Lipoproteins are critical TLR2 activating toxins in group B streptococcal sepsis


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Corrections


In the section titled Genotyping in Materials and Methods, under the subheading DRB5, the sequences for the DRB5 TaqMan primers and probes are incorrect. The correct sequences are as follows: DRB5-specific primers (forward 5′-AGCAGGATAAGTATGAGTTCATTT-3′, reverse 5′-GTTCCTTGCAAGGATAAGTA-3′) and VIC-labeled DRB5-specific probe (5′-ACGGGACGGGCGCTTTGCTGCA-3′).


Fig. 7 was published incorrectly; Fig. 6 was duplicated in place of Fig. 7. The correct Fig. 7 is shown below. The published legend is correct, but shown again for reference.

**FIGURE 7.** The signal peptidase Lsp mediates inflammatory signaling induced by extracellular GBS factors but does not essentially mediate cytokine formation by fixed GBS organisms. HEK-TLR2 cells transfected with an NF-κB dependent ELAM-luciferase reporter gene (A and B) or RAW 264.7 macrophages (C) were incubated with escalating concentrations of cell-free GBS supernatants from wild-type GBS (■) or Δ lsp GBS (△) (A) or with ethanol-fixed GBS of the same strains (B and C). ELAM-luciferase activity was measured in HEK cell lysates by luminometry and is depicted as fold activation over background (medium control). TNF in the RAW 264.7 supernatants was determined by ELISA. Data depicted are mean ± SD of triplicate wells from one representative experiment of three or more performed.