

Mobile genetic elements (MGEs) drive genetic transfers between bacteria using mechanisms that require a physical interaction with the cellular envelope. In ESKAPE species, the high-priority multidrug-resistant nosocomial pathogens, the membrane is surrounded by chemically diverse capsules, the first point of contact of virions and conjugative pili. While the capsule can be a barrier to MGEs, it also evolves rapidly by horizontal gene transfer. Here, we aim at understanding this apparent contradiction by studying the co-variation between the repertoire of capsule genes and MGEs in ~4000 genomes of *Klebsiella pneumoniae* and experimentally in natural isolates. We show that capsules drive phage-mediated gene flow between closely related serotypes. Such serotype-specific phage predation also explains the frequent inactivation of capsule genes, observed in more than 3% of the genomes. Inactivation is strongly epistatic, recapitulating the capsule biosynthetic pathway. We show that conjugative plasmids are acquired at higher rates in natural isolates lacking a functional capsular locus and in capsule mutants. This suggests that capsule inactivation by phage pressure facilitates its subsequent re-acquisition by conjugation. Accordingly, capsule re-acquisition leaves long recombination tracts around the capsular locus. The loss and re-gain process re-wires gene flow towards other lineages whenever it leads to serotype changes. Such changes happen preferentially between chemically related serotypes, hinting that the fitness of serotype-swapped strains depends on the host background. These results enlighten the bases of trade-offs between the evolution of virulence and multidrug resistance and caution that some alternatives to antibiotics by selecting for capsule inactivation may facilitate the acquisition of antibiotic resistance genes.