NEED ENVIRONMENTAL ASSESSMENT PROGRAM
ENVIRONMENTAL PROTECTION SERVICE
PACIFIC REGION

SANITARY SIGNIFICANCE OF NON-POINT POLLUTION ON SHELLFISH-GROWING WATERS

By

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1. INTRODUCTION

Bivalve molluscs are unique as food items in this hemisphere, as they are often eaten raw or partially cooked, with the entire animal being consumed, including the gastrointestinal tract and its contents. Because of their mode of feeding, bivalves can accumulate many substances which occur in their growing waters. If fecal contamination was present in the growing area, the potential exists for accumulating and concentrating pathogenic organisms. They are thus well fitted to serve as dispensers of human diseases with a fecal-oral route of spread.

The "bacteria age" was ushered in by Pasquier in 1818, who described an epidemic of illness totalling 17 cases, six diagnosed as typhoid, with two deaths (Tufts, unpublished report). The outbreak was linked to oysters that had been held in an old castle moat used for sewage disposal. More reports concerning shellfish and infectious diseases began to appear in medical literature through the 19th century. Dissemination and acceptance of the germ theory of disease gave health workers new weaponry. However, the role of shellfish in transmission of infectious microbes was not widely accepted and understood until the 20th century. As late as the 15-year period after World War I, 100,000 cases of typhoid fever occurred in France, with 25,000 deaths - all of which could be attributed to consumption of infectious bivalve shellfish. Reports since the early part of this century began to suggest that raw seafood may serve as an important vehicle in cholera epidemics. This has since been well established. The first recognized shellfish-associated epidemic of infectious hepatitis was identified in 1955 in Sweden. Since then many more outbreaks have been documented world-wide. (Tufts, unpublished report).

Following the shellfish-borne outbreak of typhoid fever during the 1924-1925 oyster harvest season in the United States, a committee report gave rise to what is presently known as the National Shellfish Sanitation Program (NSSP), (Hunt, 1977). Concerning a growing area microbiological standard, the report states "the committee is not prepared to recommend any precise bacterial standard for water ... until additional data ... have been assembled and are considered." Three significant steps in the development of an "approved" growing area microbiological standard were established : the method was standardized, the indicator group defined, and a standard recommended (Hunt, 1977).

The multiple tube fermentation method for the recovery of Bacillus coli and the relationship between the presence of B.coli and domestic sewage in receiving waters had been well established when Prescott and Winston published Elements of Water Bacteriology in 1904. Another paper published in 1904 by Eijkman noted that some organisms recovered in the test for B. coli were ubiquitous in the environment and not necessarily indicative of fecal contamination. He recommended a high incubation temperature of 46°C to inhibit growth of non-fecal types (Hunt, 1977).

These two publications provided the basic concepts which led to the development of NSSP "approved" growing area microbiological criteria and standards which are used in the United States, Canada, Japan, Korea, Mexico and perhaps other countries. The first standard was published in 1946 and defined as follows:

"The median bacteriological content of samples of water ... shall not show the presence of organisms of the coliform group in excess of 70/100 ml of water ..."

By 1965 the standard had been modified, and stated:

a) "The coliform median MPN of the water does not exceed 70/100 ml;

b) Not more than 10% of the samples ordinarily exceed an MPN of 230/100 ml for a 5-tube decimal dilution test or 330/100 ml where the 3-tube decimal dilution test is used, in those portions of the area most probably exposed to fecal contamination during the most unfavourable hydrographic and pollution conditions; and

c) These foregoing limits need not be applied if it can be shown by detailed study that the coliforms are not of fecal origin and do not indicate a public health hazard." (Hunt, 1977)

Further studies have concluded that the fecal coliform group is scientifically and logistically superior to the coliform or fecal streptococci indicator groups as a microbiological indicator of fecal pollution in estuarine waters. As an alternate standard it was proposed that:

"the median fecal coliform MPN value for a sampling station shall not exceed 14 per 100 ml of sample and not more than 10% of the samples shall exceed 43 for a 5-tube, 3 dilution test or 49 for a 3-tube, 3 dilution test."

At the present time, the NSSP accepts the total coliform or the fecal coliform criterion (Hunt, 1977).

Shellfish-growing water surveillance has traditionally focused on monitoring human fecal wastes. The most debilitating shellfish-borne infectious diseases - typhoid, cholera and hepatitis - are essentially specific to humans. Human fecal wastes for the most part are collected in centralized sewage treatment plants and released as a point source discharge.

Animals can transmit less severe infectious diseases to man via shellfish, e.g., salmonellosis and yersiniosis (Hubbert, 1975).

The present NSSP water quality standards provide a high degree of protection of shellfish food quality. However, at certain times shellfish-growing waters may show elevated indicator counts which exceed these strict standards. Investigations frequently have shown that non-point land surface run-off following storms is responsible for high indicator levels in receiving waters (Erkenbrecher, 1981; Faust, 1976). In the event that these peaks occur at a frequency to exceed the upper 10% limit of the growing water standard, shellfish areas are likely to closed.

The major objective of this paper, therefore, is to discuss the public health hazard due to non-point bacteriological pollution from land drainage - particularly with respect to shellfish-growing waters. This is a problem with many variables. It has no easy nor quantifiable solution.

The microbiological quality of land drainage varies significantly with the type of land use : urban, rural and hinterland.

Since there are a variety of shellfish-borne pathogens which can be transmitted to man, the etiology must be considered separately for each species.

Potential health hazards associated with shellfish consumption ideally should be evaluated in terms of documented incidence; however, most cases are not reported, and about 60% of all foodborne disease outbreaks reported are not identified etiologically (Centres for Disease Control, 1979). Incidence data on shellfish-borne diseases are presented as an appendix in this report.

2. TRANSMISSION ROUTES

Shellfish-borne infectious diseases are generally transmitted via a fecal-oral route. The pathway can become quite circuitous. The cycle usually begins with feces - man or animal, depending on the species of pathogen. Feces deposited on land surfaces can release pathogens into surface waters via run-off. Most freshwaters eventually empty into an estuary, where fecal bacteria and viruses may accumulate in sediment and subsequently be resuspended (Erkenbrecher, 1981). Microbes are removed from the water column by filter-feeding molluscs, and from the sediment by detritus-feeding molluscs. Molluscs concentrate micro-organisms, including those pathogenic, and humans can contract disease by eating such contaminated shellfish. Some pathogens are capable of persisting in terrestrial soil, fresh and marine waters, and aquatic sediment (Erkenbrecher, 1981; Imperato, 1974; Kampelmacher, 1977). Some are even capable of growth external to a host (Erkenbrecher, 1981).

3. SHELLFISH-BORNE PATHOGENS

This section briefly introduces pathogenic enteric bacteria and viruses which may be transmitted by shellfish as a result of filter-feeding accumulation. The agents responsible for the more severe illnesses seem to be well established. On the other hand, acute cases of diarrhea frequently are diagnosed as "gastroenteritis", a non-specific term which may be due to Salmonella, Shigella, Campylobacter or enteric viruses such as Norwalk virus. Isolation techniques are constantly developing and detecting previously undiagnosed agents, e.g., Norwalk virus (Gunn, 1982). Also strains of micro-organisms do change over time and vary in pathogenic effects. For example, Campylobacter has long been known to cause disease in animals but only recently has it emerged as a common cause of enteritis in humans (Christie, 1980).

- Salmonella typhi - Typhoid Fever

Typhoid fever is characterized by continued fever, constipation, slow pulse and involvement of the lymphoid tissues. The fatality rate may be 10% but antibiotics can reduce this to 2% to 3%.

Man is essentially the only reservoir (except possibly fruit-eating bats). (Christie, 1980).

Transmission occurs through either direct or indirect contact with the feces or urine of a case or carrier. Flies can transport the bacilli (Imperato 1974).

The organism can exist for long periods of time in water and ice and frozen ground (Imperato, 1974). Seawater is rapidly bactericidal to S. typhi (Christie, 1980). However there have been numerous outbreaks of typhoid fever attributed to eating contaminated shellfish (see Appendix). Experimentally, S. typhi has survived in infected oysters for as long as four weeks (Christie, 1980).

- Salmonella - Paratyphoid Fever

Paratyphoid fever is caused by S. paratyphi, S. schottmulleri and S. hirschfeldi. The organisms are generally obligatory human parasites, but they have also been found in lower animals - except for S. paratyphi (Imperato, 1974). The disease simulates typhoid fever, but the symptoms are less severe.

S. paratyphi B has been found to survive and reproduce in the marine environment (Buttiaux,). Paratyphoid fever has rarely if ever been reported to be transmitted by shellfish (Christie, 1980). However, the clinical features may be indistinguishable from the enteritis due to any other member of the Salmonella group (Christie, 1980).

- *Salmonella* spp. - Salmonellosis

Salmonellosis is a generic term for a spectrum of illnesses induced by certain species of *Salmonella*. Typhoid fever, paratyphoid fever and salmonella enteritis are types of salmonellosis. In theory, every *Salmonella* strain is capable of producing enteric fever, localized infections or enteritis. Salmonella enteritis is an acute gastroenteritis characterized by abdominal cramps, nausea, vomiting, diarrhea and fever (Imperato, 1974).

There are more than 1,400 different *Salmonella* types and more are constantly being described (Christie, 1980). They may be found in mammals, birds, soil and water. Swine, cattle and poultry can harbour *Salmonella* and transmission may be via meat, eggs and other animal products or by direct contact (Dauer et al., 1968, Imperato, 1974). Because *Salmonella* infect domestic animals and lead to contaminated food products, salmonellosis is the most important bacterial zoonosis in the United States (Imperato, 1974). *Salmonella* do enter fresh surface waters via untreated sewage. It has been concluded that *Salmonella* infections are maintained by cycles in which the polluted environment, and especially contaminated surface waters, play a major role (Kampelmacher, 1977).

Marine sediment eluate studies have shown growth of *Salmonella* (Erkenbrecher, 1981). Shellfish from polluted waters may lead to salmonellosis (Christie, 1980). A recently compiled record of shellfish-borne disease outbreaks in North America since 1900 (Verber, unpublished report) seldom notes *Salmonella* (other than typhoid), but "gastroenteritis" is often listed.

- Vibrio cholerae - Cholera

Cholera is an acute intestinal disease of man characterized by sudden onset, vomiting, profuse watery diarrhea, rapid dehydration with the development of acidosis and circulatory collapse. The overall fatality rate in untreated cases is about 50% (Imperato, 1974).

Man is the only known host and though infection spreads through a contaminated environment it is only man who contaminates that environment. Its presence in delta waters in endemic areas depends on constant recontamination with excreta, for the vibrio is soon overgrown by other micro-organisms. V. cholerae has been isolated from sewage-polluted sea or lagoon water. Cockles, mussels, oysters and the like have been the source of many outbreaks (Christie, 1980).

- Vibrio parahaemolyticus

Vibrio gastroenteritis had been reported exclusively from Japan till the late 1960's. The causative organism, Vibrio parahaemolyticus, seems to be present in seawaters around the world (Christie, 1980). It can be isolated from finfish, shellfish, crustaceans and organic materials in marine sediment. Infection in man is associated with consumption of contaminated sea foods eaten raw. It is unknown whether or not V. parahaemolyticus is transmitted by man (Hubbert et al., 1975).

Symptoms characteristically are severe abdominal pains, vomiting and diarrhea.

- Viral Hepatitis

Viral hepattitis is an acute infectious disease with fever, nausea, malaise and abdominal discomfort followed by jaundice. Convalescence may be prolonged for many weeks. In adults the disease is usually more severe.

The reservoir is man and certain non-human primates.

Transmission is by person-to-person contact and indirectly via the fecal-oral route. There have been many reports of food and water-borne outbreaks. Shellfish have frequently transmitted hepatitis (see Appendix).

Field and laboratory studies have indicated that enteric viruses can survive for long periods (months) in seawater and in shellfish, thus increasing the risk of infection (Gerba and Goyal, 1978).

- Escherichia coli

Research into the role of E. coli as a cause of gastroenteritis seems to establish the following points:

1) that only a few strains cause gastroenteritis,

2) that the illness occurs most often in infants,

3) that infection, as distinct from illness, is very common, and

4) that pathogenic strains vary over short periods of time in their ability to cause disease. (Christie, 1980).

The severity of E. coli gastroenteritis varies from time-to-time and from place-to-place. It can be fatal to infants (Christie, 1980).

Strains of E. coli have been isolated from mammals and birds such as cattle, dogs and chickens, but there is little evidence that infants ever derive infection other than directly or indirectly from other infants (Christie, 1980). Transmission is via the fecal-oral route. Airborne transmission may also occur (Imperato, 1974).

Outside the body, E. coli may survive for weeks or months in fresh water. In feces at $0^{o}C$ it survives for a year or more (Christie, 1980). Marine sediment eluate studies have shown growth of E. coli (Erkenbrecher, 1981).

The role of shellfish in transmitting this pathogen is uncertain. A recent compilation of shellfish-borne disease outbreaks in North America (Verber, unpublished report), does not list this organism, but it might account for some of the cases of "gastroenteritis" listed.

- Shigella

Shigellosis, also known as bacillary dysentery, is an acute bacterial disease of the intestinal tract characterized by abdominal pain, tenesmus and diarrhea (Imperato, 1974). The principal pathogenic species are Sh. dysenteriae, Sh. flexneri, Sh. boydii, and Sh. sonnei. Shigellae are pathogens almost exclusively of man and captive non-human primates (Christie, 1980).

Transmission is via the fecal-oral route through personal contact and contaminated water and food.

The organism can survive in seawater perhaps for at least 15 days (Christie, 1980). Under suitable conditions, Shigella can grow readily in food. Shigella may survive and grow in marine sediments (Erkenbrecher, 1981). Shellfish have transmitted shigellosis (Centres for Disease Control, 1977, 1979).

- Campylobacter

Species of this bacterial genus are widespread in the animal kingdom both as saprophytes and pathogens. They have been known to infect domestic and wild animals and birds for many years. In 1947, the disease was recognized in association with non-enteric human illnesses. Recently, because of improved isolution techniques, correlations were made between certain Campylobacter species and human infectious diarrhea. The epidemiology and pathophysiology of diarrhea caused by these species is not completely understood. Poultry, raw milk, contaminated drinking water and molluscan shellfish have been implicated as reservoirs for human campylobacteriosis. Only C. fetus subs. intestinalis and jejuni appear to infect humans. The enteric form of campylobacteriosis resembles bacillary dysentry. The first reported outbreak of Campylobacter diarrhea in the U.S. took place in 1978. There was a strong correlation between the illness of 2,000 townpeople and consumption of the local water supply. Recent studies in Sweden confirmed that Campylobacter is more common than Salmonella as a cause of bacterial diarrhea during summer or late fall. In 1978, the number of Campylobacter isolates in the United Kingdom exceeded the number of Salmonella isolates. In the Swedish studies there were no reports of secondary cases with Campylobacter transmission from person-to-person as there normally is with Salmonella. Campylobacter has been isolated from seawater in England. In 1980 hard clams harvested from either North Carolina or New York were strongly associated with cases of Campylobacter gastroenteritis in New Jersey. There were 18 cases. (All of the foregoing information is taken from Miescier, unpublished report).

A recent compilation of shellfish-borne disease outbreak data does not indicate any other incidents of shellfish-borne

Campylobacter in North America (Verber, unpublished report). Since wild and domestic animals and birds have been implicated as reservoirs of Campylobacter, contaminated waters from non-point drainage may be an important factor in transmission to man. Campylobacter can be expected to occur more frequently in the near future in North America.

- Yersinia enterocolitica

Yersinia enterocolitica, a bacterium, was first recognized as a distinct species in 1964. Reservoirs besides man include rodents and wild birds (Hubbert, 1975) and such domestic animals as swine, cattle and dogs (Christie, 1980). Transmission in the Pacific Northwest seems to be waterborne (Wetzler et al., unpublished report). A survey in Washington State found Yersinia in every type of water supply regardless of the treatment methods employed, and persistence for some unknown time in chlorinated systems (Wetzler et al., unpublished report). Yersinia has been isolated from mussels and oysters (Christie, 1980). Yersinia is probably not a major cause of foodborne disease but the true incidence of infection is not yet known (Christie, 1980). A recently compiled list of shellfish-borne disease outbreaks in North America does not indicate any cases due to Yersinia. (Verber, unpublished report).

The most common sympton of this pathogen is diarrhea with fever and abdominal pain. Most of the cases have been sporadic or in small family groups.

- Norwalk Virus Gastroenteritis

Gunn (1982) has described the first documented outbreak of shellfish-associated Norwalk virus gastroenteritis in the USA (January, 1980). Six of eleven persons at a party where raw oysters were consumed developed symptoms, but required no specific treatment. The revised estimate of a similar outbreak in Australia is at least 7,000 cases, with possibly as many as 15,000 cases (Metcalf, 1982).

A reliable radio-immunoassay for detection of Norwalk virus antigen and antibody has only recently become available (Gunn, 1982).

A recent compilation of shellfish-borne disease outbreaks in North America (Verber, unpublished report) does not indicate any other cases of Norwalk virus.

Symptoms are principally nausea, vomiting and diarrhea of limited duration.

4. NON-POINT SOURCES OF BACTERIOLOGICAL POLLUTION OF WATERSHEDS

Human feces are the most hazardous source of pathogenic microbes which can be transmitted by shellfish, since the most serious pathogens are essentially specific to man, i.e., hepatitis, cholera and typhoid. Priority has been given to managing human fecal wastes by centralizing collection and treatment. Discharge is then released as a point source.

Non-point fecal contamination arises, in part, from surface run-off on land exposed to fecal deposition - primarily animal. The type of land use is a key factor relating to microbial quality of surface run-off. Weather - especially precipitation regimes, soil texture, soil permeability and saturation, water-table level and slope are other factors. For purposes of discussion, land use may be classified as:

- urban,
- arable land - with or without manure fertilization,
- pasture and rangeland, and
- hinterlands.

4.1 Urban Non-Point Bacteriological Pollution

Numerous studies have shown that urban stormwater can be heavily polluted by micro-organisms - many of fecal origin. For example, the Environmental Protection Service (1978) studied the effect of urban storm drainage inputs to the lower Rideau River in Ottawa. Results showed sharp, transient increases in fecal pollutant concentrations in response to increased run-off. No major inputs of sanitary wastes were found in the study area.

It was inferred that bacteria in urban stormwater were primarily non-human, and derived mainly from rodents, cats, dogs and birds.

A study conducted jointly by the Ontario Ministry of Environment and Environment Canada (Qureshi and Dutka, 1979) investigated the microbiological quality of urban stormwater run-off at three different locations in Southern Ontario. Discharges from all three stormsewer systems contained significant quantities of pollution indicator bacteria. Among those pathogenic, Pseudomonas aeruginosa was most numerous and frequently isolated. (However, pseudomonas does not appear to be associated with shellfish-borne disease). Salmonellae were detected frequently in stormwater run-off, with no predictable pattern. Microbial populations in stormwater run-off were strikingly high throughout the entire sampling period and many times approached densities found in dilute raw wastewaters and, therefore, were concluded to constitute a health hazard. The implied public health risk was substantiated by the recovery of potentially pathogenic bacteria in discharges from the sites studied. In evaluating health risk however, infectious dose must be considered - not just isolation.

Olivieri (1980) studied the presence and levels of selected indicator and pathogenic micro-organisms, and the relationship between natural populations of these microbes in urban run-off. Most of the data was collected in Baltimore, Maryland. The relative order of the levels of pathogens in the urban streams was Pseudomonas aeruginosa > Staphylococcus aureus > enterovirus > Salmonella spp. The levels of total coliforms, fecal coliforms fecal streptococci (henceforth TC, FC and FS respectively) and enterococci suggested that the run-off at each location was heavily contaminated and from a microbiological standpoint was of

poor quality. The densities of indicator micro-organisms found in storm run-off were generally about 10-fold higher than found in urban streams and approached the indicator densities of raw sewage. The levels of indicators in storm run-off were several orders of magnitude above those found in the municipal reservoir samples. The stormwater levels of enteroviruses, Salmonella spp., P. aeruginosa and S. aureus were several-fold higher than those found in the urban streams, but significantly lower than the levels in raw sewage.

Discussing sources of micro-organisms, Olivieri notes that urban populations of dogs and cats are high. Rodent populations are particularly high in the densely populated areas in the inner city (Baltimore). The feces of large populations of animals in a confined area must represent a continual source of micro-organisms to the aquatic environment.

While great effort is expended in the modern city to collect and treat human feces and waste, a significant quantity of sanitary sewage finds its way into urban water courses. Sewer malfunction, intentional overflows and bypasses, poor sewer maintenance and inadequate collection and treatment systems all contribute to the high levels of indicator and pathogenic microbes. Olivieri cited a study of pollution in the urban streams in Baltimore (with separate sanitary and storm sewer systems) and Washington D.C. (with combined sanitary and storm sewers) which found that the largest cause of poor water quality in both cities was sewer malfunction. In Olivieri's study, the presence and levels of enteric viruses, particularly poliovirus, indicated that there was a considerable human fecal component in the urban run-off.

4.2 Rural Non-Point Bacteriological Pollution

Productive rural areas tend to form a patchwork of arable croplands, pastures and hayfields, and woodlots. Unproductive land may be used primarily as rangeland and forestland. Many studies have shown rural run-off to be heavily contaminated with fecal micro-organisms (Faust, 1976; Kunkle, 1970; Hendry & Toth, 1982). Domestic animals are a dominant source of fecal indicators and potential pathogens. Stormwater run-off is a highly significant factor in flushing these microbes from soil surfaces into surface waters.

Arable land is normally fertilized with either inorganic chemical fertilizer, or with manure. Manure may be applied as a solid or as a liquid slurry.

The Research Branch of Agriculture Canada (Culley and Phillips, 1982) studied the bacteriological quality of surface and sub-surface run-off from manured sandy clay loam soil. For six years, test plots of continuous-crop corn were treated with three differing concentrations of liquid dairy manure, or chemical fertilizer, or untreated. Treatments were applied after the fall harvest, before spring planting, or both, or during the winter while the surface was frozen or snow-covered. Bacteriological parameters were monitored during the spring especially. In Eastern Ontario, where the study took place, over 50% of the total annual run-off from two of the major agricultural catchments occurs during the snowmelt period.

Bacterial counts in snowmelt surface run-off were significantly affected by time but not rate of fertilization. Application rates averaged 105, 246 and 420 $m^3\ ha^{-1}\ year^{-1}$ - differing by less than one order of magnitude. The winter application

produced the highest indicator counts (337 TC/100 ml, 7 FC, 2442 FS). May-June stormwater run-off occurred only twice in the six-year period. During the June event, the FC counts rose 5,000 times above the snowmelt counts. This was partly due to much higher sediment concentrations and a lower dilution factor than snowmelt surface run-off. Counts of FC and FS from the June event were even higher on the chemically fertilized control plot than the mean values of plots receiving manure! (No explanation).

As with surface run-off, time but not rate of liquid dairy manure application significantly affected FS densities in sub-surface tile drainage. However, differences in both TC and FC counts in sub-surface drainage could not be related to treatment. Differential die-off of TC, FC and FS was probably a factor. Sub-surface drainage waters from non-winter treatments generally contained lower indicator densities than did surface run-off. Substantial numbers of manure FS survived winter weather conditions and were able to move through the soil profile to drains about 75 cm deep. However, survival of TC and FC in surface-applied frozen manure declined to low levels within 40 days (Culley and Phillips, 1982).

Other studies (Faust, 1982) have shown that indicator bacteria do not penetrate the soil profile. Key factors are probably soil texture and permeability, and soil saturation. In Culley's study, the sandy clay loam was probably permeable and almost certainly saturated during the spring snowmelt period.

Faust (1976) studied the coliform pollution levels in surface run-off from five sub-watershed basins of a sub-estuary of Chesapeake Bay. Each sub-watershed was a mixture of cultivated land, marsh, forest, pasture and residential area. This study

was not designed to analyze coliform pollution in terms of land use.

Discharge of FC levels varied from (0.02 to 1329)x10^6 FC animal-day^{-1}, with differences in stream flows of six log units. For all streams combined, the lowest measured FC discharge was 7.5x10^6 ha-day^{-1} and the highest was 669x10^6 ha-day^{-1}. It appeared that the relatively high FC levels present in the river originated from fecal material deposited on the watershed and transported into the river by water run-off. The decrease in numbers (concentration) of TC and FC was inversely related to increasing volume of the river. Area-yield FC loading rates from the watershed to the estuary appeared to depend on the rate of water run-off. Little or no correlation existed between rainfall and water discharge, and between FC discharge levels and rainfall. Soil saturation (undiscussed) could have been an important factor. No estimate of the effect of wild animals in the area was available. Fecal coliform concentrations collected in surface water samples varied three log units at a given station. Even with similar flow rates at a given station, FC discharge levels varied by two log units. Season may be a factor, as well as patterns in livestock presence. Differences in water flow could not explain the differences found in FC discharge between stations. For example, one day there was a 3-fold difference in water flow between two water sites, but a 26-fold difference in FC discharge. (1.060x10^6 L/day & 117,216 x10^6 FC/day, 0.330x10^6 L/day & 3,643x10^6 FC/day).

On the average, about 1% of the FC produced by cattle were washed off into the watershed. Based on the safety standard of 14 FC MPN 100 ml^{-1} for shellfish-growing water quality, the receiving capacity could be exceeded during periods of high FC discharge and mean low water.

Faust (1982) later examined the relationship between land use practices and fecal bacteria in soils. Land uses analyzed were row crop (corn), pasture and woodlot. Soil surface samples and cores were taken for bacteriological identification and enumeration, and compared seasonally. The study site was a portion of a watershed which drains into Chesapeake Bay.

Pasture sample stations had bacterial levels which generally declined with depth, except for total streptococci. Fecal coliforms varied seasonally, probably a function of survival based on temperature and moisture, and presence of grazing animals. Fecal coliforms were highest in the surface soil layers and minimal below 7 cm., except in fall. Concentrations of FS appeared to follow a similar pattern to FC levels. Aerobic heterotrophic bacteria showed more uniform seasonal concentrations. Cell counts declined somewhat with depth. As expected, the concentrations of FC and FS were much less than that of aerobic heterotrophic bacteria.

Stations sampled from cornfield sites showed that 77% of coliforms were Enterobacter aerogenes and only 8% were Escherichia coli. This indicated that coliform bacteria were primarily not of fecal origin.

In forest soils, FC were present in comparatively low numbers.

Based on this study, Faust concluded that:

1) land use practices can contribute significantly different amounts of FC bacteria to surface run-off waters,

2) that FC are discharged from the soil surface rather than groundwater, and

3) that FC remain viable for a considerable time in soils, especially at low temperatures.

Hendry and Toth (1982) surveyed the bacteriological water quality of a lake impacted by different land uses in Southern Ontario. It was shallow, entrophic, 23.5 km^2 and used primarily for summer recreation. Much of the shoreline was developed with 800 cottages; the remainder was mostly farmland - with several cattle watering sites, and a bird sanctuary. There were no direct inputs of municipal or industrial effluents to the lake.

The lake was intensively sampled during two 5-day periods in May-June and July. Impairment of water quality was limited to shoreline locations near suspected sources. Impairment was regarded as an elevated FC geometric mean density relative to that of the main body of water surrounding that location. After a rainstorm event, a 10-fold increase in FC and FS levels was found in all lake surfaces. This effect lasted only 2-3 days. Mouths of rural inflowing streams were the commonest source of impairment, followed by shoreline at the bird sanctuary. None of the cattle watering sites were positive for Pseudomonas aeruginosa, although beach sites were. Fecal bacteria originated from cattle watering sites, farms, public beaches, some flooded and substandard septic tanks, wildlife at a bird sanctuary, and rural stormwater from the inflowing streams. Mid-lake stations had considerably lower numbers of fecal indicators than nearby shoreline stations - probably due to die-off and dilution.

Kunkle (1970) experimented with farm surface run-off in relation to the hydrologic regime and to the background levels of indicator bacteria from a manure-free field. The 0.75 km^2 watershed, in Vermont, contained 37% hayfields, 38% pasture and 25% forested

land. The 0.2 ha control plot was in a hayfield free from recent manure application and domestic animals. Natural storms and sprinkler irrigation events were monitored. Surface run-off samples were collected from flumes, and stream samples from a wier.

During periods of storm run-off from the watershed, concentrations of TC and FC rose drastically, with distribution of the values closely related to the hydrography (unlike Faust, 1976). Over 90% of the storm run-off observations contained higher concentrations of FC and FS than is allowed for swimming waters (200 FC/100 ml). Concentrations of TC in run-off from the hayfield were similar to the partially grazed watershed. However, FC concentrations were far greater in run-off from the grazed area than from the hayfield plot, where FC levels were usually less than 5% of TC levels. During all seasons, the range of TC and FC levels was far lower than observed during storm events. Kunkle concluded that TC were of questionable value as indicators of pollution, and that FC were much more specific.

4.3 Hinterland Non-Point Bacteriological Pollution

Very little information regarding microbial water quality from hinterlands was found. Most papers obtained dealing with shellfish-growing waters were of American origin. Of these, the majority of studies were conducted on the East Coast - which is heavily populated and developed. The Gulf Coast is also an important shellfish-producing region. It has extensive tidal marshes and bayous which are often uninhabited by man and domestic animals. Nevertheless, many Gulf Coast shellfish-growing areas cannot meet the bacteriological standards. Sanitary surveys in some areas have shown that high TC and FC levels are not due to humans or domestic animals.

Presnell (1971) collected soil, water and vertebrate samples in a bayou in Mississippi and analyzed them for TC, FC and Salmonella spp. Wild mammals and birds were the most likely sources of FC from water and soil samples at "unpolluted" stations. Salmonellae were isolated from the feces of one racoon, one rail (Rallus sp.) and from soil frequented by wild animals. On the basis of current coliform standards, all the "unpolluted" waters would have been classed as prohibited. In general, the bacterial quality of the water entering the bayous was better than that of the bayous, indicating bacteriological pollution by local wildlife.

To what extent data based on studies of the warm, stagnant Gulf Coast can be extrapolated to conditions on the North Pacific Coast is questionable.

Information on microbiological water quality from drainage of extensive forest lands, such as exist in coastal British Columbia, was not found.

5. ENTERIC BACTERIA IN MARINE RECEIVING WATERS AND SEDIMENT

While there are numerous studies dealing with bacteriological pollution from urban and agricultural drainage, the health significance to man is generally only inferred by the presence of indicators, or less often, pathogenic microbes. Most investigations of non-point pollution do not follow through with the impact on marine receiving waters, and shellfish quality in particular. A review of some factors in estuary ecology and microbe-shellfish interactions is required to evaluate the significance of land surface run-off with respect to shellfish quality.

Survival and possible growth of enteric bacteria and viruses in marine waters, sediment and shellfish are the major factors in evaluating the significance of bacteriologically contaminated freshwater input, especially in the case where high indicator levels are only observed after storms. One would hope that a rapid die-off would follow.

Not all potentially pathogenic enteric bacteria in coastal waters are derived from freshwater drainage. Waterbirds are often abundant during the winter on sheltered coastal waters. Birds, like mammals, are sources of indicator bacteria and potential pathogens. Hussong (1979) investigated the microbial impact of Canada Geese and Whistling Swans in Chesapeake Bay. His objective was to obtain qualitative and quantitative measures of goose and swan bacterial flora, and to determine their fate. Fresh fecal material was collected from autopsied birds and from fed captives. Stations were sampled for water and sediment bacteria and compared with calculated values based on bird-days.

No birds collected yielded Salmonella spp., nor could any be detected from roosting sites. Most of the facultatively anaerobic bacteria

isolated were Escherichia and Streptococcus spp. The ratio of FC to FS varied from bird to bird. Results suggested that it is not possible to separate waterfowl fecal contamination from human on the basis of FC:FS ratios. Clostridium botulinum was routinely isolated from waterfowl, but no C. tetani. After 220 bird-hours, the FC MPN count rose from 1 FC/100 ml in surface water to 2,400/100 ml in a saltwater pond. Sediment FC counts rose 2 log units. Neither Salmonella nor Shigella could be isolated. The FC die-off rate was estimated to be on average 24 hours. The major conclusion of this research was that healthy swans and geese on the Northeast flyway did not harbour detectable pathogenic enteric bacteria.

Gould (1977) studied the fecal flora of British seagulls (Larus spp.). Nine individuals representing four species were individually caged and fed for three weeks. Their feces were collected for analysis. Besides the standard indicator organisms (TC, FC, FS), Clostridium welchii (C. perfringens) and Salmonella sp. were assayed.

Ratios of FC/FS were inconsistent and showed large inter- and intra-specific variations. C. welchii was detected in comparatively low numbers, but Salmonella sp. was not. Considerable evidence does exist, however, of the presence of Salmonella sp. in gull droppings (Gould, 1977). The daily loads of fecal coliforms for Larus argentatus and L. fuscus were comparable to those of man. Hence concentrations of gulls in shellfish-growing waters might result in high levels of indicator organisms in the water column.

The role of sediment in marine waters is of concern. It serves as a potential reservoir of both enteric viruses and bacteria.

Erkenbrecher (1981) has published one of the few detailed investigations of indicator bacteria in estuarine sediments. Eleven sub-tidal stations were selected in an urbanized sub-estuary of Chesapeake Bay.

Water and sediment samples were collected at least bi-monthly for one year. Counts were calculated on a volumetric basis to compare the water column to the sediment.

The density of sediment bacteria averaged two to several log units higher than in the water column. Concentrations of total suspended solids were associated with peak densities of FS and TC, which are known to be associated with plants and soils, but not with FC densities. Fecal coliforms can desorb from benthic sediments under conditions of reduced salinity. This conclusion was supported by a strong correlation between water column and sediment FC levels. Densities of all indicator bacteria were always significantly higher in the sediments than in the overlying water column.

Sediments have been shown to provide a suitable environment for the survival and growth of some indicator bacteria, due in part to an increase in organics in sediments over those in the water column. Sediment eluate studies have shown growth of E. coli, Enterobacter aerogens, and the pathogenic bacteria Shigella, Salmonella, and Arizona (Erkenbrecher, 1981).

The dominant factor in peak aquatic densities was heavy rainfall associated with a concomitant increase in storm water run-off during the 24-hour period before sampling.

The major problems in the estuary were non-point in origin - especially septic tanks and drainage fields unable to cope with the high water-table and heavy rainfall typical of the area.

Fecal indicator organisms, rather than specific potential pathogens, are most commonly studied in estuaries. Sayler et al. (1975) undertook an investigation in Upper Chesapeake Bay to determine the incidence of selected bacterial species considered to be potentially pathogenic, and

the relationship of their occurrence with indicators. Salmonella spp., Clostridium botulinum and Vibrio parahaemolyticus were selected for study. These potential pathogens are known to occur in the aquatic environment. Shellfish were assayed for pathogens.

It was concluded that these species occur at a very low frequency in the water and sediment of Upper Chesapeake Bay, in spite of the fact that the bay contains a large number of fecal indicator organisms. However, only Salmonella is associated with sewage - not C. botulinum and V. parahaemolyticus. There was a higher incidence of successful isolation of Salmonella spp. in areas of high TC and FC densities.

The low frequency of V. parahaemolyticus was concluded to be related to the low salinity of the upper bay. Clostridium spp. possess a relatively high tolerance to salinity, as well as an over-wintering capability in the natural environment. Hussong's (1979) later research showed that, at least in sediments of a freshwater pond densely populated by geese, Clostridium spp. can be abundant. Nevertheless, the incidence of botulism in humans in the U.S.A. is extremely low (Sayler et al., 1976).

Oysters were found to be free of the pathogens assayed, and did not exceed the allowable limit for FC MPN counts.

6. ENTERIC VIRUSES IN MARINE RECEIVING WATERS AND SEDIMENT

Development of a polyelectrode method allowed De Flora et al. (1975) to isolate animal viruses from nearshore shallow waters and clastic marine sediments. Most viruses isolated could be classified as enteroviruses, including some strains of vaccine-like poliovirus. Appreciable amounts of virus could be eluted from sediments after simple mechanical shaking.

Transmission of viral disease by shellfish has been less documented and studied than enteric bacterial disease. Viruses may remain infectious for several weeks or longer after discharge into receiving waters, allowing sufficient time for them to be transported to shellfish-growing waters. When ingested by shellfish, their survival rate appears to be further prolonged (Gerba and Goyal, 1978).

Infectious hepatitis has been well established to be transmitted by shellfish since the first record in 1955. Even in non-epidemic times, a relationship between consumption of shellfish and occurrence of hospitalized cases of infectious hepatitis is known to exist, indicating that the true incidence of shellfish-associated viral disease is probably greatly underestimated (Gerba and Goyal, 1978).

An outbreak of hepatitis attributed to flooding of the contaminated Mississippi River on coastal oyster beds indicated that the virus remained viable for 1-2 months after flooding (and harvest closure), and at levels sufficient to cause disease (Gerba and Goyal, 1978).

A study of non-epidemic infectious hepatitis cases among patients in 10 Boston hospitals concluded that, when considered together, ingestion of steamed clams or raw shellfish was as frequent a potential exposure to hepatitis as was contact with jaundiced persons (Gerba and Goyal, 1978).

Examination of only the water in shellfish-growing waters cannot be used as an indication of the presence of viruses. Viruses can be retained by shellfish even after sewage pollution is no longer evident in the area (Gerba and Goyal, 1978).

No enteric virus multiplication has ever been demonstrated in shellfish. They are only concentrated (Gerba and Goyal, 1978).

Field and laboratory studies have demonstrated that enteric viruses can survive from a few days to over 130 days in marine waters. Survival becomes greatly prolonged once they become associated with sediments, and their concentration may be much greater than in the overlying water column. Several good reviews on virus survival in marine water are available (Gerba and Goyal, 1978).

There is increasing evidence that those enteric viruses which cannot be recovered conveniently in some host cell system may occur more often in polluted growing waters than originally thought. This is especially true for Norwalk-type and hepatitis A viruses. Special methods for their detection make routine examinations impractical. There is no conclusive evidence that the culturable enteric viruses are responsible for shellfish-associated illness (e.g., polio virus). Public health agencies and epidemiologic surveillance methods have frequently failed to recognize the existence of some infections and the etiologic role played by shellfish-transmitted enteric viruses. All of the examples of unrecognized shellfish-associated illnesses detected retrospectively have involved non-culturable enteric viruses. Thus, there is reason to believe that the incidence of shellfish-borne viral disease has been grossly underestimated (Metcalf, 1982).

7. CONCLUSIONS

Most shellfish-borne diseases apparently are due to human fecal contamination. Direct contact with infected persons is a greater risk for contracting cholera, typhoid fever and perhaps infectious hepatitis, than is shellfish consumption. These are the three most serious diseases, and animals are not reservoirs.

Mammals and birds and humans can carry pathogenic Salmonella, Campylobacter, and Yersinia - other concerns to public health.

Shigella, Salmonella, Vibrio and hepatitis A virus can persist for some time in the external environment, and they can be transmitted by shellfish.

Non-human fecal contamination is clearly less hazardous to public health. Thus, bacteriological water quality standards, which are based on point sources of human sewage, may be overly conservative in cases where fecal pollution is non-human. Enteric bacterial pathogens transmitted by animals to man usually result in relatively mild gastroenteritis. Mild foodborne infectious outbreaks are often not reported to public health authorities. Outbreaks involving Escherichia coli, Yersinia enterocolitica and Campylobacter fetus ssp. jejuni are often not considered in clinical, epidemiologic and laboratory investigations (CDC, 1979). Thus the true incidence of "gastroenteritis" is unknown and the etiology often undiagnosed. Therefore a "safe" standard for non-point, non-human fecal contamination in shellfish-growing waters remains undefined, with regard to acute gastroenteritis.

Urban non-point run-off usually contains some sanitary waste, even in separate storm sewer systems, due to sewer malfunction.

Bacteriological water quality of rural run-off is significantly affected by type of land use - namely, presence of domestic grazing animals, and manure applications on crop land. Septic tanks are another potential source of fecal contamination.

Normally, most microbial run-off is transported over the soil surface, rather than by groundwater.

Stormwater run-off causes very sharp but transient increases in indicator bacteria levels in receiving waters.

Even shellfish from certified growing waters can transmit infectious diseases. The probability of contamination is reduced, but the possibility remains.

Enteric viruses are problematic. Those pathogenic and shellfish-transmitted are not culturable in convenient host cell systems. Norwalk-type virus has only recently been established to cause gastroenteritis and to be transmitted by shellfish. Other viruses may remain to be identified.

Therefore, bacteriological pollution due to non-point land surface run-off does represent a potential human health hazard through contamination of shellfish-growing waters. The degree of risk varies, depending on many variables.

Further studies would be required to determine whether the present 14 FC/100 ml shellfish-growing water standard could be relaxed when the pollution source was not human sewage.

Due to the numerous variables affecting quantity and quality of enteric bacteria and viruses in shellfish-growing waters, watersheds and receiving waters should be examined on an individual basis. In

addition to the usual indicators, water, sediment and shellfish samples should be assayed for specific potential pathogens (*Salmonella*, *Campylobacter*, *Yersinia*, etc.). Field surveys should be conducted following stormwater loading. Further liaison with local public health agencies could help to determine species and frequency of foodborne pathogens currently known to cause gastroenteritis.

Setting a revised standard for non-human fecal contamination will require more information on the etiology and incidence of shellfish-associated gastroenteritis.

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APPENDIX

INCIDENCE OF SHELLFISH-ASSOCIATED INFECTIOUS OUTBREAKS

INCIDENCE OF SHELLFISH-ASSOCIATED INFECTIOUS OUTBREAKS

In the United States, foodborne disease surveillance is administered at the federal level by Centres for Disease Control (CDC). Individual states also have surveillance programs. Information sharing is not ideal, judging from the meagerness of CDC's pooled data.

Annual CDC summaries of documented outbreaks give some indication of incidence and etiology of outbreaks associated with various foods, including shellfish. They emphasize, however, that there are limitations in the quantity and quality of the data. There is considerable variation in reporting to CDC between states. The responsible pathogen was not identified in over 60% of outbreaks reported to CDC in the five-year period ending 1979. The number of outbreaks reported by this surveillance system clearly represents only a small fraction of the total number that occur (CDC, 1979). However, this number has remained relatively constant over the ten-year period ending 1979.

Data for shellfish list the number of reported outbreaks (not cases) for each etiological agent. A summary is presented in Table 1. (CDC annual reports, 1975-1979).

TABLE 1.

CDC - FOODBORNE DISEASE OUTBREAKS
BY VEHICLE OF TRANSMISSION AND SPECIFIC ETIOLOGY

VEHICLE - SHELLFISH

ETIOLOGY:	19/ 75	76	77	78	79
BACTERIAL					
Vibrio cholerae	-	-	1	1	1
V. parahaemolyticus	1	-	1	2	2
Shigella	-	-	1	-	2
Staphylococcus	-	-	1	-	-
CHEMICAL					
Paralytic shellfish poisoning	-	4	-	4	-
VIRAL					
Hepatitis A	-	-	1	-	-
Hepatitis (non-B)	-	-	-	-	1
UNKNOWN	8	4	7	9	3
TOTAL:	9	8	12	16	9

DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
FOOD AND DRUG ADMINISTRATION
Shellfish Sanitation Branch

SHELLFISH BORNE DISEASE OUTBREAKS

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July 1983

This report on Shellfish Borne Disease Outbreaks is an extension and revision of work begun by others, with the last compilations being made by Mr. R. Macomber, 1953 and Dr. N. Tufts, 1970. Tables 3 and 4 by McFarren and others in 1956 on paralytic shellfish poisoning have also been updated.

The information has been gleaned from books, news accounts, National Center for Disease Control (CDC) reports, City health files, Public Health Service Regional files, case histories and old files. The data are arranged in four tables; two for Paralytic Shellfish Poisoning and two for all other diseases. The tables have been divided with breakdowns for the United States and Canada separated from the other countries of the world on which data were available.

Only cases involving clams, oysters and mussels are shown. In a few instances the data were of insufficient detail, therefore a (?) is used to denote the uncertainty or the lack of specification.

Epidemiology is the science which deals with the incidence, distribution and control of disease in a population. The evidence on hand implicates shellfish as a disease carrier if not adequately controlled. The fact that outbreaks still occur is proof that the control system has not always worked. Both incidence and distribution have been identified over the years, but the causitive agent has not been defined.

References, citing most of the outbreaks mentioned on the preceding tables have been listed alphabetically; also included are background data on the uptake of bacteria and viral contaminants by shellfish. As can be seen, no inhabited area of the world is safe from outbreaks when there are inadequate controls over the harvesting of shellfish. In the United States

and Canada, there have been over 11,500 cases of shellfish borne disease since 1900 (not including the more than 878 PSP cases). The major disease breakdowns by decades are shown in Table 5.

Vibrio parahaemolyticus causes gastroenteritis and closely resembles Salmonellosis, has been quite common in the Orient, especially Japan. In the United States, Vibrio was first "probable" associated with oysters in 1969. It is a true marine bacteria and may be expected to cause more outbreaks in the future. Vibrio cholera appeared in oysters in Florida, the first cases in nearly 90 years, occurring in 1979 and 1980.

Gastroenteritis and other associated diseases are vague and undefined terms covering a host of pathogens. Another term, Food Poisoning, accounted for hundreds of cases in the 1940's. In a few instances, specific causitive agents have been identified; i.e., Salmonella, E. coli and B. cereus. Campylobacter (Sp.), a bacteria frequently identified in livestock, was isolated from the stool specimens of some victims of the 1980 New Jersey outbreak.

Typhoid's last recorded shellfish association was an outbreak in Denmark 1972 from French oysters. This disease alone accounts for nearly one half of all shellfish borne disease outbreaks.

From 1961 through 1980, Infectious Hepatitis occurred along the Gulf and East Coast from Mississippi to Connecticut for a total of 1,394 cases. Similar to typhoid in the 50's, the number of occurrences of hepatitis have steadily decreased during the 1960's to six cases in 1969; but one of the biggest outbreaks occurred in 1973 from oysters harvested in Louisiana.

During the past seventy years, there is an apparent epidemic peak during alternate decades. The percents of each decade for the total 70-year record are shown in Table 5. Although epidemics do exhibit some cyclic rhythm, the

percentages indicated in this table could be coincidental.

Table 6 lists the medical consequences due to illnesses and intoxications associated with the consumption of shellfish.

Norwalk virus has been confirmed from oysters in Florida. The extent of this virus in the past is unknown but may account for some of the unknown etiology indicated earlier.

TABLE 1

SHELLFISH DISEASE OUTBREAKS IN

U.S. AND CANADA

Year	Etiology	Cases	Vehicle/Source	Reference
1900	Typhoid	4	Mussels, Maine	102
	Typhoid	10	Raw Soft Clams, Massachusetts	102
1902	Typhoid	80	Oysters, Clams, New Jersey	70
	Typhoid	10/52	Oysters, 52 cases not specified, Massachusetts	102
	Typhoid	25	Unknown, Maine	102
1904	Typhoid	21	Oysters, New York	102
1905	?	?	Clams, disease not specified, New Jersey	102
1908	Typhoid	5	Mussels, clams, Connecticut	102
	Typhoid	110	Mussels from Ipswich, Connecticut	102
1909-10	Typhoid	45	Clams, New Jersey	102
1911	Typhoid	1/13	Oysters, 13 cases not specified, Newburgh, New York	99
	Typhoid	17/66	Oysters, 66 cases unspecified, New York	102
1915	Typhoid	38	Oysters, New York	102
1916	Typhoid	30	Oysters, Illinois, source unknown	102
1917	Typhoid	33	Unknown, California	102
	?	?	Clams, New York	3
	Gastro-Enteritis	?	Unknown, California	102
1919	Typhoid	10	Oysters, New Jersey	102
1921	Typhoid	30	Oysters, Florida	102
	Typhoid	5	Unknown, New York	105
1922	Typhoid	6	Unknown, New York	105
1923	Typhoid	32	Oysters, Illinois, source unknown	102
	Typhoid	8	Unknown, New York	67
1924	Typhoid	1500	Oysters, 150 deaths known, W. Sayville, New York	69
	Typhoid	10	Clams, Connecticut	102

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1925	Typhoid	244	Unknown, New York	105
1926	Typhoid	8	Clams, Connecticut	102
	Typhoid	95	Clams, New Jersey	102
	Typhoid	67	Unknown, New York	102
1927	Typhoid			
	Gastro-Enteritis	--	Oysters, Virginia	102
	?	3	Oysters, poisoning, USN	102
	?	28	Mussels, poisoning, California	102
	Typhoid	50	Unknown, New York	105
1928	Typhoid	27	Unknown, New York	105
1929	Typhoid	45	Unknown, New York	105
	Typhoid	3	Clams, Connecticut	105
1930	Typhoid	26	Unknown, New York	105
	Typhoid	3	Clams, Connecticut	105
1931	Typhoid	27	Unknown, New York	105
	Typhoid	4	Clams, Connecticut	105
1932	Typhoid	14	Unknown, New York	105
	Typhoid	5	Clams, Connecticut	105
1933	Typhoid	2	Clams, Connecticut	105
	Typhoid	7	Oysters, New York	105
	Typhoid	4	Mussels, New York	105
	Typhoid	83	Clams, New York	105
1934	Gastro-Enteritis	11	Clams, New York	105
	Typhoid	3	Oysters, New York	105
	Typhoid	1	Mussels, New York	105
	Typhoid	23	Clams, New York	105
1935	Typhoid	5	Oysters, New York	105
	Typhoid	2	Mussels, New York	105
	Typhoid	52	Clams, New York	105
	Gastro-Enteritis	33	Clams, New York	105
1936	Gastro-Enteritis	1	Clams, New York	105
	Typhoid	10	Oysters, New York	105
	Typhoid	3	Mussels, New York	105
	Typhoid	23	Clams, New York	105
	?	26	Oysters, Maryland	102

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1937	Typhoid	5	Oysters, New York	105
	Typhoid	3	Mussels, New York	105
	Typhoid	29	Clams, New York	105
	Gastro-Enteritis	2	Clams, New York	105
1938	Gastro-Enteritis	1	Clams, New York	105
	Typhoid	4	Oysters, New York	105
	Typhoid	7	Mussels, New York	105
	Typhoid	27	Clams, New York	105
1939	Typhoid	12	Clams, New York	105
	Gastro-Enteritis	22	Oysters, New York	102
	Typhoid	87	Oysters from illegal harvesting, Louisiana	82
1940	Food Poisoning	30	Clams, New York	102
	Food Poisoning	15	Oysters, New York	102
	Food Poisoning	60	Clams, New York	102
	Food Poisoning	8	Clams, New York	102
	Gastro-Enteritis	20	Clams, New York	105
	Typhoid	5	Oysters, New York	105
	Typhoid	1	Mussels, New York	105
	Typhoid	24	Clams, New York	105
1941	Food Poisoning	73	Oysters, New York	102
	Typhoid	4	Oysters, Florida	102
	Typhoid	11	Oysters, Florida	102
	Typhoid	2	Oysters, North Carolina	102
	Typhoid	1	Oysters, New York	105
	Typhoid	12	Clams, New York	105
	?	300	Oysters, Maryland, outbreak New York	102
1942	Food Poisoning	38	Oysters, California	102
	Typhoid	66	Oysters, Florida	102
	Gastro-Enteritis	3	Clams, New York	105
	Typhoid	2	Oysters, New York	105
	Typhoid	8	Clams, New York	105
1943	Typhoid	5	Clams, New York	105
	Food Poisoning	16	Oysters, New York	102
	Typhoid	2	Mussels, New York	102
	Typhoid	3	Oysters, New York	105
1944	Food Poisoning	17	Oysters, New York	102
	Gastro-Enteritis	400	Clams, Massachusetts	102
	Typhoid	7	Clams, New York	102
	Typhoid	1	Mussels, New York	105
	Typhoid	2	Clams, New York	105

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1945	Typhoid	23	Clams, New York	102
	Gastro-Enteritis	2	Oysters, Massachusetts	102
	Gastro-Enteritis	?	Oysters, Massachusetts	102
	Typhoid	14	Clams, Connecticut	102
	Typhoid	8	Clams, New York	102
	Typhoid	1	Oysters, Washington	102
	Typhoid	1	Oysters, New York	105
1946	Diarrhea	300	Oysters, Texas	102
	Typhoid	3	Clams, California	102
	Typhoid	1	Clams, New York	105
1947	Food Poisoning	118	Oysters, Alabama	102
	Food Poisoning (Sal. Thompson)	100	Oysters, Florida	102
	Food Poisoning	100	Oysters, North Carolina	102
	Typhoid	5	Oysters, Florida	102
	Typhoid	3	Clams, New York	105
1948	Food Poisoning	2	Clams, Kentucky, source unknown	102
	Gastro-Enteritis	13	Clams, Washington	102
	Typhoid	1	Clams, Connecticut	102
	Typhoid	5	Clams, New York	105
	Typhoid	1	Oysters, New York	105
1949	Typhoid	1	Clams, New York	105
	Typhoid	1	Oysters, New York	105
	Gastro-Enteritis	1	Clams, New York	105
1951	?	12	Clams, source and infection unspecified	102
	Typhoid	2	Clams, New York	105
1952	Gastro-Enteritis	66	Clams, New York	102
	Typhoid	1	Clams, New Jersey	105
1953	Gastro-Enteritis	16	Oysters, California	102
1954	Food Poisoning	6	Oysters, Florida	105
	Typhoid	1	Clams, New York	105
1961	Infectious Hepatitis	84	Oysters, Mississippi, Alabama	102
	Infectious Hepatitis	459	Clams, New Jersey, New York, others	102
	Infectious Hepatitis	15	Clams, Connecticut	102
	Infectious Hepatitis	31	Oysters, Alabama	105
	Food Poisoning	>3	Mussels, location not specified	105

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1962	Food Poisoning	4	Oysters, Florida	105
	Infectious Hepatitis	3	Clams, New York	102
1964	Infectious Hepatitis	249	Clams, New Jersey, outbreak Philadelphia	14,9
	Infectious Hepatitis	123	Clams, Rhode Island, outbreak Connecticut	14,95,9
	Infectious Hepatitis	3	Oysters, North Carolina	9
	Infectious Hepatitis	43	Clams, New York	105
	Infectious Hepatitis	2	Oysters, British Columbia	105
	Infectious Hepatitis	3	Clams, New Jersey, outbreak Washington, D. C.	9
1966	Infectious Hepatitis	4	Clams, Massachusetts, Maryland, outbreak New Jersey	30
	Infectious Hepatitis	3	Clams, Massachusetts	105
	Gastro-Enteritis	5	Oysters, Illinois, source unknown	105
	Gastro-Enteritis	3	Clams, Rhode Island	105
	Gastro-Enteritis	66	Clams, Massachusetts	105
	Gastro-Enteritis	100	Clams, Unknown, outbreak Connecticut	17
	Gastro-Enteritis	33	Clams, Massachusetts, Maryland, outbreak New Jersey	30
	Infectious Hepatitis	4	Clams, Massachusetts, outbreak New Jersey	30
	Gastro-Enteritis	2	Clams, Virginia	105
1967	Infectious Hepatitis	3	Clams, oysters (harvested in Mexico)	105
	Gastro-Enteritis, Salmonella	22	Oysters, England, outbreak New York, Kingston B.W.I.	89
1968	Gastro-Enteritis	17	Clams, Connecticut	20,12,21
	Infectious Hepatitis	3	Clams, New York	97
1969	E. Coli	2	Oysters, Unknown, outbreak Washington	21
	Vibrio (Prob.)	71	Oysters/Clams, unknown, outbreak Washington	21
	Infectious Hepatitis	6	Clams, New York	21
	Bacillus cereus	4	Oysters, Unknown, outbreak Indiana	21
	Infectious Hepatitis	13	Oysters, Florida	26
1970	Staphylococcus (Unconf)	5	Clams, New York	23
	Unknown	7	Oysters, Unknown, outbreak Washington	23
	Unknown	3	Clams, Unknown, outbreak Colorado	91

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1971	Infectious Hepatitis	5	Clams, Unknown, outbreak Massachusetts	7
	Infectious Hepatitis	3	Clams, Rhode Island	33
1972	Infectious Hepatitis	1	Clams, Florida	43
	Infectious Hepatitis	1	Clams, Florida, outbreak Massachusetts	43
1973	Infectious Hepatitis	293	Oysters, Louisiana (outbreak 268-Houston, Texas; 15-Calhoun County, Georgia; and 10 in New Mexico, Oklahoma, Missouri and other Texas locations.)	87,108
	Infectious Hepatitis	1	Clams, Minneapolis, Minn.	113
1975	Unknown	50	Clams, Connecticut	132
	Unknown	2	Clams, New York City	132
1976	Unknown	36	Oysters, Hawaii	133
	Unknown	9	Oysters, Hawaii	133
	Unknown	3	Clams, New York City	133
1977	Shigella flexneri	9	Clams, Massachusetts	134
	Staff areus	5	Shellfish, Nevada	134
	Vibrio cholera	2	Shellfish, Guam	134
	Vibrio parahaemolyticus	20	Shellfish, Guam	134
	Unknown	3	Shellfish, California	134
		50	Shellfish, Connecticut	134
		23	Shellfish, Connecticut	134
		3	Shellfish, Delaware	134
		47	Clams, Rhode Island	134
		3	Shellfish, Washington	134
		5	Shellfish, Guam	134
	Infectious Hepatitis	17	Shellfish, Washington	134
1978	Unknown	2	Clams, California	135
		23	Clams, Connecticut	135
		4	Clams, Connecticut	135
		10	Clams, Connecticut	135
		6	Clams, Connecticut	135
		2	Clams, Connecticut	135
		26	Clams, New Jersey	135
		10	Shellfish, Guam	135
1979	Shigella flexneri	26	Shellfish, Arizona	136
	Shigella sonnei	11	Shellfish, California	136
	Vibrio parahaemolyticus	3	Shellfish, Guam	136
	Infectious Hepatitis	8	Shellfish, ?	136
	Infectious Hepatitis	10	Oysters, Florida (outbreak - 7 cases Mobile, Alabama, 3 in Albany, GA)	117
	Cholera	10	Oysters, Florida (Non-O-1 Vibrio cholerae)	118,120
1980	Norwalk Virus	6	Oysters, Florida	137

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1980	Gastroenteritis	46	Oysters, Florida	120
	Vibrio parahaemolyticus Vibrio cholera?	4	Oysters, Florida	120
	Cholera	1	Oysters, Florida	121,126
	Gastro. (Campylobacter?)	8	Clams, New Jersey	122
	Gastroenteritis	17	Clams, North Carolina (Outbreak - New Jersey)	123
	Cholera	3	Oysters, Florida (1 death)	124
	Gastroenteritis	40-50	Oysters, North Carolina	125
	Gastroenteritis	10	Oysters, North Carolina	125
	Gastroenteritis	6	Clams, New York (Outbreak - New Jersey)	127
	Infectious Hepatitis	1	Clams, New York (Outbreak - New Jersey)	127
1981	Gastroenteritis	210	Clams, England (Outbreak - New York)	128
	Cholera	1	Clams, Rhode Island or Massachusetts	131
1982	Gastroenteritis	443	Clams, S/F from: MA, RI, NY, NC or PEI 29 outbreaks in NY - May to Sept.	138
	Gastroenteritis	659	Clams, S/F from: NY, outbreak in NY	139
	Gastroenteritis	230	Oysters, from MA, outbreak in NY	139
	Cholera	1	Oysters, S. Carolina	140
	Gastroenteritis	472	Oysters, Outbreak in LA and TX oysters from LA	141
	Gastroenteritis	15	Oysters, Alabama, S/F from Florida	142
	Gastroenteritis	9	Oysters, FL, S/F from Florida, two outbreaks	143
1983				
4/1	Pliesmonas Shigelloides Vibrio parahaemolyticus Edwardsiella tarda	18	Oysters, Florida from Florida	144
4/5	Salmonella Pliesmonas Shigelloides	2	Oysters, Florida from Florida	144
4/5-6	Pliesmonas Shigelloides	9	Oysters, Florida from Florida	144
4/?	Gastroenteritis	3	Clams, New York from New York	145
4/15	Gastroenteritis	5	Clams, New York from EN-1	145

TABLE 1 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
4/21	Gastroenteritis	63	Clams, New York from EN	145
4/20	Gastroenteritis	7	Clams, Keron Citron, NJ from EN-3	146
4/21	Gastroenteritis	4	Clams, Lennys Clam Bar, NY from EN-1	147
4/23	Gastroenteritis	16	Clams, New York from New York/MA	147
	Gastroenteritis	2	Clams, High Seer Restaurant, NY from ?	147
	Gastroenteritis	5	Clams, New York from New York/MA	147
4/24	Gastroenteritis	24	Clams, New York from RI/NY/NC	147
4/25	Gastroenteritis	4	Clams, New York from MA/NC	147
4/27	Gastroenteritis	2	Clams, Sea Cottage Restuarant, NY from EN-3	147
4/29	Gastroenteritis	10	Clams, Holiday Inn, NJ from EN-3	146
5/?	Gastroenteritis	5	Clams, Unicorn Live Lobster Co., Hawaii from EN-1/RI	148
5/10	Gastroenteritis	135+	Clams, Farrington Manor, NJ from EN-3	146
5/13	Gastroenteritis	20	Clams, Foodtown Supermarket, NJ from EN-3	146
5/21	Gastroenteritis	4	Clams, New York from Rhode Island	147
5/28	Gastroenteritis	24	Clams, New York from New York	147
5/29-30-31	Gastroenteritis	14	Clams, New York from EN-1	147
6/4	Gastroenteritis	33	Clams, Colonial Caterers, New York New York from EN-4	149
6/16	Gastroenteritis	11	Clams, Casa Mia, New York, from New York	147
6/18	Gastroenteritis	?	Clams, Tironi's, New York, from RI/NC?	147
6/18	Gastroenteritis	36+	Clams, Brothers of Mercy, New York from EN-4	149
6/23	Gastroenteritis	15	Clams, Kloc's Grove, New York from NY	150
6/24	Gastroenteritis	400+	Clams, Homelife Ins., New Jersey from EN-2	151
6/24-25	Gastroenteritis	14(+)	Clams, Otto's Seafood/Tides, New York from EN-1/NY	152
6/25	Gastroenteritis	1,100+	Clams, Ortho Pharm., Forest Lodge, Warren Township, NJ from EN-2	153

TABLE 2

SHELLFISH DISEASE OUTBREAKS

OUTSIDE OF NORTH AMERICA

Year	Etiology	Cases	Vehicle/Source	
1815	Typhoid	6/11	Oysters, France, 11 cases unspecified	102
1883	Typhoid	2	Mussels, E. Kent, England	102
1885	Typhoid	4	Oysters, Naples, Italy	102
1893	Cholera	?	Oysters, England	102
1894	Typhoid	37	Oysters, England	102
	Typhoid	15	Oysters, England	81
	Typhoid	6	Clams or Mussels, England	81
1895	Typhoid	several	Mussels, England	81
	Typhoid	29	Oysters, England	102
	Typhoid and Gastro-Enteritis	?	Oysters, Naples, Italy floated near sewer outfall	102
	Unspecified	?	Unknown, Monte Carlo	102
	Typhoid	10	Oysters, England	102
1896	Typhoid	2	Oysters, France, 14 cases unspecified	102
	Typhoid	2	Oysters, England	102
	Gastro-Enteritis	17	Oysters, Corsica	102
	Typhoid	?	Mussels, England	102
1897	Unspecified	?	Oysters, France	102
	Typhoid	7	Oysters, Truro	102
	Typhoid	--	Mussels, England	81
1898	Typhoid	several	Clams and mussels, England	102
1899	Typhoid	58	Cockles, England	102
	Typhoid	6	Cockles, England	102
	Typhoid	23	Cockles, England	102
1900	Typhoid	37	Unknown, England	102
1902	Typhoid	11	Unspecified for 44 cases, England	102
	Typhoid	4/17	Oysters, 17 other cases unspecified, England	102
	Typhoid	10	Oysters, New Zealand	71
	Typhoid	numerous	Oysters, Turkey	92
	Typhoid	82	Unknown, England	102

TABLE 2 (Cont'd)

Year	Etiology	Cases	Vehicle/Source	Reference
1903	Typhoid	8/2	Mussels, 2 cases not specified England	102
	Typhoid	12	Cockles and oysters, England	102
	Typhoid	76	Unknown, England	102
	Typhoid	26	Unknown, England	102
1904	Typhoid	8	Unknown, England	81
	Typhoid	4/13	Unknown, England, 13 cases not specified	102
	Typhoid	2	Unknown, England	102
1905	Typhoid	143	Unknown, England	102
1906	Typhoid	3	Clams, England	102
1907	Typhoid	33	Oysters, France	102
	Typhoid	80	Unknown, England	102
1908	Typhoid	17	Unknown, England	102
1909	Poisoning	61	Mussels and unspecified, England	102
1911	Typhoid	1/14	Oysters, England, 14 cases unspecified	99
	Typhoid	17	Oysters, England	99
1922	?	?	Unspecified, France	102
1924	Typhoid	3/80	Oysters, 80 cases unspecified, England	102
	Typhoid	813	Oysters, Japan	54
1926	Typhoid	1411	Oysters, Japan	54
1953	Minamata Disease (Mercury Poisoning)	111	Oysters, Japan	60
1956	Infectious Hepatitis	691	Oysters, Sweden	
1968 1971	Infectious Hepatitis	425	Oysters, Clams, Mediterranen countries, outbreak Germany	100
1970	Vibro (Spp)	38	Oysters, Great Britain	90
1972	Infectious Hepatitis	5	Oysters, France, outbreak Denmark	41
1973	Cholera	282	Mussels, Italy	76,16,111

TABLE 2 (Cont'd)

YEAR	Etiology	Cases	Vehicle/Source	Reference
1976	Infectious Hepatitis	7	Mussels, Victoria, Australia	114
	Virus-like particles identified	797	Southampton and Chelmsford, U.K. Cockles	115
1978	Parvo virus	2000	Oysters, New South Wales, Australia	116, 119
1974	Cholera	2467	Cockles, Portugal	129
1981	Gastroenteritis	<1000	Mussels, Spain	130

TABLE 5

MAJOR DISEASE CASES

	1900 1909	1910 1919	1920 1929	1930 1939	1940 1949	1950 1959	1960 1969	1970 1979	1980 1983	Totals	%
Typhoid	310	129	2,130	471	224	4	0	-	-	3,268	29
Gastroenteritis, Food Poisoning, Diarrhea, Etc.	-	-	-	70	1,316	88	261	6	4,132	5,873	51
Infectious Hepatitis	-	-	-	-	-	-	1,051	342	1	1,394	12
Norwalk Virus	-	-	-	-	-	-	-	-	6	6	-
Vibrio	-	-	-	-	-	-	71	35	38	144	1
Unspecified	52	79	31	26	300	12	-	328	-	828	7
TOTALS	362	208	2,161	567	1,840	104	1,383	711	4,177	11,513	100%
Percent of Total	3	2	19	5	16	1	12	6	36	100%	

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