

Multidrug-Resistant *Vibrio cholerae*: A Global Threat

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Cholera, acute diarrheal disease is the cause of around 120,000 deaths per year, particularly children under 5 years of age. The disease occurs by ingestion of the Gram-negative bacterium "*Vibrio cholera*" contaminated food or water and it can pass through the human gastric acid barrier into the small intestine with cell multiplication and secretion of cholera toxin. Once *Vibrio cholerae* successfully colonize in the small intestine, the bacterial cells will secrete cholera toxin that activates the cystic fibrosis transmembrane conductance regulator (CFTR) in the epithelial cells lining the small intestine. This mechanism contributes to massive fluid efflux into the lumen of the small intestine. The toxin coregulated pilus (TCP), another virulence factor of *Vibrio cholerae* that allows the bacteria to aggregate and can protect the bacterial cells from shearing forces in the small intestine. On the basis of the somatic O antigens, this bacterial organism is a genetically versatile bacterial species that more than two hundred serogroups were identified. O1 and O139 are two major virulent strains. O1 virulent strain, a frequently identified isolate from several outbreaks is classified into two biotypes, classical and El Tor, based on phenotypic characteristics. Both biotypes are subdivided into serotypes, Inaba, Ogawa, and Hikojima.

Vibrio cholerae becomes resistant to antibiotics by exporting drugs through chromosomal mutations, efflux pumps encoding by *vexIJK*, *vexGH*, *vexEF*, *vexCD*, *vexRAB* operons or developing genetic resistance via the exchange of integrons, conjugative transposons, conjugative plasmids or self-transmissible chromosomally integrating SXT elements. Many countries were threatened by antibiotic-resistant *Vibrio cholerae*, such as 2003 in Vietnam, 2004 in China, India and Mozambique, 2005 in Bangladesh and Iran, 2006 in Namibia, 2007 in India, 2008 in Iran, 2008-2009 in Nepal, and 2009 in Zimbabwe. In 2014, the outbreak occurred in Haiti and revealed that mutations in the QRDR regions of the *gyrA* and *parC* genes and the presence of ICEVchHai1 containing the *sul2*, *strAB*, *floR*, and *dfrA1* resistance genes were the causes of resistance. The *EmrD-3* gene is another involved antibiotic-resistance gene that is identified in the El Tor strain.

In conclusion, whereas antibiotics are widely used for the cholera treatment that gradually increases *Vibrio cholerae* resistant strains, the development of drugs that inhibit known virulence mechanisms is another promising therapeutic approach.

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