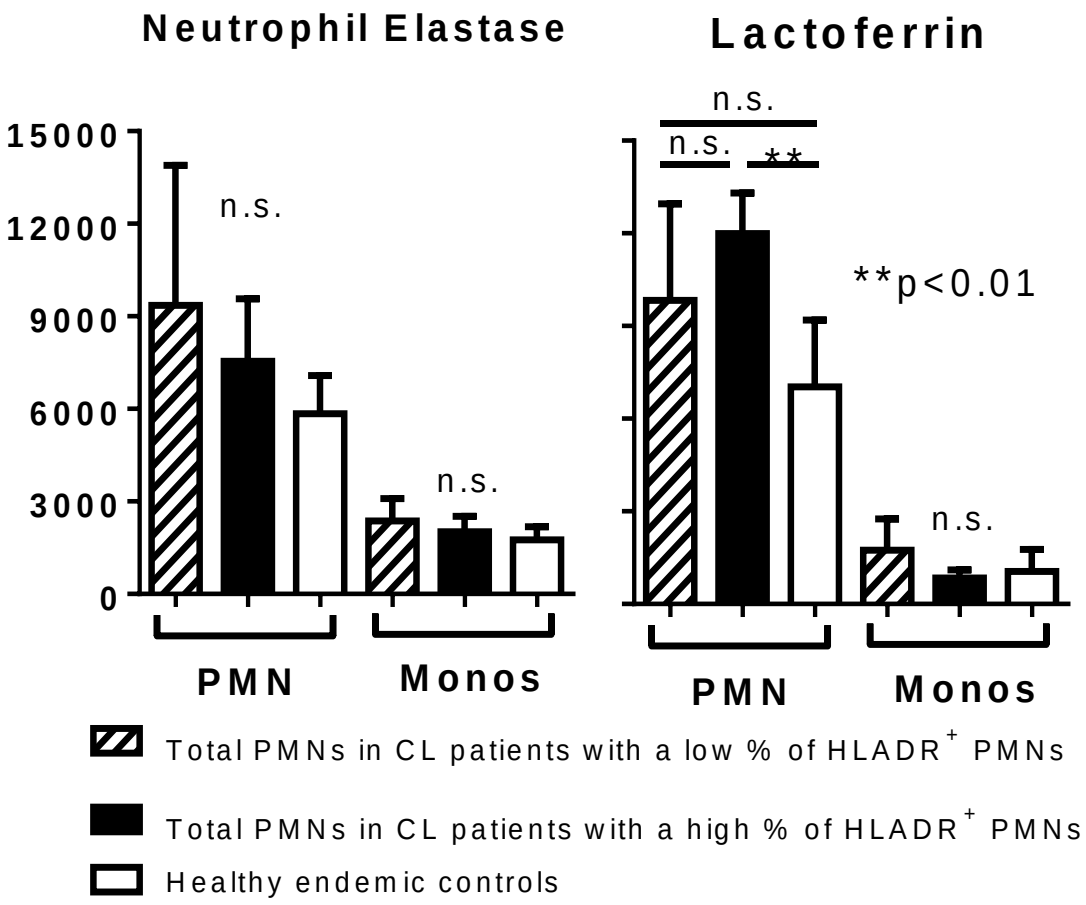


B. **Differential of CL Patient and Control whole blood**



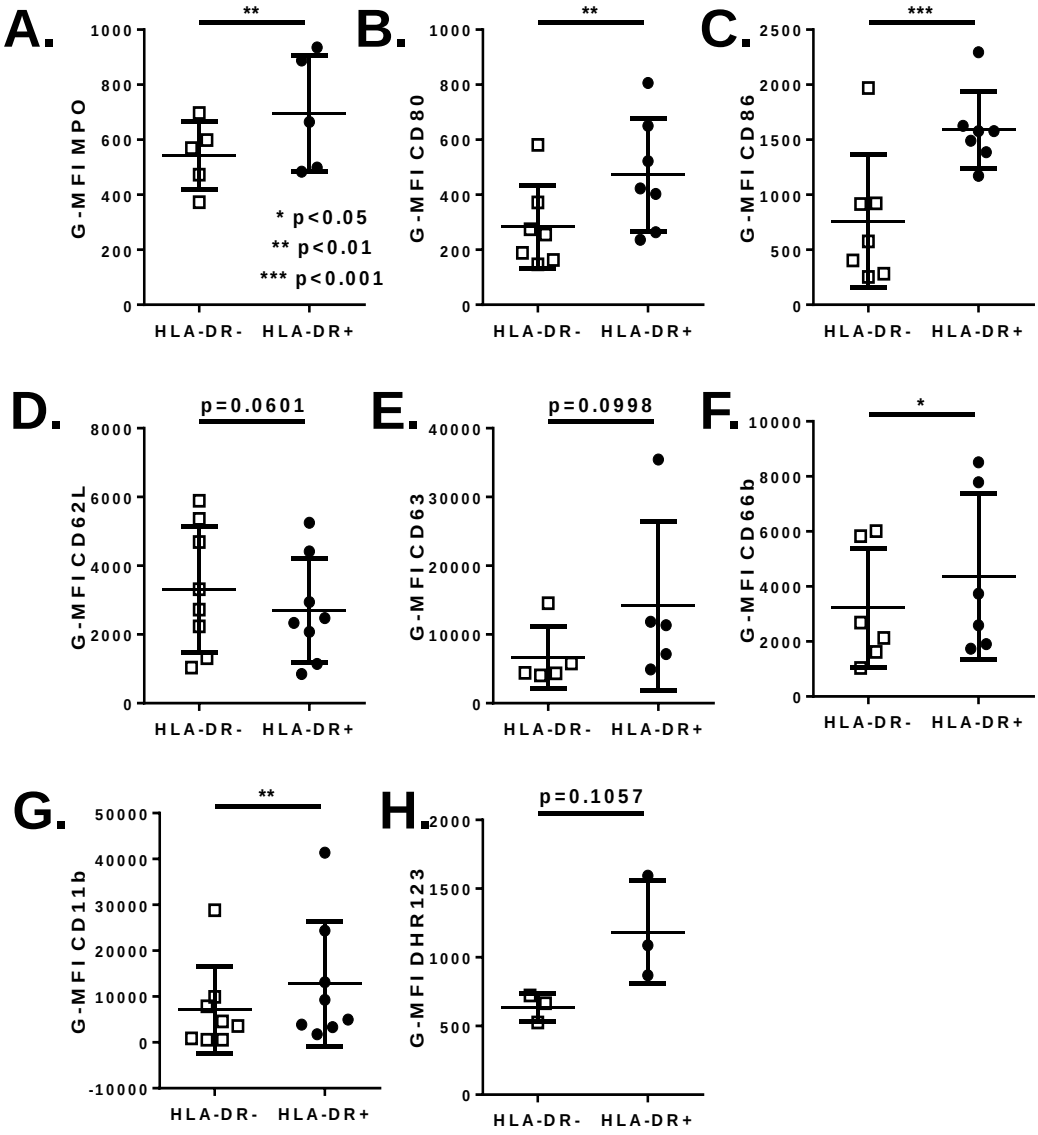
Supplemental Figure 3: Differential blood counts of leukocytes in whole blood from CL patients and healthy controls. (A) Image from CL patient blood smear showing a labeled lymphocyte, monocyte, as well as two mature PMN, and a metamyelocyte neutrophil form. (B) CL patients were divided into HLA-DR high (where >5% PMN were HLA-DR+) and HLA-DR low (<5% PMN expressing HLA-DR). There was no significant difference in percentages of PMN, lymphocytes, monocytes, basophils or eosinophils present when comparing any of the three groups. The presence of HLA-DR+ PMN doesn't relate to any measurable gain or loss in any other major leukocyte subset.

Supplemental figure 4: Total Neutrophil elastase and lactoferrin content does not differ between CL patients with high versus low proportions of HLA-DR+ PMNs



Supplemental Figure 4: Total Neutrophil elastase and lactoferrin content does not differ between CL patients with high versus low proportions of HLA-DR+ PMNs. Whole blood from CL patients (n=12 for MPO, n=7 for NE and LF) and healthy controls (n=5) was surface stained for CD66b, fixed and stained with markers for intracellular granules, neutrophil elastase, and lactoferrin.

Supplemental figure 5: HLA-DR+ PMN from Healthy Controls also show increased co-stimulatory molecules, and markers of neutrophil activation.



Supplemental Figure 5: HLA-DR+ PMN from Healthy Controls also show increased co-stimulatory molecules, and markers of neutrophil activation. Comparing HLA-DR+ and HLA-DR- PMNs from endemic healthy controls reveals similar phenotypes as in CL patents. HLA-DR+ PMNs have significantly increased intracellular MPO (A) and costimulatory molecules CD80 (B) and CD86 (C). HLA-DR+ PMNs showed greater, no not significant, activation direct ex vivo in terms of decreased surface CD62L (D), and increased markers of degranulation, CD63, CD66b and CD11b, though only CD66b and CD11b were significantly increased (E, F, G). ROS production by PMNs (as measured by DHR123 following stimulation) was increased, but not significant. Statistical comparison between paired HLA-DR- and HLA-DR+ PMNs were paired student t-tests.