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In vitro co-culture of commensal *Escherichia coli* strains enhances Stx2a production by the German *E. coli* O104:H4 outbreak strain

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In 2011, a novel shiga toxin-producing *E. coli* (STEC) O104:H4 strain was associated with a large foodborne disease outbreak centered in Germany. The outbreak was characterized by a much higher rate of the hemolytic uremic syndrome (HUS) than typically occurs following STEC O157:H7 infections. Interestingly, this O104:H4 strain produced much lower levels of Stx2a than an STEC O157:H7 outbreak strain in the laboratory. Because the amount of Stx2a produced by O157:H7 strains is correlated with the development of severe clinical illness, such as STEC-associated HUS in humans, we wished to see if Stx2a-encoding phages released by these two STEC strains would increase toxin production by infecting commensal *E. coli.* In this study, we examined the role of commensal non-STEC in amplifying Shiga toxin 2a (Stx2a) production by the toxin-encoding phage released spontaneously from STEC. Co-incubation of *E. coli* K-12 C600 with the STEC O104:H4 strain ON-2011 and O157:H7 strain *EDL933* resulted in 21-and 8-fold increases in shiga toxin production, respectively. However, among commensal non-STEC, only isolates of serotypes OR:H19 and O46:H31 from two of ten human fecal samples significantly increased Stx2a production following co-incubation with ON-2011, and no increase was observed following co-incubation of commensal *E. coli* with *EDL933*. While stable Stx2a phage ΦON-2011 and 933W *E. coli* C600 lysogens were readily isolated following co-culture with these two pathogens, only ΦON-2011 lysogens were isolated following co-incubation with the commensal *E. coli.* Two genes encoding putative phage receptor-binding determinants were present in the ΦON-2011 genome but not that of 933W. While further study is required, it seems likely that differences in 933w and ΦON2011 commensal *E. coli* host range may result in variability in the levels of Stx2a produced in certain individuals during the course of infection which could contribute to differences in the severity of STEC-associated disease.

Biography

Yongxiang Zhang is a biologist from National Microbiology Laboratory of public health agency of Canada. He has experience in studying the evolution and virulence of shiga toxin-producing *Escherichia coli* and the shiga toxin-encoding phage.

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