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Beta-lactam antibiotics, the human microbiome and the risk of invasive *Candida albicans* infection

Candida albicans is an opportunistic fungal pathogen in humans. While it normally colonizes the gut and skin as a commensal yeast in healthy people, it is also a common cause of life-threatening invasive infection in immunocompromised patients, leading to ~400,000 deaths every year worldwide. What causes *C. albicans* to transform from a harmless resident in our body to a deadly pathogen? The use of broad-spectrum antibiotics is one of the well-recognized risk factors for invasive *C. albicans* infection, although the underlying mechanism remains unclear. β-lactams, the most commonly used class of broad-spectrum antibiotics, act by inhibiting peptidoglycan (PGN) polymerization in bacteria, leading to the accumulation and release of PGN subunits upon bacterial cell lysis. In a previous endeavor to identify the molecules in the human blood that promote *C. albicans* infection, we detected bacterial PGN subunits and demonstrated that some subunits were extremely potent inducers of *C. albicans* hyphal morphogenesis, the invasive form of the fungus. This discovery inspired us to propose and test a potential mechanism by which β-lactam antibiotics could increase the risk of invasive candidiasis. We hypothesized that β-lactams promote *C. albicans* infection by forcing trillions of bacterial cells in the human microbiota to suddenly release a massive amount of PGN subunits which in turn drive *C. albicans* to undergo the yeast-to-hyphal transition. We have obtained compelling *in vitro* and *in vivo* evidence that supports our hypothesis, which could lead to new strategies for the prevention and management of invasive *C. albicans* infection.

Biography

Yue Wang has his expertise in fungal pathogens with a focus on *Candida albicans*. His main interest lies in the identification and characterization of both host and fungal factors that determine the pathogenicity of *Candida albicans*. His main discoveries include the hypha-specific gene HGC1 that controls hyphal morphogenesis and a range of Hgc1/Cdc28 substrates that play various roles in polarity control, vesicle transport and virulence. He also discovered that bacterial peptidoglycan subunits in the blood are potent inducers of *C. albicans* hyphal growth.

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