Antibiotic treatment selects for cooperative virulence of Salmonella typhimurium

JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)

ID 4480739
Author(s) Diard, Médéric; Sellin, Mikael E.; Dolowschiak, Tamas; Arnoldini, Markus; Ackermann, Martin; Hardt, Wolf-Dietrich
Author(s) at UniBasel Diard, Médéric ;
Year 2014
Title Antibiotic treatment selects for cooperative virulence of Salmonella typhimurium
Journal Current Biology
Volume 24
Number 17
Pages / **Article-Number** 2000-2005
Antibiotics are powerful therapeutics but are not equally effective against all cells in bacterial populations. Bacteria that express an antibiotic-tolerant phenotype (”persisters”) can evade treatment [1]. Persisters can cause relapses of the infection after the end of the therapy [2]. It is still poorly understood whether persistence affects the evolution of bacterial virulence. During infections, persisters have been found preferentially at particular sites within the host [3, 4]. If bacterial virulence factors are required to reach such sites, treatment with antibiotics could impose selection on the expression of virulence genes, in addition to their well-established effects on bacterial resistance. Here, we report that treatment with antibiotics selects for virulence and fosters transmissibility of Salmonella Typhimurium. In a mouse model for Salmonella diarrhea, treatment with the broad-spectrum antibiotic ciprofloxacin reverses the outcome of competition between wild-type bacteria and avirulent mutants that can spontaneously arise during within-host evolution [5]. While avirulent mutants take over the gut lumen and abolish disease transmission in untreated mice, ciprofloxacin tilts the balance in favor of virulent, wild-type bacteria. This is explained by the need for virulence factors to invade gut tissues and form a persistent reservoir. Avirulent mutants remain in the gut lumen and are eradicated. Upon cessation of antibiotic treatment, tissue-lodged wild-type pathogens reseed the gut lumen and thereby facilitate disease transmissibility to new hosts. Our results suggest a general principle by which antibiotic treatment can promote cooperative virulence during within-host evolution, increase duration of transmissibility, and thereby enhance the spread of an infectious disease.
Publisher Cell Press
ISSN/ISBN 0960-9822 ; 1879-0445
edoc-URL https://edoc.unibas.ch/64698/
Full Text on edoc No;
Digital Object Identifier DOI 10.1016/j.cub.2014.07.028
PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/25131673
ISI-Number WOS:000341541300024
Document type (ISI) Article