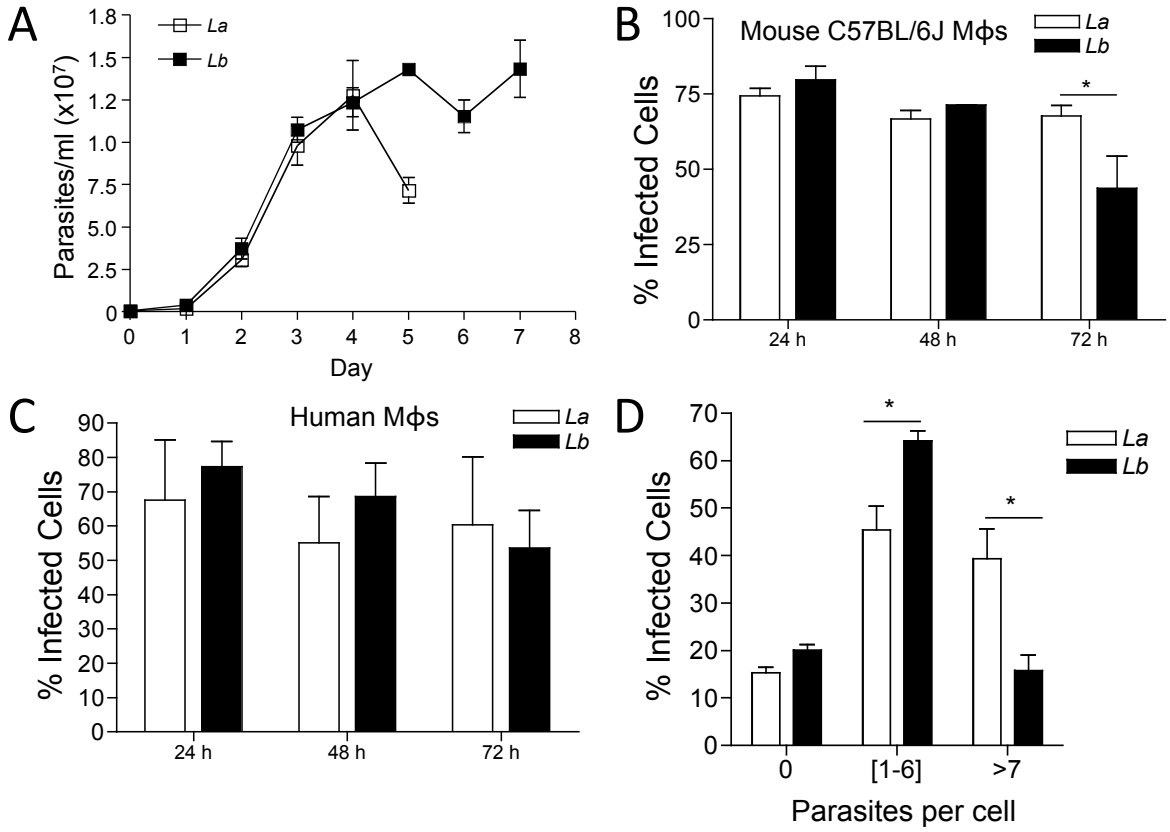

Figure S1. The growth rate and infectivity of *L. amazonensis (La)* and *L. braziliensis (Lb)* parasites. (A) For estimating parasite growth, *La* and *Lb* promastigotes were cultured at 23^0C in complete Schneider's *Drosophila* medium, and parasite numbers per ml was counted daily using a hemocytometer. (B) For estimating parasite infectivity *in vitro*, peritoneal macrophages of C57BL/6 mice (B) and human monocyte-derived macrophages derived from healthy donor blood samples (C and D) were seeded on glass cover slips (2×10^5 cells) in 24-well plates and infected with promastigotes at a 10:1 parasite-to-cell ratio at 32^0C. At indicated hours post-infection, cover slips were washed and stained with a Diff-Quik staining kit. The percentage of infected cells (B and C) and parasite loads per cell at 24 h (D) were determined by counting >250 cells in each group. Shown are pooled results from 2 independent experiments. * (p<0.05) indicates statistically significant differences by one-way ANOVA.

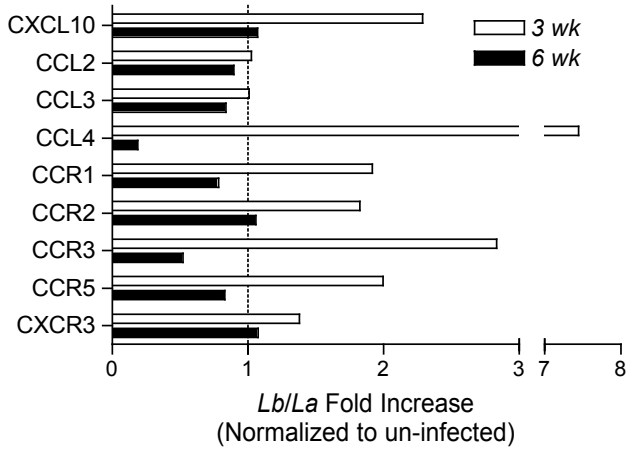

Figure S2. Early expression of proinflammatory mediators during *L. braziliensis* infection *in vivo*. C57BL/6J mice (n = 4) were infected in the ear dermis with stationary promastigotes of *L. braziliensis* or *L. amazonensis* (1×10^6). At 3 and 6 weeks post-infection, ear lesion tissues were collected and pooled for RNA extraction. The whole genome microarray analysis was performed by Miltenyi Biotec. The expression levels of indicated genes were normalized to un-infected ear tissues of age-matched mice. Results are presented as fold increases between the *Lb* and *La* groups at the indicated time points. The dotted line indicates comparable expression levels between the two infection groups.

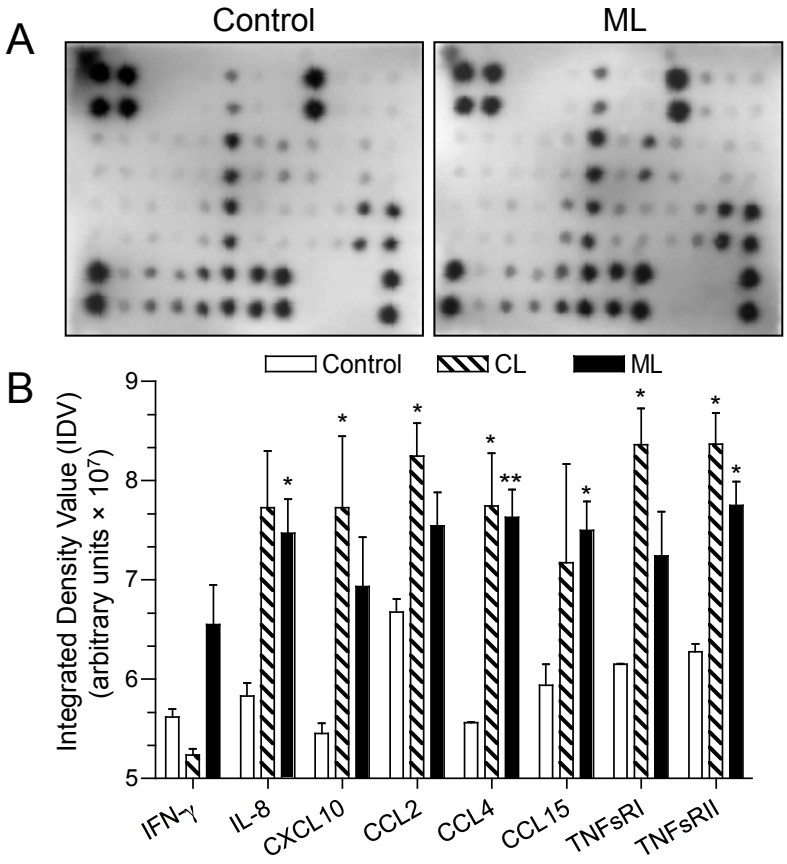

Figure S3. Increased production of proinflammatory mediators in the serum of American tegumentary leishmaniasis patients. Serum samples from 2 healthy volunteers, 2 CL and 4 ML patients were subjected to dot blot analysis. (A) Shown are two example images of the RayBiotech ® Human Inflammation Antibody Array for the detection of 40 inflammatory factors in the control and ML samples. (B) Densitometric analysis was performed to quantify the differences between groups. *($p < 0.05$) and **($p < 0.01$) indicate statistically significant differences by Student's *t*-Test.

Parameter	Control (n = 13)	CL (n = 13)	ML (n = 14)
Gender (%)			
Male	69%	69%	86%
Female	31%	31%	14%
Age (years)			
Mean ± SD	33 ± 9	41 ± 19	37 ± 10

Table S1. Demographic features of leishmaniasis cases and control subjects used in this study. Serum samples were obtained from patients after clinical confirmation of the disease but before the beginning of antimonial therapy. Table shows the gender and age distributions within the analyzed groups. Abbreviations used: Control, non-infected volunteers; CL, cutaneous leishmaniasis patients; ML, mucocutaneous leishmaniasis patients; SD, standard deviation.

Chemokine	Forward primer (5’ – 3’)	Reverse primer (5’ – 3’)	Product size (bp)
CXCL10/IP-10	CCAGAATCGAAGGCCATCAA	TGGAAGATGGGAAAGGTGAG	383
CCL2/MCP-1	CCCCAGTCACCTGCTGTTAT	GAGTTTGGGTTTGCTTGTCC	201
CCL3/MIP-1α	TGCATCACTTGCTGCTGACACG	CAACCAGTCCATAGAAGAGG	331
CCL4/MIP-1β	CCAAACCAAAAGAAGCAAGC	AGAAACAGTGACAGTGGACC	314
β-actin	GGCATCCTCACCCTGAAGTA	GGGGTGTTGAAGGTCTCAAA	203

Table S2. Oligonucleotide primer pairs used to amplify human chemokine genes. Primer sequences for CCL2, CCL3 and CCL4 were used as previously reported [Infect Immun. 1998. 66(3): 1121-6]. Primer sequences for CXCL10 (NM_001565) and β-actin (NM_001101) were designed using Primer-BLAST (NCBI, National Library of Medicine). Abbreviations used: bp, base pairs.