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A proposed new selective growth medium for the isolation of enteroaggregative shiga-toxigenic strains of the emerging pathogen *Escherichia coli* O104:H4

The year 2011 was an eventful year for infectious emerging pathogens especially for the European continent. The shiga-toxin producing *Escherichia coli* O104:H4 was responsible for one of the largest outbreaks of gastroenteritis centered primarily in Germany leading in many cases to the life-threatening hemolytic uremic syndrome. In the same year in the United States there were at least two smaller outbreaks of food poisoning caused by *E. coli* O157:H7. The outbreak in Germany was found to be associated with contaminated bean sprouts while those in the US were associated with hazelnuts and romaine lettuce. Strains of *E. coli* O104:H4 were received from the Centers for Disease Control and Prevention. Comparative biochemical studies were performed with *E. coli* O157:H7 and *E. coli* O104:H4 using Biolog GEN III microplates and selective differential plates for identification of metabolic differences. It was also noticed the ability of the *E. coli* O104:H4 strains to form biofilms and their ability to bind Congo Red. The ability to activate the host innate immune response was also evaluated using human embryonic kidney (HEK) cells transfected with individual Toll-like receptors (TLR). It was during the course of these studies that it was found that *E. coli* O104:H4 could grow in the special medium used to measure TLR activation but neither *E. coli* ATCC25922 nor *E. coli* O157:H7 could not. With the result of these studies, a solid medium was devised that could support growth of *E. coli* O104:H4 but not *E. coli* O157:H7 or *E. coli* ATCC25922. The results of the studies will be presented with the proposed new selective medium that could potentially be used to differentiate enteroaggregative STEC stains of *E. coli* O14:H4 from *E. coli* O157:H7.

Biography

Kei Amemiya received his Doctoral degree from Rutgers University in Microbiology in 1973. He did his Postgraduate studies in Gene Regulation in the laboratory of Lucy Shapiro at Albert Einstein College of Medicine, Bronx, NY. Later, he went to the National Institute of Neurological Diseases and Stroke in 1986, where he examined gene regulation in JC virus that caused the demyelinating disease progressive multifocal leuko-encephalopathy in immune suppressed patients. In 1999, he went to the US Army Medical Research Institute of Infectious Diseases, Bacteriology Division, where he has been involved in vaccine development for Burkholderiamallei and Yersinia pestis. His primary interest has been in the immune response and innate immunity in animal models.

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