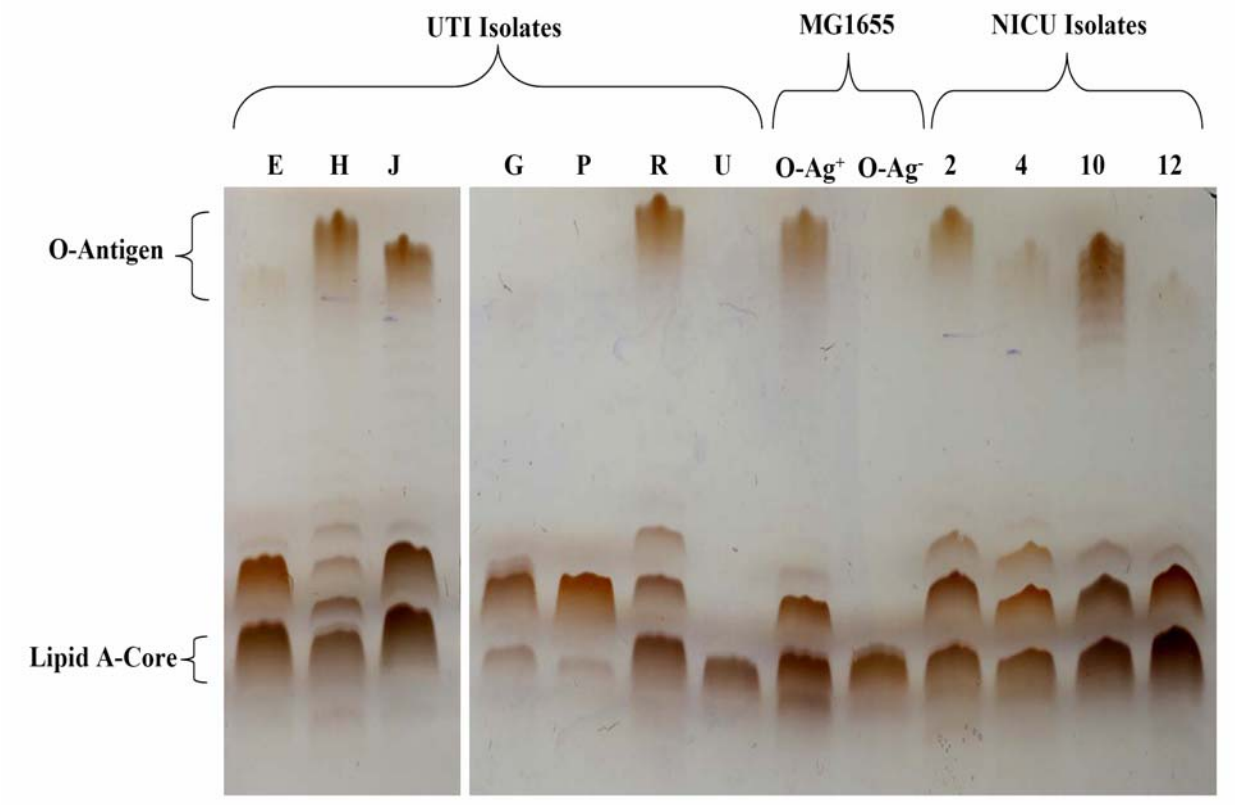

Figure S6. O-antigen profiling of clinical and laboratory strains of *E. coli*.

LPS samples extracted from 11 clinical isolates of *E. coli* together with two samples from the MG1655 strain with or without plasmid pMF19 are analyzed on SDS gels after silver staining. Plasmid pMF19 contains a functional copy of rhamnosyl-transferase gene, *rfaL*, which is mutated in *E. coli* K-12 [2]. Transformation of MG1655 strain with this plasmid allows production of O16 antigen [3]. Among the 11 clinical strains, there were 7 urinary tract infection (UTI) isolates, including UTI-E, -G, -H, -J, -P, -R, and –U, and 4 blood isolates from patients in neonatal intensive care units (NICU), including NICU-2, -4, -10, and -12 [4]. Based on the gel, UTI-U was clearly O-antigen$^-$ and UTI-P showed only the first smooth LPS band together with the lipid A-core band. Mutants with LPS banding pattern similar to UTI-P were reported not to react with anti-O antiserum and are considered phenotypically O-antigen$^-$ [5]. UTI-E and UTI-G produced less of high

molecular weight O-antigen variants, but they were clearly O-antigen^{+} when the gel was overloaded (data not shown).