**E.coli outbreak investigation**

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**Abstract**

Bacterial resistance to antibiotics is a global problem for modern science. Antibiotics are one of the most effective treatment against pathogens. And appearing in bacteria resistance to antibiotics is the cause of mass diseases, especially in economically poor regions. Therefore, one of the most important tasks is to study the causes of resistance of pathogenic organisms

**Introduction**

Bacterial antibiotic resistance causes by genetic features like many other bacterial characteristics [1]. One way for bacteria to get resistance is a single mutations in genes that are responsible for interaction of bacteria cell with the host-cell. This resistance variant could be powered by different mutagens presence in environment or by biotic features like polymerase replication mistakes which produce new variants of alleles that could be selected in future. Another way to get resistance is horizontal genetic transfer. It is caused by a transfer of some DNA molecules in bacterial cell and their introduction into genome. HGT could be powered by bacterial genes exchange [2]. When antibiotic resistance allele is acquired by one cell, it could be distributed among the population. Another type of HGT is represented by the mobile elements. Mobile genetic elements or transposons are DNA fragments that could travel through the genome and integrate in different positions cause changes in the genetic structure. Bacteriophages also carry molecules of nucleic acid which is necessary to infect bacterial cell. However, in some cases, they can remain in the stage of prophage causing no harm. On the contrary, it could improve host-cell by providing new features including antibiotic resistance[3]. In this article we present a study of E. coli X strain antibiotic resistance and toxicity reasons. It’s a highly pathogenic strain that was the reason of HUG (hemolytic uremic syndrome) and an epidemic in Germany.

**Materials and methods**

In the study libraries (SRR292678, SRR292862, SRR292770) from the TY2482 sample were used, which were generated at Beijing Genome Institute and deposited into the Short Read Archive (<http://www.ncbi.nlm.nih.gov/sra>). Raw reads were quality-checked with FastQC v. 0.11.8. Assembly of a single library of sequencing (paired end) reads from E. coli X. was performed using assembler SPAdes. Quality Assessment was performed using QUAST. Gene prediction and annotation was carried out using Prokka. Finding similar genes was accomplished by BLAST. 16s rRNA gene location was determined using genes prediction tool Barrnap. To search for genes responsible for antibiotic resistance, we used ResFinder. More information is in the repository [4].

**Results**

Data received after FastQC is in the lab journal [4]. When mate-pair libraries were added to the assembly, the N50 value decreased and the L50 value increased. This can be explained by the fact that the number of contigs increased, whose total length was at least half of the total assembly length.

According to the results, the blast analysis closest to the studied organism was Escherichia coli strain 503025 (CP025892.1). It was used as a reference.

**Discussion**

In our study, we found two toxic genes in the assembled genome of E. coli X (Shiga-like toxin II subunit B precursor and Shiga-like toxin II subunit A precursor). Blast analysis showed that they are the genes of the E. coli, however, many genes related to prophages were found in the environment of these genes. Therefore, it can be assumed that pathogenic genes were introduced into the genome as a result of the virus infection. Also we identified two genes responsible to antibiotic resistance (β-lactam antibiotics) - bla1 and bla2. Surrounded by a bla2 gene, the Tn3 transposon was found. The Tn3 transposon is a mobile genetic element, found in prokaryotes. It encodes the protein: β-lactamase, an enzyme that confers resistance to β-lactam antibiotics (and is encoded by the gene Bla). Therefore, the proposed mechanism for acquiring sustainability is a donor-recipient mechanism. Multiple origins and replication factors are located near the gene. There is a speculation that such structures may affect the acquisition of resistance to β-lactam antibiotics [5].

**References**

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