Vibrio cholerae **can transition from the human gut where it can cause disease into marine environments where it often occupies densely packed microbial biofilms on chitinous surfaces. Chitin and quorum sensing signaling trigger a DNA uptake apparatus for horizontal gene transfer and a Type VI Secretion System (T6SS) that delivers lethal effector proteins to kill adjacent competitors but not clone mates with identical protective immunity. Based on sequencing results, we identified an environmental and a clinical isolate predicted to engage in mutual T6-mediated killing due to distinct effector-immunity genes, which are adjacent on the chromosome. Mathematical modeling indicated that well-mixed dense populations of the two isolates should rapidly undergo phase separation as the cells within genetically-uniform groups no longer risk T6SS-mediated death. When incubated in co-culture the two *V. cholerae* strains spatially segregated and non-killing controls remained well-mixed, recapitulating the dynamics predicted by our models. Genome sequencing also predicted distinct effector-immunity pairs can be exchanged between bacteria. Indeed, chitin promoted horizontal transfer of the effector-immunity locus from the environmental to clinical *V. cholerae* isolate via the DNA uptake apparatus. The acquired T6 genes are functional and provide the recipient with T6-killing efficacy that is distinct from either parental strain. These results suggest that *V. cholerae* can adapt by rapid horizontal acquisition of new molecular weaponry.**