

Environment Arrays: A Method for Predicting Changes in Bacterial Disease Potential in Water

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Opinion

Although current genetic approaches for identifying bacteria in water have proven to be effective, they do not consistently forecast disease outbreaks. Genomics-based techniques will aid in the early detection of diseases before they grow into a population that poses a public health problem. We believe that genetics is just one tool in the toolbox that will be required to discover new waterborne dangers. We propose an approach based on mobile genome activity that goes beyond genomics. This method employs a novel gadget known as an environment array. The array will rely on the same research as genomics-based detection, but it will not necessitate any prior information. Molecular profiles of infectious components that transmit between bacteria are used to create environment arrays. The array has the advantage of monitoring the mobile genome's activity rather than the presence of specific DNA sequences. Traditional hybridization or PCR-based approaches that target already known DNA sequences should consequently be many times more sensitive than environmental arrays. Mobile elements are known to respond to new environmental conditions that could indicate a chemical pollution or a bacterial pathogen bloom, potentially allowing for a far larger applicability in identifying undiscovered biological and chemical threats. All public health practitioners want to be able to avoid disease outbreaks or even isolated instances. The best public health management strategies enable us to treat diseases and, in some cases, even prevent the spread of outbreaks. When infectious organisms are well-known ahead of time, current microbiological techniques can provide early warnings. Genomics-based techniques are intended to expand the detection envelope even farther, but they, too, can only detect what we already know to look for. In this light, we feel it is already appropriate to propose post-genomic methodologies for tracking the emergence of biological and chemical agents that are regarded hazardous to human health or the environment. The difficulty with existing environmental monitoring systems is recognising small populations or rare genes in a vast volume of data. We propose that we track changes in the genes that make up the mobile genome for this purpose. The vectors, such as plasmids, transposable elements, integrons, phages, and the genes they carry, make up this genome. These genes are constantly moving and changing, and they rarely collect in large numbers within species. They can, however, cause bacteria to quickly adapt to new surroundings. Antibiotic resistance genes, emblems of the mobile genome, first appeared in bacteria that were human diseases roughly 60 years ago, demonstrating this rapid adaptation. According to this research, even little and local changes in the environment might cause alterations in the mobile genome. A flood of adaptive genes will modify the genomic profile

of more than just potential diseases, because all organisms in a changing environment must adapt or die. Because genes can transfer hundreds to thousands of times even among bacteria that are not dividing, seeing changes in a large number of microorganisms at once can be a huge signal. To dramatically raise the copy number of a particular (e.g., virulence) gene in an environment, one would have to wait for several generations of cell division. Genes that help bacteria adapt to new settings frequently don't become citizens of the genome until they reach a specific population density on mobile vectors. These genes eventually settle onto chromosomes, similar to many antibiotic resistance genes and restriction-modification systems. A method for characterising the gene population of mobile pathogens fast. It is argued that a possible pathogen can be detected by looking for genes or gene products that have structural or functional similarities to genes or gene products that are known to be pathogenic. The genomes of different isolates of the same bacterial species can differ by 20%. Smaller changes can have a big impact. For example, the difference between a deterministic pathogen like shiga toxin-producing *E. coli* and our regular colonic *E. coli* can be as little as a few genes, if not only one. These genes are frequently found in plasmids or phage, mobile gene vectors that traverse strain, species, and even biological kingdom genetic boundaries, and they are not uniformly distributed across all genomes of a single species. Saprophytic microbes could become deterministic pathogens thanks to mobile genes. The distinction between non-pathogenic *Vibrio cholerae* in aquatic or estuary water and pathogenic *Vibrio cholera*. The mobile genome is made up of genes that don't have a specific genome home and are thus not always evenly distributed among isolates of the same species. Horizontal gene transfer (HGT) is the asynchronous movement of genes (or subgene sequences) between organisms that allows the mobile genome to replicate. HGT frequently results in novel virulence gene combinations. However, it is the mix of historical acquisitions of genes from gene vectors combined with other chromosomal genes that makes an organism a likely threat to human health or the environment, not just the last genes transported in by phage or plasmids. Because it is impossible to know the history of each bacteria in an environment ahead of time, monitoring the mobile genome for activity would dramatically lengthen warning times and detection limitations. HGT is shown in the short term by the rapid development of antibiotic-resistant superbugs, but it is difficult to detect using traditional molecular approaches. HGT has had a profound influence on the structure of organismal genomes, and is the source of unanticipated combinations of novel catabolic pathways and emerging diseases, according to a retrospective account of historical HGT occurrences.

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