The History of Water Pollution

by

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I. History of Water Contamination

A. Early Contamination

1. Typhoid fever
2. Shigella dysenteriae (Montezuma's revenge)
3. Asiatic cholera

B. Recent Contamination

1. Traveler's diarrhea
2. Bacterial enterotoxins
3. E. coli 0157:H7 hemolytic uremic syndrome and hemorrhagic colitis
4. Salmonellosis
    a. Salmonella typhimurium
    b. Salmonella enteritidis
5. Enteric viruses
    a. Rotavirus
    140 million people infected yearly
    1 million deaths
    6% of all gastroenteritis
    20% deaths in 3rd world nations
    US spends $250,000,000 yearly and it infects 3.5 million people
    b. Polio virus
    c. Hepatitis A
    d. Sampling Water
    Collection of large volume of water 300-500 Liters Positively charged I MDS filter
    Elution of retained particulate from filter
    Concentration by centrifugation
    Cytopathological effects on tissue culture
    Limits: Not established
6. Cryptosporidium parvum
    a. Outbreaks:
    First two human cases in 1976: Three year old child had severe but self limited enterocolitis. 39 year old immunosuppresseed man (drugs) had severe diarrheae and malabsorption that quit when he went off immunosuppressive drugs.
    Rawanda in 1984 had 3% of adults and 10% children with diarrhea.
    In 1984, 4% of the preschool children in Costa Rica.
    In 1987, 17% of Haitian children.
    400,000 people in Milwaukee in 1993.
    b. Present in 65-97% of surface waters. All outbreaks of treated water met current state and federal regulations
    c. Prior to AIDS fewer than 10 cases
    d. Symptoms: Profuse water diarrhea, nausea, vomiting, abdominal cramps, and fever, Diarrhea of traverlers, daycare centers, and waterborne outbreaks

e. Life Cycle:

Infection inititiated by ingestion of sporulated oocycts that were excreted in the feces of an infected host such as cattle or humans. Sporozoites are released (excystation) and enter the brush border of the epithelial cells. The sporozoites occupy a unique intracellular location at the apex of the enterocytes within the host cell membrane but not in the cytoplasm. They undergo asexual multiplication (merogony) to produce merozoites, which can invade other cells. Some of the progeny produce sexual stages, micro and macrogamonts. Fertilization of the latter form oocysts (4 to 6 um) which contains four sporozoites. These may be thin-walled for autoinfection or thick-walled which are exceted. Prepatient period (interval between infection and oocyst shedding) ranges from 5-21 days. This may be as long as a month in normal host or longer in immunocompromised host.

f. Spread:

Feces of infected animals (especially young calves)
Childrens diapers
Sexual practices that result in exposure to feces
Untreated water

g. Diagnosis:

Stool specimen for oocysts by modified acid fast, geimsa or immunofluorescence.

h. Prevention:

Avoid contact with infected material, autoclave contaminated instruments and equipment.
Wash hands after sex, going to the bathroom and cleaning up feces from animals and humans

i. Removal:

Most commercial disinfectants are not effective.
Chlorination of drinking water not effective
5% sodium hypocholorite, 5-10% household ammonia
May be filtered with sand filter, cloth fiber filter of floculation.
(1 micrometer point of use filter)
Boiling water for 1 minute
Distillation and RO

7. <u>Giardia</u> <u>lamblia</u>

a. Outbreaks:

First recorded water-borne outbreak involved travelers to St. Petersburg, Russia. Between 1970 to 1980, 23% of the 1500 tourists to the Soviet Union became ill with giardiasis, “Leningrad’s curse”. Giardia has been isolated from wild animals such as beavers, muskrats, and water voles. This occasionally occurs in backpackers and mountain climbers (camper’s diarrhea). Twenty two percent of New Yorks scuba divers were infected (Polluted Hudson and East rivers).

b. Phases:
Cyst-acquired by ingestion from feces of infected humans or wild animals.
Trophozoites-invade the duodenum and upper jejunum and sometimes bile ducts and gallbladders. Do not invade the tissue.

c. Symptoms:
Explosive watery-diarrhea with foul smelling stools. May include nausea, upper intestinal cramping, pain, malaise, flatulence, abdominal distention, belching, anorexia, vomiting, heartburn, fever and chills.

d. Incubaction:
9 to 21 days (an Alkaline environment and rich carbohydrate diet favor multiplication.)

e. Diagnosis:
Demonstration of cysts in formed stools or Trophozoites in diarrhea stools by immunofluoresce.

f. Treatment:
Quanacrine HCL (Atabrine) or Metronidazole

8. Collection of Giardia and Cryptosporidium from Water:
Pathogenic intestinal protozoa are concentrated from a large volume of water by retention on yarn-wound filter. Retained particulates are eluted from the filter with and eluting solution and concentrated by centrifugation. Giardia cysts and cryptosporidium oocysts are seperated from other particulate matter by flotation on a Percoll-sucrose solution with specific gravity of 1.1 A monolayer of water layer/Percoll-sucrose interface is placed on membrane filter and stained with fluorescent anitbody examined with dark-field epifluoescence microscopy. Results in cysts or oocysts per 100 liters.
Limits 100/100 liters (raw water)
Limits 1/100 liters (finished water)
Monitoring began July 1997
9. Campylobacter jejuni
10. Helicobacter pylori
Etiological agent of stomach ulcers and cancer

II. Monitoring requirements

Total Coliform rule:

MPN indicators: Coliforms are lactose fermentors with gas in 24-48 hr. and gram negative bacteria
MF: 100 ml or M endo agar
January 15, 1991: Expanded monitoring and must us 100 ml.
MF or MMO-MUG: Colilert
100 ml: Total Coliforms and E.Coli (cholinesterase)
P/A
Coliert Quantitray

Only required monitoring of drinking water as of today is for coliform bacteria

Total Coliform: Enterobacter, Klebsiella andCitrobacter
Fecal Coliform: E.Coli

III. Costs
   A. We may spend as much as 3,000,000,000/year on hospital costs associated with waterborne diseases.
   B. However, less than 10% or all cases reported.
   C. 20 to 30 outbreaks yearly

IV. Why contamination with use of disinfectants
   A. Population increases
   B. Indiscriminate use of antibiotics and disinfectants
   C. Reclamation of the rain forests
   D. Susceptible hosts
   E. Mutations

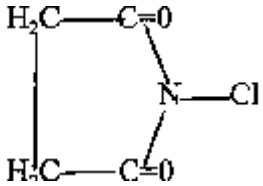
V. Recent U.S. Outbreaks (see Mar 96 AWWA journal)

Etiologic Agent	Outbreaks	Cases	States	Sources	Setting
Cryptosporidium	5	403,271	MN	Lake	Resort
			NV	Lake	Community
			WA	Well	Private
			WA	Well	Community
			WI	Lake	Community
Giardia lamblia	5	385	PA	Well	Trailer Park
			SD	Well	Subdivision
			NH	Reservoir	Community
			NH	Lake	Community
			TN	Reservoir	Correctional facility
AGI	5	495	PA	Well	Ski Resort
			SD	Well	Resort
			IN	Well	Restaurant
			ME	Well	Camp
			PA	Well	Resort
Campylobacter	3	223	MN	Well	Resort
			NY	Well	Subdivision
			MN	Well	Park
Salmonella Typhimurium	1	625	MO	Well	Community
Shigella Sonnei	1	230	NY	Well	Camp
Vibrio Cholerae	1	11	Northern Mariana Islands (Saipan)	Well	Bottled Water

VI. Disinfectants and the Killing mechanism:

A. Chlorine first used in 1835

1. Types
    a. Chlorine gas
    b. Halazone



    c. Chloramine
    d. Chlorine dioxides
2. It takes 30 minutes to kill most bacteria and it is most effective at a pH of 7.2-7.6.
3. Resistance
    a. Bacterial slime
    b. Cysts of Protozoa
4. Killing Mechanism is the oxidation of proteins (same for all halogens)

B. Bromine

C. Iodine

D. Ozone

1. Advantage: More effective than chlorine
2. Disadvantage: Nor residual antimicrobial and more prone to chance contamination. Ozone needs to be regenerated from air on site and only 10% of electricity is used to generate and therefore less cost effective than chlorine.
3. Killing mechanism is the oxidation of proteins and membrane transport.

VII.

Disinfectant by-product	Potential Health effects	Source
Bromate	Cancer	Ozonation by-product
Bromodichloromethane	Cancer, liver, kidney, and reproductive effects	Drinking water chlorination by-product
Bromoform	Cancer, nervous system, liver and kidney effects	Drinking water chlorination by-products
Chloral hydrate	Liver effects	Drinking water chlorination by-product
Dibromochloromethane	Nervous system, liver, kidney, reproductive effects	Drinking water chlorination by-product
Dichloroacetic acid	Cancer, reproductive, developmental effects	Drinking water chlorination by-product
Haloacetic acids	Cancer and other effects	Drinking water chlorination by-products
Trichloroacetic acid	Liver, kidney, spleen, developmental effects	Drinking water chlorination by-product

VII. Monitoring requiements

A. Oganic Material

B. Disinfection amount

National primary drinking water disinfectant standards

Disinfectant	MRDLG* mg/L	MRDL mg/L
Chlorine[+]	4 (as Cl_2)	4 (as Cl_2)
Chloramines[=]	4 (as Cl_2)	4 (as Cl_2)
Chlorine dioxide	0.8 (as Clo_2)	0.8 (as Clo_2)

*MRDLG-maximum residual disinfectant level goal; MRDL-maximum residual disinfectant level

[+]Measured as free chlorine

[=]Measured as total chlorine

VIII. Disinfection by-products

Contaminant	MCLG mg/L	MCL mg/L
Total Trihalomethanes (TTHMs)	N/A	0.080
Chloroform (P)	Zero	See TTHMs
Bromodichloromethane (P)	Zero	See TTHMs
Dibromochloromethane (P)	0.06	See TTHMs
Bromoform	Zero	See TTHMs
Haloacetic acids (HAA)	N/A	0.060
Dichloroacetic acid (P)	Zero	See HAA
Trichloroacetic acid(P)	0.3	See HAA
Chlorite	0.8	1.0
Bromate (P)	Zero	0.10