Commentary

iMedPub Journals
www.imedpub.com

Journal of Applied Microbiology and Biochemistry
ISSN 2576-1412

2021
Vol.5 No.11:55

A Report on *Vibrio Cholerae* Biotype

Nurshad Ali

Department of Biochemistry and Molecular Biology, Shahjalal University of Science and Technology, Sylhet, Bangladesh

Received: November 05, 2021; **Accepted:** November 19, 2021; **Published:** November 26, 2021

***Corresponding author:** Nurshad Ali

Department of Biochemistry and Molecular Biology, Shahjalal University of Science and Technology, Sylhet, Bangladesh.

Nurshadali586@gmail.com

Citation: Ali N (2021) A Report on *Vibrio Cholerae* Biotype. J Appl Microbiol Biochem Vol.5 No.11:55

Commentary

Vibrio cholerae is a gram-negative, facultative anaerobe, comma-shaped bacteria. The bacteria are found in brackish or saltwater, where they easily attach to the chitin-containing shells of crabs, shrimp, and other shellfish. Some strains of *Vibrio cholerae* are pathogenic to humans and cause the deadly disease cholera, which can be contracted through the consumption of raw or undercooked marine life.

The structure of the cell surface lipopolysaccharide O antigen is used to classify *Vibrio cholerae* into more than 200 serogroups, only two of which, O1 and O139, are capable of causing epidemic or pandemic cholera. The O1 serogroup is further subdivided into two biotypes, classical and El Tor, which evolved from separate lineages and have genotypic and phenotypic differences.

Two major virulence factors of *Vibrio cholerae* O1 are cholera toxin (CT) and the toxin-coregulated pilus (TCP). The cholera toxin is encoded by the CTX prophage genes ctxA and ctxB and is responsible for diarrheal disease with severe water and electrolyte loss. The TCP is required for *Vibrio cholerae* colonisation of the small intestinal epithelium and is encoded by the tcp operon in the Vibrio pathogenicity island (VPI). Although ToxT primarily regulates these essential virulence factors via the ToxR virulence regulon (along with AphA and AphB) in both classical and El Tor biotypes, the genes are differentially expressed between the biotypes, particularly under in vitro inducing conditions.

The world has experienced seven cholera pandemics since 1817, the first six of which were caused by the classic O1 biotype. The current (seventh) pandemic, which began in 1961, is the result of the El Tor O1 biotype, which has largely replaced the classical biotype globally since 1993. Despite the fact that the El Tor biotype was isolated in 1937 and its pandemic potential was revealed 24 years later in 1961, both biotypes coexisted until 1992. During this time, the El Tor biotype was responsible for the majority of outbreaks; however, until 1992, the classical biotype was responsible for isolated incidents. Among these incidents were a large outbreak in West Pakistan in 1968 and the appearance of the classical biotype in Bangladesh in 1979, where it remained until the end of 1992.

However, since 2001, a number of reports have been published revealing clinical isolates dating back to the early 1990s that are of El Tor biotype background but exhibit some classical biotype traits. The ctxA gene, for example, which codes for the A subunit of the cholera toxin and is the first gene of the ctx phage operon, is completely conserved in sequence between the two O1 biotypes. The second gene in the operon, ctxB, which codes for the B subunit of the cholera toxin, is identical in sequence between the biotypes except for two bases, a key difference used to differentiate the biotypes. In the classical O1 biotype, the bases at positions 115 and 203 are cytosines, whereas in El Tor O1 biotypes, they are both thymines. These base transitions, which result in H39Y and T68I amino acid conversions, are completely conserved within each biotype, allowing them to be used as reliable biotype markers. These clinical El Tor O1 strains were discovered to have the classical biotype cholera toxin B subunit gene (ctxB) and were dubbed El Tor variants. Since other El Tor variants have been identified in Asia and Mozambique, as well as recently in Haiti, these El Tor variants have become a major focus of research. The recent publication of results indicating that some clinically isolated El Tor variants produce different levels of cholera toxin, including some isolates that produce higher levels than classical biotype strains, is of particular concern.

Despite evidence that the El Tor biotype has displaced the classical biotype globally; the emergence of El Tor variants has necessitated the use of standard genotypic and phenotypic assays to determine the biotype background of new isolates. Polymyxin B resistance, hemolysis assays, phage sensitivities, and the Voges-Proskauer tests are all common phenotypic assays used in some combination. To classify isolates as El Tor variants, genetic markers such as the ctxB gene have been used to distinguish the cholera toxin between classical and El Tor biotypes. The sequences of tcpA, which codes for the pilin subunits of the TcpA apparatus, and rstR, the repressor found in the RS1 element, which codes for the phage replication regulatory region, have also been used to determine the biotypes of new isolates.