

COMMENTARY

by Bernard Dixon

AN APOSTROPHE TO AEROMONADS



It must be weird, being a microbe. Exactly what do you have to do to get noticed? Some members of your community—*Saccharomyces cerevisiae*, *Escherichia coli*—attract constant, rapt attention from researchers all over the world, while others are almost totally ignored. Some microbes are given famous binomials, and then re-named into oblivion. Some are thrust into the scientific spotlight, with impressive write-ups in so-called learned journals, only to fall from favor within a matter of months. In 1892, for example, *Haemophilus influenzae* is successively discovered, heralded, and reviled as the agent of influenza. But for the next half century, “experts” dismiss this fastidious rod as the organism that does not cause flu, allowing it to continue causing many other diseases without let or hindrance. Other members of the protista, like *Legionella pneumophila*, actually kill people quite frequently, the extent of their predations coming to light only when they go over the top, produce a sizeable epidemic during a gathering of important people, and trigger the scrutiny of deceased individuals’ blood samples by serological archeology.

Consider, then, what it must mean to be a member of the venerable and extensive genus *Aeromonas*. Although a German bacteriologist named Zimmermann notices your distinguished dynasty for the first time in the late nineteenth century, the vast majority of your kinsmen continue to be denied publicity. Decade after decade, they provoke illness in metazoa as diverse as catfish, carp, and *Homo sapiens*, without really breaking into the textbooks in a big way. Just the occasional footnote in chapters about *Vibrio cholerae*, nothing more. Then, out of the blue, all hell breaks loose. So-called clinical microbiologists, ichthyologists, and biotechnologists suddenly begin falling over themselves to learn more about you and your brethren. The number of research papers on *Aeromonas* triples between 1970–75 and 1980–85, and in September 1986 you are honored with a hugely popular satellite meeting during the 14th International Congress of Microbiology, held in Manchester, England. Though mildly insulted to be described as “microbiological latecomers” by Professor Alexander von Graevenitz (isn’t it their latecoming *H. sap* should be discussing?), you find the newfound fame welcome for all that. It’s certainly a giant leap forward from that solitary mention by Zimmermann, O.E.R. in *Ber. naturw. Chemnitz* 1:38, 1890.

But why are they newly interested in you? One clue emerges from research workers at the Instituto cantonale Batteriosierologico in Lugano, Switzerland. They make the journey to Manchester to boast of their triumph in so-called “typing” your fellow aeromonads, and how this has led to possible methods of attack. They have, it seems, been scouring lakes, rivers, and fish tanks not only for *Aeromonas* strains but also for bacteriophages capable of

devastating those strains. Using 99 phages (including some which they unaccountably have to acquire from the Institute of Technology in Adelaide, Australia), Drs. Peduzzi and Demarta have been able to so-called “characterise” 70–80 percent of aeromonads. “The lysotypic profiles obtained with the 25 phages that react with strains of environmental and human origin confirm the hypothesis of the hydric origin of *Aeromonas* infections in Man,” they tell the Manchester cabal, plausibly enough. “In fact, bacterial strains of different origin present the same lysotypy and can thus be considered identical.”

It’s when the well-intentioned pair from Lugano start to talk of “therapeutic and prophylactic uses” of phages that one begins to question whether to welcome the unaccustomed celebrity suddenly thrust upon the proud name of *Aeromonas*. “Pisciculture is a great reservoir of this germ,” Peduzzi and Demarta assert. They go on to describe “sanitary problems” in fish farms. Then they report how, on the basis of “successful” experiments in which they added lytic phages to tanks brimming with trout, they plan to curtail the so-called problem of *Aeromonas* infection during fish breeding programs. We shall see.

Far more far-sighted in his attitude and approach is another Swiss-based researcher, Ken Timmis from the University of Geneva. Along with Didier Favre, he has taken a close interest in the capacity of *Aeromonas hydrophila* to secrete a diversity of interesting proteins into the culture medium. They join the Manchester symposium to explain why this capacity is so fascinating and potentially beneficial. The transport of proteins across two lipid bilayers is inherently interesting to intelligent people, Professor Timmis vouchsafes, and quite rightly. But just as exciting is the opportunity for biotechnologists to exploit this excretion mechanism to create a host-vector system for the secretion of products of cloned genes.

Timmis and Favre describe how the screening of a cosmid gene bank of *A. hydrophila* DNA (constructed, inevitably, in *E. coli*) led to the identification of clones coding for four excreted proteins—an amylase, an RNase, a cytotoxin, and a lipase. “Following subcloning into pUC vectors, mapping of these genes was carried out by Tn 1000 mutagenesis,” they continue, in the strangely passive prose scientists always employ. “Hybridisation of probes representing internal fragments of the cloned genes to digested chromosomal DNA of *A. hydrophila* showed that all of the isolated genes are present in the genome in multiple copies. All four proteins were found in the periplasm of *E. coli*, indicating that the cloned genes contained information for the transfer of their products across the cytoplasmic membrane but not the outer membrane.”

Good luck and very well done. That’s what I would be saying to Professor Timmis, had I been born of *Aeromonas* stock. I would also be using my single polar flagellum to steer clear of Lugano. Fame is all very well, but not at the price of lytic humiliation.

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