Differential gene expression in biofilm cells suggests that adding the derepressed conjugative plasmid R1drd19 increases biofilm formation by affecting genes related to envelope stress (rseA and cpxAR), biofilm formation (bssR and cstA), energy production (glpDFK), acid resistance (gadABCEX and hdeABD), and cell motility (csgBEFG, yehCD, yadC, and yfcV); genes encoding outer membrane proteins (ompACF), phage shock proteins (pspABCDE), and cold shock proteins (cspACDEG); and phage-related genes.Cells with class II mutations (those in gatC, yagI, ompC, cspA, pspD, pspB, ymgB, gadC, pspC, ymgA, slp, cpxP, cpxR, cstA, rseC, ompF, and yqjD) displayed increased biofilm formation compared to the wild-type strain but decreased biofilm formation upon the addition of R1drd19.We hypothesize that the pili formed upon the addition of the conjugative plasmid disrupt the membrane (induce ompA) and activate the two-component system CpxAR as well as the other envelope stress response system, RseA-?E, both of which, along with BssR, play a key role in bacterial biofilm formation.Therefore, proteins encoded by the genes corresponding to the class I mutant phenotype are involved in R1drd19-promoted biofilm formation, primarily through their impact on cell motility.To investigate the link between the identified genes and biofilm formation upon the addition of R1drd19, 40 isogenic mutants were classified according to their different biofilm formation phenotypes.Cells with class I mutations (those in rseA, bssR, cpxA, and ompA) exhibited no difference from the wild-type strain in biofilm formation and no increase in biofilm formation upon the addition of R1drd19.