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A chimeric protein (mTcd138) comprising the glucosyltransferase and domains of toxin B and the receptor binding domain of toxin A provides full protection against *Clostridium difficile* infection in mice

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Clostridium difficile is the major cause of hospital-acquired infectious diarrhea and colitis in developed countries. The pathogenicity of *C. difficile* is mainly mediated by the release of two potent large exotoxins, toxin A (TcdA) and toxin B (TcdB), both of which are pathogenic and require neutralization to prevent disease occurrence. We have constructed a novel recombinant fusion protein, designated mTcd138, containing the receptor binding domain of TcdA and the glucosyltransferase and cysteine proteinase domains of TcdB and expressed it in *Bacillus megaterium*. To ensure mTcd138 is atoxic, two point mutations were made in the glucosyltransferase domain of TcdB, which essentially eliminates the toxicity. Parenteral immunizations of mice with mTcd138 induced highly protective antibodies to both toxins and provided full protection against parenteral toxin challenges with lethal doses of toxins and infection with a hyper-virulent *C. difficile* strain UK6. Our studies demonstrate the potential of mTcd138 as a vaccine candidate against CDI in humans.

Biography

Xingmin Sun is an Assistant Professor in research focusing on *Clostridium difficile* infection at Tufts University, USA. He received his PhD (*magna cum laude*) in Natural Sciences from University of Kiel, Germany, and did his Post-doctoral training in Molecular Microbiology, Cell Biology & Biochemistry at Brown University, USA. He received Young Scientist Award from Federation of European Microbiological Societies in 2002, and Infectious Diseases Fellows Grant from American Society for Microbiology and the Infectious Diseases Society of America in 2008. His research is supported by NIH and private sectors. He is investigating *C. difficile* toxin-mediated signal transduction, leading to the production of proinflammatory mediators. He is also developing preventive and therapeutic approaches targeting *C. diffcile* toxins and proinflammatory mediators in *Clostridium difficile* infection.

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