**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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