Cholera in Australia — no cause for alarm

Cholera is a disease with a frightening reputation. The very name sounds fearsome, and in our minds is often associated with filth, excrement, and squalid mediaeval conditions.

Since the development of modern medicine and knowledge of the existence of bacteria and their transmission, most countries that can afford it have generally succeeded in banishing cholera by means of efficient sanitary engineering, and education in basic hygiene for the population. Nowadays, we in the developed world tend to think of cholera — somewhat smugly — as a disease that can only be found in hot, primitive places.

But only last century cholera epidemics used to sweep through cold British cities, and of course all music-lovers know of the untimely death of Tchaikowsky when he drank unboiled water (some say in a deliberate attempt to take his own life) during a cholera epidemic in Moscow in 1893.

Little comma-shaped bacteria, 2–4 μm long and called vibrios, cause the disease. Many different species of the genus *Vibrio* exist, divided into various strains or serotypes, not all of which can make us sick. *Vibrio cholerae* type 01 is the only cause of 'classical' full-blown cholera.

The bacteria produce a protein toxin, one part of which attaches to the cells that form the epithelial lining of the intestine. The other part of the toxin enters the cell, and activates an enzyme, stimulating it to function at a rate far higher than normal. The result is a vast secretion of water and various salts by the cells of the intestinal lining. Consequently, the victim suffers extreme diarrhoea, losing in that uncomfortable way up to 20 litres of fluid per day in severe cases. (Also some degree of vomiting is often present.)

The result of this, of course, is severe dehydration causing clinical shock and collapse. Furthermore, the loss through the diarrhoea of electrolytes (mineral ions vital in maintaining osmotic and ionic balance in the body) can, if sufficiently severe, affect many other aspects of physiology including the working of the heart muscle. So if the dehydration *per se* doesn't kill you, spasm of the heart may.

V. cholerae may be a natural part of the microflora of many Australian estuaries.

But nobody need die of cholera. Mild cases may clear up without treatment, while in severe cases careful replacement of lost fluids and electrolytes by an intravenous drip until the disease has run its course will usually keep the victim alive. But nevertheless, the debilitating effects and suffering mean that it is a disease best avoided.

Nowadays, most of us would not expect to find *Vibrio cholerae* bacteria in Australia. But in 1977, in Brisbane, doctors found *V. cholerae* in the stools of a patient who had not left the country. Painstaking investigation revealed that the patient had probably acquired the bacteria from contact with river water or foodstuffs contaminated with it, and upon investigation some rivers were indeed found to harbour the bacteria.

And in 1981, the New South Wales Department of Public Health, in response to a finding of *V. cholerae* on oysters grown in the Georges River near Sydney, briefly closed down the oyster-growing industry there.

Traditionally, the presence of *V. cholerae* has always been associated with human faecal pollution. Environmental microbiologists now believe that the old idea that cholera bacteria only appear following a major disruption in sewage disposal and water supplies is in need of revision. Although scientists had long known that water was the major vehicle for the transmission of cholera, most believed that the cholera vibrios did not persist for long in natural waterways, and that they only arrived there following recent contamination by infected people. (In a country where cholera bacteria were not thought to be endemic, any occurrence would be due to individuals infected overseas unwittingly bringing them in.)

Dr Michael Eyles, of CSIRO's Division of Food Processing, in collaboration with Mr George Davey and others of the New South Wales Department of Health, began a study in 1980 of what influences the presence in oyster-growing waters of *V. cholerae* and other *Vibrio* species.

During the 1970s, reports from overseas told of various serotypes of *V. cholerae*, but not the dangerous 01 types, being widespread in aquatic environments. Dr Eyles and his collaborators set to work sampling the Georges River estuary in Sydney and Brisbane Water near Gosford, which are the most important areas for the cultivation of the oyster *Crassostrea commercialis* in New South Wales, to see if they too harboured the vibrios.

Sampling at 3 sites every 2 weeks for a year, they detected *V. cholerae*, including some 01 strains, in oysters grown in the Georges River. This was the first finding

An oyster purification plant in New South Wales. Clean water is continually circulated through the racks.

of 01 strains in oyster-producing areas in New South Wales. They also found the bacteria in the water and sediment; one-fifth of the sampled oysters contained *V. cholerae*, 30% of the water samples did, and only 11% of the sediment ones did. The important questions were whether the bacteria represented a threat to health, and if their presence was the result of faecal contamination.

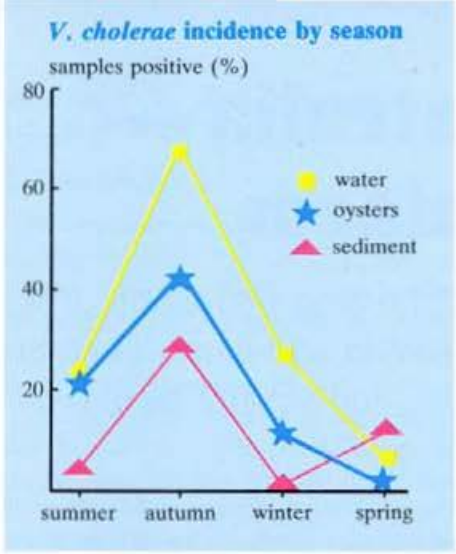
Firstly, detailed analysis revealed that most of the *V. cholerae* detected were strains other than 01. Although these can cause food poisoning, they cannot give rise to epidemic cholera. Analysis by the Enteric Pathogenicity Laboratory of La Trobe University of the 01 types that did turn up showed that they could not produce the crucial toxin; apparently, they did not possess the necessary genes for toxin manufacture.

Secondly, the vibrios seemed commoner in the warmer months, particularly early autumn. They were rare in the winter. By contrast, the numbers of *E. coli* — a normal inhabitant of the human gut that cannot live for long elsewhere — did not vary seasonally. Scientists have long known that the presence of *E. coli* is certain proof of faecal contamination. Numbers of *E. coli*, and of *Salmonella* species — common causes of food poisoning and of typhoid — increased with rainfall. The researchers expected this, because heavy rainfall causes urban run-off and sewage overflows, depositing faecal bacteria into the estuary.

Therefore, *V. cholerae* does not follow the behaviour expected of a faecal contaminant. The factors affecting the levels of *E. coli* and *V. cholerae* were clearly different. However, there was some connection; following an appearance and rise in numbers of *E. coli*, the *V. cholerae* counts also tended to rise. Dr Eyles and his colleagues believe this may be due to other factors, such as an increase in nutrients in the water following sewage overflow and run-off. The vibrios may benefit from the improved food supply and hence increase in number.

A natural part

It seems unlikely that faecal contamination was the source of the cholera bacteria. The scientists could find no reports in recent years of cholera infection in people in the region around the Georges River, and the fact that a mixture of strains were isolated at the same time is further evidence against contamination being the origin (infection is usually with just one strain). Besides, the scientists knew that the oysters had not been subject to serious sewage pollution in the days before they took their samples, because the *E. coli* counts on the cholera-positive oysters were low.



This graph shows the total incidence of *V. cholerae* at all sites in samples taken from oysters, river water, and sediment. Incidence varies with the season.

This work, taken with the results of other studies, indicates that *V. cholerae*, including some 01 strains, may well be a natural part of the microflora of many Australian estuaries, including the Georges River. The species apparently is part of the ecosystem, and as such may form a close association with other organisms, such as oysters. By contrast, *E. coli* or pathogenic gut bacteria — such as *Salmonella* and *Shigella* species — are adapted specifically for the human or mammalian body, and would only form a transient part of the river ecosystem. *V. cholerae* is probably only accidentally a human pathogen — causing cholera is perhaps not its normal way of life!

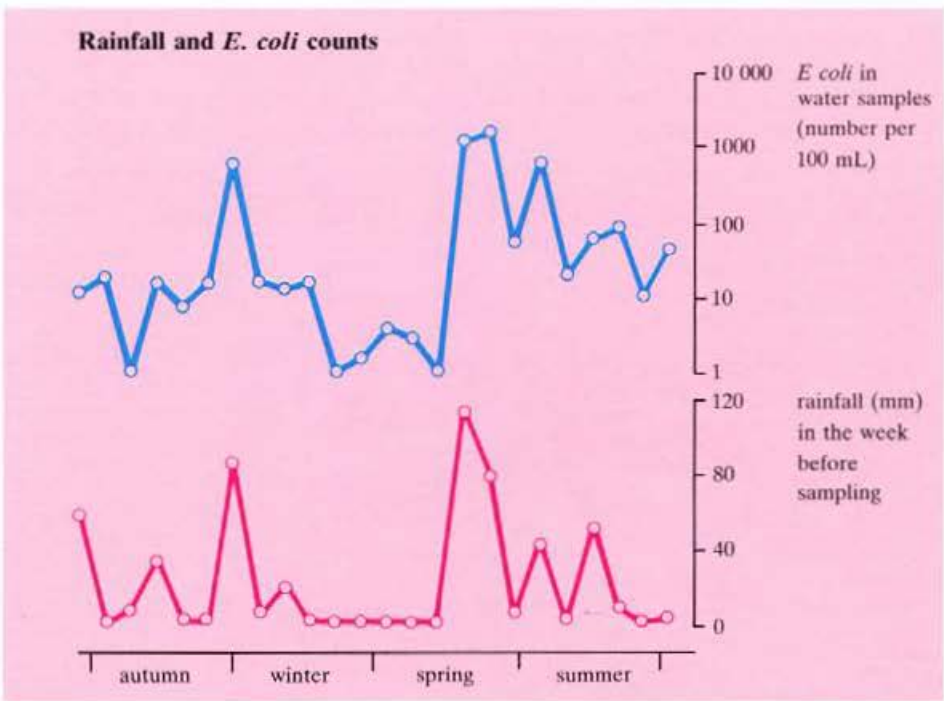
Don't worry

Now, if you find a few bits of chopped oyster in your spaghetti *alla marinara* you certainly won't be contracting cholera, because the bacterial toxin is destroyed by thorough cooking. If you take your oysters raw, you may like some further reassurance: all commercially grown oysters must undergo purification before they reach the consumer. The commonest means is by depuration.

In this procedure, the oysters are kept in clean water for 36 hours, and temperature, salinity, and dissolved oxygen are maintained at the correct level to induce the oysters to feed. This opens up their gut, and any bacteria or viruses in there are excreted. We know that, properly carried out, depuration effectively removes *E. coli* and other human gut bacteria from the oysters. But Dr Eyles' work has shown that it does not necessarily do so for *V. cholerae*.

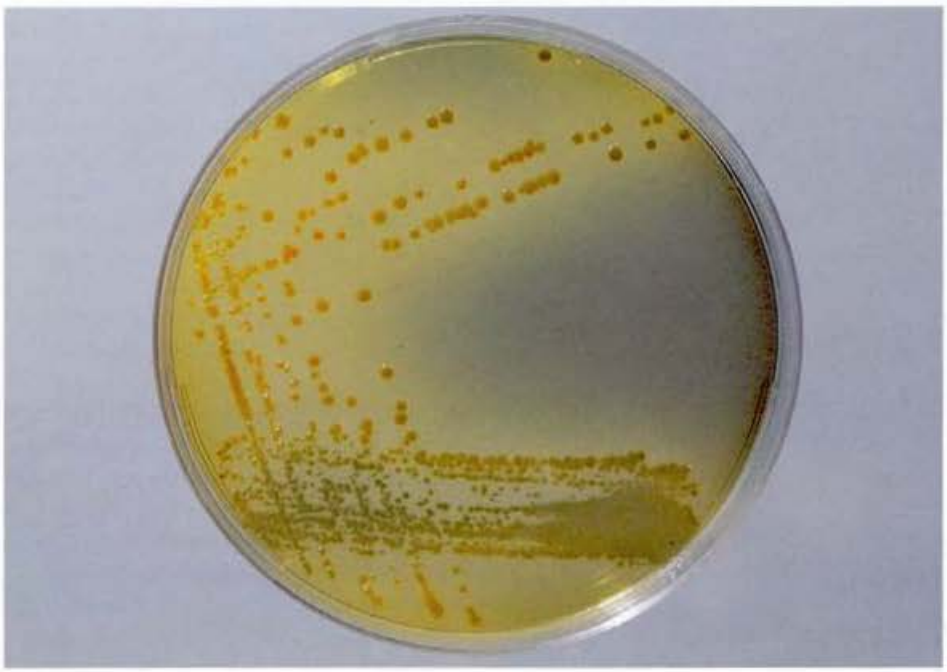
To understand the reason we must return to the ecosystem, where the naturally occurring *V. cholerae* may associate with the oysters, rather than just passing through them as other bacteria are. Because of this, normal depuration is not enough to remove them. It wasn't intended to get rid of the micro-organisms that are a normal part of an oyster's environment, although it may accomplish it in some cases. Dr Eyles feels that it may be unreasonable to expect purified oysters to be totally free of *V. cholerae*.

But you still needn't worry. The highest level of *V. cholerae* that the scientists found



A clear correlation between rainfall and *E. coli* counts is evident from data collected at a sampling site in the Georges River for a period of just over a year.

Collecting oysters for sampling from the Georges River estuary, near Sydney.



Colonies of *Vibrio cholerae* showing orange on a special type of medium, containing sucrose and a pH indicator. The *V. cholerae* bacteria possess the ability to ferment sucrose, producing acid that turns the indicator orange.

was three bacteria per gram of oyster. The infective dose for the full-blown disease — assuming the bacteria are of a strain capable of producing the toxin — is about 1–10 million bacteria, taken in total (of course, the figure would be less for somebody already sick or immunodeficient). That represents a quantity of oysters that would defeat even the most gluttonous gourmet. Other foodstuffs also contain small quantities of potential pathogens as part of their normal microbial flora, but hygienic food-handling and storage procedures that prevent their growth and spread minimise the risk of illness. The same should be true for oysters.

The fact that rivers on the Australian east coast may well contain small numbers of various *Vibrio* species and types should not be cause for alarm, as long as our excellent water supply and purification system remains operational. The public should not contaminate food with raw river water, and Australian doctors should consider testing for *V. cholerae* in the stools of patients with cholera-like symptoms occurring and with a history of recent exposure to river-water or food possibly contaminated by it. But to put the situation in proportion: between 1977 and 1984 scientists have only identified five incidents of cholera infection acquired from Queensland rivers. During that time, microbiologists in medical laboratories near the affected rivers in Queensland surveyed 72 000 faecal samples from other people with the likely symptoms without finding a single one positive for *V. cholerae*.

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More about the topic

Detection of *Vibrio cholerae* in oysters, water and sediment from the Georges River. G.R. Davey, J.K. Prendergast, and M.J. Eyles. *Food Technology in Australia*, 1982, **34**, 334–6.

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Investigation of cholera acquired from the riverine environment in Queensland. A.C. Bourke, Y.N. Cossins, B.R.W. Gray, T.J. Lunney, N.A. Rostron, R.V. Holmes, E.R. Griggs, D.J. Larsen, and V.R. Kelk. *The Medical Journal of Australia*, 1986, **144**, 229–34.