The term atypical EPEC is used to define the EPEC strains that does not have the EAF plasmid. This EPEC category has been found in association with diarrhea in several countries, including Brazil. At present they are the most common bacterial agent in child endemic diarrhea in Brazil. Atypical EPEC strains may belong to serotypes of the classical EPEC. The serogroups (classical serotypes) or to serotypes in other the serogroups (non classical serotypes). The classical atypical EPEC serotypes have been identified in several studies but the non-classical ones are less known. The classical serotypes are genetically related to *E. coli* O157:H7 and like this serotype have been isolated from bovines and other animal species. The most common virulence factors identified so far in atypical EPEC strains are the toxins EAST and Hly but recent studies suggest that there are many others factors. The objectives of this project may be divided in two groups: one group is aimed at determining the virulence mechanisms of these organisms and includes studies on their toxins, adhesins, genetic of these virulence factors, interaction mechanisms with the host cell, cell response and proteomics. The other group of objectives includes the characterization of the serotypes and clonal groups of atypical EPEC, genetic relationships of the human and animal strains an development of animal models.
The present study is focused on distinct aspects of atypical enteropathogenic Escherichia coli (aEPEC). Initially, the prevalence of aEPEC was determined in a study of the etiology of infantile acute diarrhea in Salvador (BA), where aEPEC was the second most prevalent bacterial pathogen, indicating the emergence of this pathotype in our country. Characterization of 90 aEPEC strains showed that they belonged to 62 different serotypes and presented 14 different intimin subtypes. Adherence assays showed that 42% of them did not adhere to HEp-2 cells and the remaining strains presented localized-like, diffuse, aggregative, localized or undetermined adherence patterns, which were also maintained in intestinal cell lines in culture. The analysis of the attaching-effacing (A/E) caused by aEPEC detected a delay in the bacterial adherence and the A/E formation that correlated to the late expression of intimin, Tir and EspA, caused by the lack of the perABC regulator. Genetic studies showed that genes encoding different virulence factors of other diarrheagenic E. coli pathotypes were present in different frequencies. Analysis of genetic relations among strains of aEPEC of human and animal (rabbit, monkey, bovine, ovine, cat and dog) origins suggested that these animals are aEPEC reservoirs. Also, an antiphagocytic effect induced by aEPEC was observed in macrophages J774A1. The enterohemolytic activity of aEPEC presented new features regarding its expression in different culture media and fibronectin binding capacity. Antisera against different adhesins and toxins were produced and employed in immunological assays aiming the differentiation of aEPEC from typical EPEC and EHEC. Other aspects of this project, such as proteomic analysis, biofilm formation, plasmids and adhesins characterization, and development of a murine model of infection are still in progress.

SUMMARY OF RESULTS TO DATE AND PERSPECTIVES

MAIN PUBLICATIONS


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