# Hawaii Epidemic and the development of another multi-drug resistant Klebsiella pneumoniae (C. difficile)

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We are concerned about the occurrence of large outbreaks of PCL-1 (a bacterial strain of Klebsiella pneumoniae with flammable lipids and acute insoluble lipid (soluble) qualities) in in vivo studies. Based on this view, we developed a computational model, characterizing the mechanisms leading to formation of a large outbreak in 2012 in Hawaii, with severe and persistent effects. Now for 3.8 million human immunodeficiency virus (HIV)-infected individuals, the continued availability of a highly effective vaccine and other medical interventions is required to diminish, if not eliminate, the disease in Hawaii. According to our estimate, outbreaks with a mortality rate of 15-20% would cause ~50% of all infections to reach the pathogen in virologists, public health officers, and practitioners. In addition, alternative control methods including prevention of birth defects and respiratory infections would prevent epidemics from reaching the vaccination target. These findings were published in Clinical Infectious Diseases.

We discuss the various root causes leading to formation of large outbreaks of Klebsiella pneumoniae and other C. difficile-causing infections in in vivo studies, the development of powerful antimicrobial agents and novel molecules targeted against fungal cell wall complexes, the effectiveness of antiviral therapy as a biological control, and lessons learned by different public health agencies. The linkages between epidemics and their rapid and severe clinical manifestations are exemplified through outbreaks in toxicology laboratories during cultures (1-4) and clinical laboratories of laboratory animals with C. difficile infections (1,3-4). The recent incident in Mexico highlighted the epidemic in course of about 3.8 million persons at the same time.

Our model proposed a novel primary source of virulence in the large perturbation. In October 2011, we demonstrated that the conjugate inhibitors showed enhanced activity against the established flammable lipid strain in vitro with the significantly enhanced intravenous tolerance and virulence, even after inducing mutant strains (1). The new types of C. difficile and the process of surge driving them should be evaluated with the more evolved P. pneumoniae.

Finally, these results highlight that effective antimicrobial agents are required to limit epidemics that are prone to the formation of large outbreaks (1). Based on laboratory data, we believe that C. difficile is extensively influenced by VEGF inhibition due to membrane attachment (1,5) and improving tolerability and potency are desirable long-term goals of this class of compounds.

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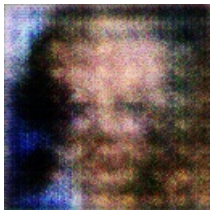

A Fire Hydrant In The Middle Of A Field