

### III. Enteric infections

#### *Aeromonas* in enteric infections: Introductory comments

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My laboratory at Colindale has had a general interest in *Aeromonas* as a possible cause of diarrhoeal disease for many years, but a year or so ago we were rather suddenly required to provide a reference service for these organisms for the whole of England and Wales. Obviously it is impossible to become an expert in such a complex field overnight but I was driven to take a fresh look at the literature in much the same way as a complete newcomer would. My impressions and emotions at that time, and now, can be summed up in one word – confusion. Surely there cannot be any other area in the whole of medical microbiology that contains so much contradictory evidence.

I understand that an association between diarrhoea and the presence of *Aeromonas* in faeces was first reported as early as 1937 – perhaps others may know of even earlier reports. In any case this means that the role of *Aeromonas* in diarrhoeal disease has been under investigation now for 50 years or more and yet, even now, have we reached the stage where it can be unequivocally stated that *Aeromonas* causes diarrhoea?

Let us briefly look at just some of the problems. Since the early 1960's there have been numerous reports in all five continents, first (and rather rarely) of outbreaks of diarrhoea apparently associated with *Aeromonas* and, secondly, of varying frequencies of isolation of *Aeromonas* from individuals with and without diarrhoea. The reported variations resulted in part from differences in methodology but also seemed to indicate genuine differences between different populations. In most countries the faecal carriage rate in asymptomatic people was low, less than 4%, and most isolates came from diarrhoea cases. But in some countries, Thailand for example, *Aeromonas* was found with equal frequency in diarrhoeal and healthy stools – the carriage rate being about 27%.

In the 1980's the situation seemed to improve with a few studies directly comparing the isolation rates in diarrhoea and asymptomatic groups. Several of these studies, and notably those in Australia, have shown that *Aeromonas* is found 10 times more frequently in diarrhoea cases than in asymptomatic controls. However, other studies showed that patients with diarrhoea yielded an even higher frequency of isolation if another pathogen was also present at the same time – could it perhaps be that some aspect of the pathogenic process of the other pathogen was somehow stimulating the shedding in the faeces of a non-pathogenic *Aeromonas*, or might the *Aeromonas* simply be an indicator

of the consumption of contaminated water that also contained the other pathogens?

In the case of other enteropathogenic organisms, especially *Escherichia coli*, the discovery of pathogenicity factors such as enterotoxin production and enteroinvasiveness led to a much better understanding of the role of the organism in diarrhoeal disease. Similar studies in *Aeromonas* seemed only to further confuse the issue. Enterotoxins, cytotoxins and haemolysins were all described – some workers were convinced that they were all different manifestations of the same toxin, while other groups thought that they were each distinct and even managed to clone the different DNA segments responsible for their production. Some workers detected invasive ability while others tried and failed to confirm it. Even human challenge experiments failed to clarify the problem since strains carrying some of these pathogenicity markers failed to cause diarrhoea when fed to human volunteers.

Even the taxonomy of the organism has increased the confusion. I won't attempt to summarise the history but the current vogue seems to be to divide the motile, mesophilic aeromonads into three species – *A. hydrophila*, *A. sobria* and *A. caviae*. This has opened up the possibility of correlating pathogenicity with species and several groups have confirmed that the accepted pathogenicity markers can be found in *A. hydrophila* and *A. sobria* but not in *A. caviae*. This must be progress because many of the strains referred to us are *A. caviae* – surely we can now tell the clinicians that these strains at least are not causes of diarrhoea. Unfortunately it appears not to be so, since some groups of workers have produced clinical and epidemiological evidence that *A. caviae* also may cause diarrhoea and they conclude that the absence of the accepted pathogenicity markers does not exclude the possibility of enteropathogenicity – a statement that sounds very familiar to those of us who were involved in the debate during the 1970's over the pathogenicity of the classical enteropathogenic *E. coli* or EPEC as they are now called.

In other organisms the use of serotyping as a very precise epidemiological marker has also helped to resolve some of the problems and we are fortunate to have Dr. Sakazaki here today to describe the first steps in this direction for *Aeromonas*. Perhaps he and the other eminent speakers here this morning can help to throw some light in our, or at least on my, confusion.

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#### *Aeromonas* and *Plesiomonas* – enteric infections and fecal carriage

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*Aeromonas* spp. were first recognized as pathogens for cold-blooded animals. After pioneer studies by Caselitz<sup>7</sup> and Laurot<sup>18</sup> in the sixties these members of Vibrionaceae have come to be recognized as primary human pathogens. A wide variety of clinical infections has been reported, from extraintestinal infections in immunocompromised as well as healthy persons to intestinal ones involving the small and/or the large intestine<sup>21</sup>. *Plesiomonas shigelloides*, also a member of the Vibrionaceae is

rarely isolated from extra-intestinal infections<sup>21</sup>. Progress in recent years in the development of selective media to isolate and quantitate *Aeromonas* and *Plesiomonas* from water, food and human as well as animal stools<sup>23, 24, 35</sup> has increased our knowledge of the environmental spread of these organisms.

Both species can be isolated from fresh and salt waters ubiquitously<sup>21</sup>. They show a seasonal variation with higher bacterial counts during the summer<sup>14, 16, 17</sup> in spite of the ability of *Aero-*