MAMALA BAY STUDY

RISK OF SWIMMING-ACQUIRED ILLNESSES IN MAMALA BAY

PROJECT MB-7

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1 EXECUTIVE SUMMARY

Swimming, windsurfing, canoeing and other recreational activities in polluted waters containing fecal coliform and virus levels similar to those detected on occasion in Mamala Bay have been associated with disease. Low levels, even one virus or protozoan cyst, have the potential to cause disease. Risk estimates can be made for specific microbial pathogens contaminating waterways using dose-response models. These models have been used to evaluate health risks by the Environmental Protection Agency under the Safe Drinking Water Act for the development of proper controls.

The potential of acquiring an infection from Enteroviruses, Adenoviruses, *Cryptosporidium* and *Giardia* was determined for four beaches where there were monitoring data available. Risk of acquiring a viral or protozoan infection was estimated for swimmers who were exposed for 7 days. Here it was assumed that throughout the year the average numbers of viruses or protozoa represent the best estimate of a daily concentration.

The risks for viruses range from a low of 1/1000 at Waikiki Beach to a high of 5/1000 at Queens Surf Beach. The total viral risks at the four beaches were compared to the risk of acquiring a viral infection in general by other routes (for example, contact with someone who is excreting a virus, person to-person contact). Chances of acquiring a viral infection from swimming at these beaches is equivalent or greater than the chance of acquiring an infection from all other sources during the fall and spring, but is 2 to 10 times lower in the summer when virus infections are generally greater in the population.

The risks for protozoa range from a low of 2/100,000 at Hanauma Bay to a high of 9/100,000 at Waikiki Beach. In this case, swimming is a greater risk by as much as 4.5 times compared to other means of acquiring the infection. In fact, Center for Disease Control suggest that 60% of all cases of *Giardia*sis are waterborne. It should be kept in mind that these risks represent infections and not clinical illness. However, those individuals who do become infected can serve as a source for spreading the disease to others.

Risks were examined based on exposure for 7 days estimated from the modeling efforts to determine the transport of pathogens from the outfall and canal to the beaches. In this case, risks were 1.3/100 for viruses and 3.1/10,000 for the protozoa.

Queen's Surf Beach and Ala Moana Beach and Waikiki Beach, which are influenced by the outfall and the Ala Wai Canal are more contaminated than Hanauma Bay, based on the presence of pathogens detected at the beaches. The risk of acquiring a waterborne recreational infection at these beaches range from 1/100 to 1/1,000,000. This is dependent on the time of the year, the beach which is used, the type of pathogen and the numbers of days one is exposed. Viral risks are much more significant than risks from the protozoa. It should be kept in mind that enterovirus infections have now been reported to be associated with insulin-dependent diabetes and various forms of heart disease. This could occur without the individual realizing that he or she was infected. Therefore not only are the virus risks greater but the consequences of the infection could be much more serious.

Risks could be better estimated if the specific type of virus could be identified. If epidemiological studies are attempted they should take into consideration that their sensitivity must be able to detect infections associated with swimming of less than 1/1000. In addition, the time of the year that these studies take place could be timed to the season associated with the greatest level of contamination estimated from the models.

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2 INTRODUCTION: DEFINING THE HUMAN HEALTH HAZARD ASSOCIATED WITH SWIMMING IN POLLUTED WATERS

2.1 Hazard Identification

There are three groups of microbial agents of concern which have been associated with recreational waterborne disease, bacteria, viruses and protozoa. These microorganisms produce an immense range of diseases from mild diarrhea to more serious illnesses such as cholera, hepatitis, heart disease, and diabetes. A percentage of the people affected may die. The bacteria of major importance in the United States are listed in Table 1. *Salmonella* causes typhoid fever and acute gastroenteritis (known as AGI, a diarrheal illness associated with cramping and fatigue). *Salmonella* is the number one cause of identifiable hospitalizations attributable to ingestion of contaminated food or water (Steahr and Roberts, 1993). This bacterium can also initiate an arthritides condition in 2.3% of the cases (Smith et al., 1993). *Shigella*, another bacterium, has caused 34.6 percent of all the hospitalizations (304 hospitalized cases) associated with waterborne outbreaks from 1970 to 1990 (Gerba et al., 1995). As shown in Table 1, public health officials list exposure to or ingestion of contaminated water as a significant cause of the illnesses associated with these enteric pathogens. The percentage of illness caused by exposure to contaminated water ranges from 3 percent for all *Salmonella* cases up to 75 percent for all pathogenic *E. coli* cases (Bennet et al., 1987). There are also a number of deaths reported each year as a result of these bacterial infections and the case-mortality ratio (number of deaths divided by the total number of illness or cases) ranges from 5 per 10,000 for *Yersinia* to 1 per 1000 for *Campylobacter* and *Salmonella* (Bennet et al., 1987).

Typhoid and cholera are rare in the United States (Craun, 1986;). The cholera epidemic in South America has lead to imported cases in the United States but no indigenous cases have been identified to date. Typhoid outbreaks have occurred on occasion (1 to 2 per 10 years; Craun, 1988). However, exposure to untreated wastewater has been well documented as a cause of typhoid and this was the suspected cause of an outbreak which occurred in Homestead, Florida (Pfeiffer, 1973).

The parasites of primary public health concern are the protozoa and helminths. An important characteristic of these organisms is the development of a cyst or ova stage which is excreted in the feces of infected individuals and aids in their survival in water. The pathogenic protozoa include *Giardia lamblia*, *Cryptosporidium parvum* and Entamoeba

histolytica. There has been an increase in the reported incidence of waterborne *Giardia*sis in the U.S. since 1971 (Craun, 1988).

Table 1. Waterborne Bacterial Agents of Concern

Bacteria	*Annual Reported Cases in the United States*	*Percent Waterborne*	*Case Mortality Ratio (percent)*
Campylobacter	8,400,000	15	0.1
Pathogenic *E. coli*	2,000,000	75	0.2
Salmonella non-typhi	10,000,000	3	0.1
Shigella	666,667	10	0.2
Yersinia	5,025	35	0.05

(Bennet et al., 1987)

*The percentage of all cases which have been associated with exposure to contaminated water.

**The case-mortality ratio (number of deaths divided by the total number of illnesses or cases).

Cryptosporidium was first diagnosed in humans in 1976. Since that time, it has been well recognized as a cause of diarrhea. Anywhere from 0.6 to 20 percent of the diarrheal cases will be caused by this microorganism (Fayer and Ungar, 1986). *Cryptosporidium* first emerged as a significant human pathogen in the 1980s and is now thought to be the third most common enteropathogen causing diarrheal illness. As there is no effective therapy for cryptosporidiosis, a preventative approach is required. The duration and the severity of the disease are significant, lasting as long as 7 days and causing watery diarrhea and dehydration and nausea (MacKenzie et al., 1994). In a highly publicized event, *Cryptosporidium* contaminated portions of the drinking water supply in Milwaukee, Wisconsin, causing 400,000 illnesses. This was the largest waterborne outbreak ever recorded in the United States (MacKenzie et al., 1994).

Some of the major disease-producing viruses are shown in Table 2. Over 120 possible enteric viruses may be found in untreated wastewater. These viruses have the capacity for producing diarrhea, aseptic meningitis, paralysis, conjunctivitis, myocarditis and hepatitis. The major viruses include enterovirus, rotaviruses, Norwalk viruses and

hepatitis A virus. Coxsackie B virus is also known to be associated with diabetes (Toniolo et al., 1988).

Table 2. Characteristics of Enteric Viruses in Wastewater

Virus Group	Annual Reported Cases in the United States	Case-Mortality Ratio (%)**	Diseases
Enteroviruses	6,000,000	0.001	Paralysis Aseptic meningitis Herpangina
Poliovirus	7	0.90	Paralysis
Coxsackievirus A		0.50	Aseptic meningitis
Coxsackievirus B		0.59-0.94	Paralysis fever Aseptic meningitis Pericarditis Myocarditis Congenial heart anomalies Diabetes
Echovirus			Respiratory infection Aseptic meningitis Diarrhea Pericarditis Myocarditis Fever and rash
Hepatitis	48,000	0.60	Infectious hepatitis A
Adenovirus	10,000,000	0.01	Acute conjunctivitis
Rotavirus	8,000,000	0.01	Infantile gastroenteritis
Norwalk agent (probably a calcivirus)	6,000,000	0.0001	Gastroenteritis

**The case-mortality ratio (number of deaths divided by the total number of illnesses or cases).

(Bennet et al., 1987; Melnick and Gerba, 1980; Gerba et al. 1985; Gerba and Rose, 1993)

2.2 History of Water Quality Standards

Coliform bacteria are found in the feces of mammals (including humans) and are those members of the family *Enterobacter*iaceae, belonging to the genera *Escherichia*, *Citrobacter*, *Enterobacter*, and *Klebsiella*, which can ferment lactose to carbonic acid with formation of hydrogen (H2) and carbon dioxide (CO2). Total coliforms have been traditionally distinguished from fecal coliforms by the incubation temperature of 37 degrees Celsius (°C) while fecal coliforms grow at a higher temperature of 44.5° (American Public Health Association et al., 1994; Rheinheimer, 1992). Fecal coliforms are more indicative of fecal contamination than are total coliforms.

Prior to 1968, total coliforms were generally accepted as the indicator for monitoring the water quality of recreational waters in the United States. Fecal coliforms were first proposed as a more precise indicator in 1968, and 95 percent of the States had adopted this microbial group as a bacterial indicator by 1984. Some States continue to use both total and fecal coliforms (Dufour 1984). In the presence of fecal indicator bacteria, the presence of fecal contamination can be estimated. The level and type of risk resulting from exposure, however, have been difficult to evaluate. As early as 1932, the American Public Health Association and the Joint Committee on Bathing Places considered that exposure to sewage-contaminated waterways was associated with hazards associated with eye, ear, nose, throat and skin infections. Levels between 100 and 1000 coliforms/100mL (total coliforms) were considered acceptable in terms of water quality (Joint Committee on Bathing Places, 1932). In 1953, Stevenson performed an evaluation of beaches along the Ohio River and the health effects upon persons exposed to the river water (Stevenson, 1953). He examined gastrointestinal (GI) illness (diarrhea), and eye, ear, nose and throat infections. Levels of an average total coliform count of 2,300 and 2,700 were related to an increased risk of illness for swimmers (Stevenson, 1953). Stevenson then attempted to determine the ratio of fecal to total coliforms in the river. He concluded that, of the 2,300 total coliform bacteria in a 100mL sample of river water, 400 would be fecal coliform bacteria. To ensure a margin of safety, many States, then set their water quality standards at 200 fecal coliforms per 100mL (Dufour, 1984).

While total and fecal coliform levels can be used as indicators of bacterial contamination in recreational waters, they may not be particularly useful in predicting the concentrations of protozoa or viruses. Accordingly, the indicator coliform bacteria are not always useful in predicting the safety of water, particularly as it relates to viral and protozoan enteric pathogens. The absence of total and fecal coliforms in surface water does

not suggest the complete absence of risks to humans from protozoa or viruses. This fact is particularly true in marine waters, where the coliform bacteria will die off much more quickly than the viruses (Gerba et al., 1979). The data, to date, does not demonstrate a correlation between the indicator bacteria and the enteric protozoa and viruses (Rose and Carnahan, 1992; Gerba et al. 1979). No indicators have been shown to predict the presence or more importantly the absence, of viruses or protozoa nor have they been effective at predicting the survival of enteric pathogenic microorganisms in the environment.

Microbial risks may remain significant in the absence of the indicator bacteria.

In recognizing the inherent limitations of fecal coliform as an indicator of water quality in marine waters, in States such as Florida, a recent comprehensive management plan was developed for the Biscayne Bay and recommended that 100 CFU/100mL be used as the standard for indicating chronic contamination (South Florida Water Management District, 1994). The plan additionally recommended that marine recreational areas where fecal coliform measurements chronically exceed 100 CFU/100mL be permanently posted to warn swimmers of potential health risks.

2.3 Epidemiological Studies Linking Contaminated Recreational Waters to Health Risks

There is a great deal of documentation showing that exposure to wastewater will transmit disease. There continue to be drinking water outbreaks associated with wastewater contamination (Craun, 1986). In addition, recreational waterborne outbreaks are associated with these enteric bacteria, viruses and protozoa (Herwaldt et al., 1991; Moore et al., 1993; Levine and Craun, 1990; Craun, 1986). *Shigella* and viruses, associated only with human fecal material have led to many disease outbreaks from exposure to recreational waters, with as many as 1000 people ill (Herwaldt et al., 1991; Moore et al., 1993; Levine and Craun, 1990; Craun, 1986). Fecal contamination of swimming pools has led to recreational outbreaks of both *Giardia* and *Cryptosporidium* (Morbidity & Mortality Weekly Report, 1994; Joce et al., 1991; Porter et al., 1988).

Balaragan et al., (1991) studied marine beaches in England, they reported that 24.2% of the individuals using the recreational site reported symptoms of ear infections, sore throat, sore eyes, respiratory disease and gastrointestinal illness. Risk was shown to increase with an increase in exposure from waders to swimmers to surfer to divers (going

from least risk to highest risk for the various water activities). Alexander et al. (1992) likewise found risks associated with using marine waters containing virus levels of 20 to 40 viral units per 100L, a level detected on one occasion in the Mamala Bay study. Children were found to be most at greatest risk displaying symptoms of diarrhea, vomiting and itching skin. Fewtrell et al., (1992) reported that canoeing on highly polluted waters (fecal coliforms 285 CFU/100mL and 1,984 viruses per 100L) was associated with increased risk of gastrointestinal illness even though there was no direct exposure to the water through full-body contact. This has implications for recreational boaters in the Ala Wai Canal.

Swimming, windsurfing, canoeing and other recreational activities in polluted waters containing fecal coliform and virus levels similar to those detected on occasion in Mamala Bay have been associated with disease (Corbett et al., 1993; Dadswell, 1993; DeWailly et al., 1986; Fewtrell et al., 1992; Sullivan et al., 1989; Popovich and Bondarenko, 1989).

2.4 Potential Use of Models for Predicting Health Risks

Low levels, even one virus or protozoan cyst, have the potential to cause disease (Regli et al., 1991). In addition, levels of bacteria as low as 17 colony forming units per liter (CFU/L) have been associated with water-borne outbreaks of *Salmonella* (Chalker and Blaser, 1988). Therefore, even with dilution of fecal matter in wastewater and in receiving bodies of water, the public health risks remain significant. Studies in human volunteers have demonstrated these risks and have resulted in dose-response models which can be used to evaluate low levels of risk through exposure to contaminated waters (Regli et al., 1991; Rose et al., 1991). Risk estimates can therefore be made for specific microbial pathogens contaminating waterways using dose-response models (Rose et al. 1991; Regli et al., 1991).

These models have been used to evaluate health risks by the Environmental Protection Agency under the Safe Drinking Water Act for the development of proper controls (U.S. EPA, 1989).

3 MICROBIAL DOSE-RESPONSE MODELS

The objective of microbiological dose-response models is to relate an ingested dose (d) of organisms to the probability that a particular consequence will occur. Infection has been chosen as a useful target consequence for the following reasons:

- for a number of pathogens, a substantial proportion (e.g., more than 25 %) of the individuals infected become ill (Gerba and Haas 1988; Regli et al. 1991; Rose et al. 1991; Haas et al. 1993)
- there are known methodological limitations in the recovery and enumeration of pathogens from environmental samples, that may tend to underestimate the exposure (Rose 1990; Clancy et al. 1994)
- the focus on infectivity provides a level of protection for sensitive subpopulations, for whom a greater proportion of infections may result in frank and severe illness

Several models have been developed for the response (probability of infection) of subjects exposed to ingestion of infectious microorganisms. The exponential model assumes that organisms in a dose are a random sample from a large environmental compartment, that one organism reaching a susceptible location within the host is sufficient to initiate infection, and that each organism ingested has an identical and independent probability of in vivo survival to initiate such an infection(Haas 1983). If "d" is the mean dose ingested, then the infection probability (π) is given by:

$$\pi = 1 - \exp\left(-\frac{d}{k}\right) \tag{1}$$

where k represents the reciprocal of the individual organism survival probability.

Analysis of experimental data has shown that the dose-response curves of microorganisms are often shallower than predicted by (1), indication a greater variability in response. This can be described by a probability distribution in the chance that a single organism will survive to initiate an infection. If this is given by a beta distribution, then the dose response curve can approximately be given by (Furumoto and Mickey 1967a; Furumoto and Mickey 1967b; Haas 1983):

$$\pi = 1 - \left[1 + \frac{d}{N_{50}}\left(2^{1/\alpha} - 1\right)\right]^{-\alpha} \tag{2}$$

This is termed the beta-Poisson dose response model. In (2), N_{50} is the median infectious dose, and α is a parameter indicating the shallowness of the dose-response curve. Small values of α indicate shallow dose response curves; as this approaches infinity, equation (1) is realized. Figure 1 indicates the relative comparison of the exponential dose response model with the beta-Poisson model for various values of the parameter α.

Using the method of maximum likelihood, dose-response models were fit to available experimental data on three particular organisms of concern: rotavirus, *Giardia lamblia* and *Cryptosporidium parvum*. Table 3 provides details of the data sets used to develop these models, and the references in which the analyses are described in detail.

Table 3. Summary of Experimental Data Sets and Description of Analyses for Dose-Response Parameters.

Organism	Experimental Data Sets	Results Analyzed and Reported
rotavirus	(Ward et al. 1986)	(Haas et al. 1993)
Giardia lamblia	(Rendtorff 1954; Rendtorff and C.J. 1954)	(Rose et al. 1991)
Cryptosporidium parvum	(Dupont et al. 1995)	(Haas et al. submitted)

An example of a fitted dose curve is shown in Figure 2. This curve is for the rotavirus infectivity fitted by the beta-Poisson model. Also shown are the 95 % confidence limits to the dose-response relationship.

The results of this fitting process are summarized in Table 4 for the three organisms in Table 1. These dose-response parameters were then used for the development of the risk assessment in the case at hand.

Table 4. Summary of Dose-Response Models Used in the Analysis.

Organism	Dose-Response Model	k	N_{50}	α
rotavirus	beta-Poisson	--	6.17	0.253
Giardia lamblia	exponential	50.2	--	--
Cryptosporidium parvum	exponential	238.6	--	--

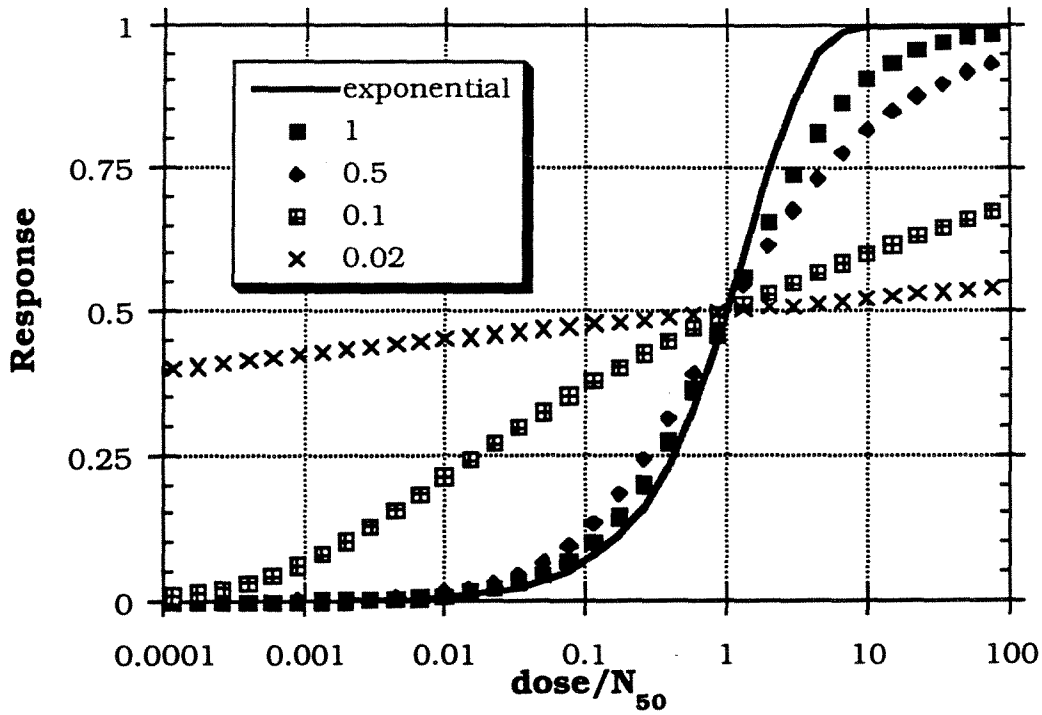


Figure 1. Comparison of Exponential Dose-Response Model with beta-Poisson Model as a function of Parameter α.

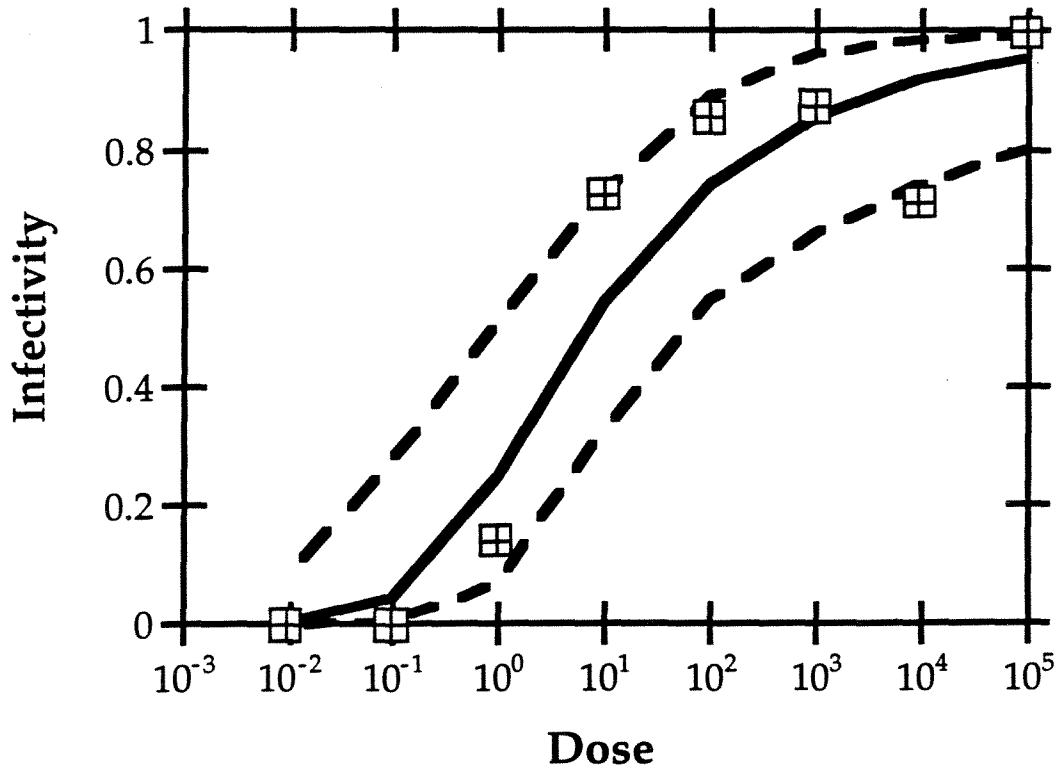


Figure 2. Beta-Poisson Dose Response Model fit to Exponential Data (symbols). Solid Line is Maximum Likelihood Estimate; Dashed Lines are 95% Confidence Limits

4 EXPOSURE CHARACTERIZATION

The exposure was characterized at the beach sites using an "MPN" equation applied to all sampling data for a single organism. The number of positive samples (P) out of the total collected (T) and the typical volume per sample (V) were used to determine an MPN average (m) by the following equation:

$$m = \frac{1}{V}\log\left(\frac{T}{T-P}\right)$$

In the case of the two potential source sites, the exposure concentration was characterized as a geometric mean. Table 5 summarizes the resultant estimates for water concentration of organisms. Figure 3 indicates the fraction of positive samples at each site.

Table 5. Summary of Pathogen Concentrations (Best Estimates) at Individual Sites.

	MPN (#/100L)					
	Sand Island Outfall	Ala Wai 1	Waikiki Bch 1	Queens Surf Bch	Ala Moana Bch	Hanauma Bay
Enterovirus	2.07	1.74	0.04	0.12	0.087	0.167
Adenovirus	12.8	2.47	0.133	0.693	0.133	0.118
Cryptosporidium	5.07	1.18	0.184	0.059	0.131	0.04
Giardia	9.11	1.48	0.184	0.126	0.183	0.04

Using the concentrations in Table 5, along with dose-response curves described above, the risk of infection for a single exposure was computed. For both enteroviruses and adenoviruses, the dose response model for rotavirus was used. A single exposure was assumed to result in the ingestion of 30 mL of water. This is used as a best estimate of risk to exposed individuals. Details of this computation are noted below.

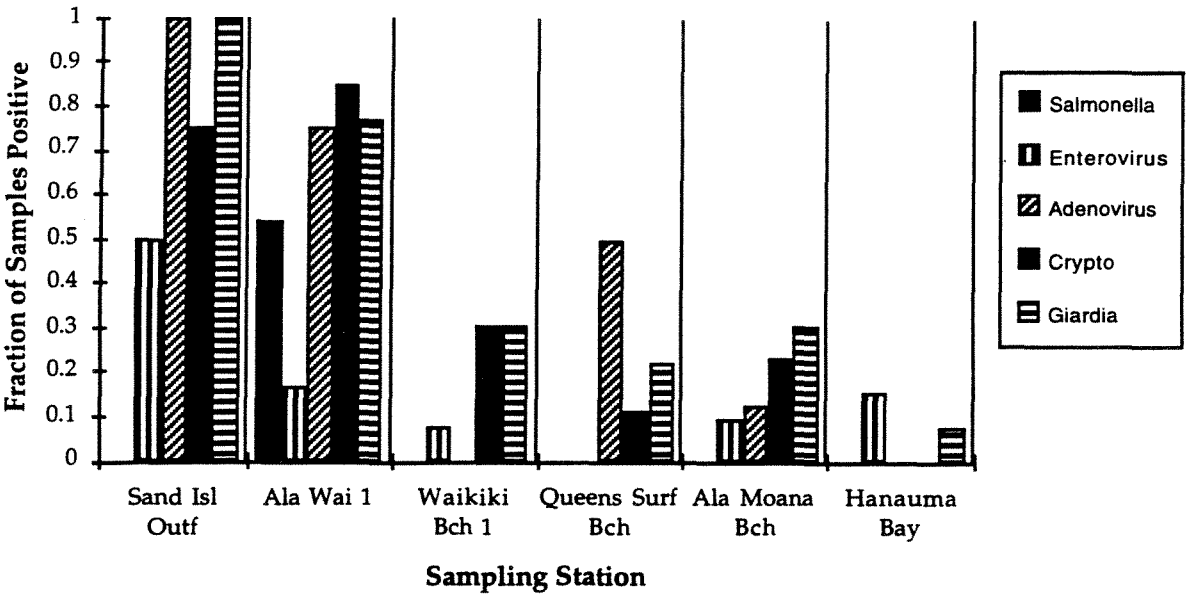


Figure 3. Fraction of Samples at each site Positive for Various Pathogens

4.1 RISK CHARACTERIZATION

Risks Based on Monitoring Data

Risks for Swimming for One Day

The potential of acquiring an infection from Enteroviruses, Adenoviruses, *Cryptosporidium* and *Giardia* was determined for four beaches where there was monitoring data available. Table 6 shows the risks for the best-estimate for a single day of exposure. Risk of an enterovirus infection was greatest when swimming at Hanauma Bay (1.4/10,000), followed by Ala Moana Beach (7.4/100,000) with a slightly lower risk at Waikiki Beach (3.4/100,000). No Enteroviruses were detected at Queens Surf Beach and thus the risk is at least less than 1/10,000 based on the limits of detection of the monitoring data. For Adenoviruses the risks were greatest at Queens Surf Beach (5.9/10,000) followed by Ala Moana Beach (1.13/10,000). The risk of acquiring a *Giardia* infection was less than acquiring a virus infection and the risks of acquiring a *Cryptosporidium* infection were approximately ten times lower. This is due to the lower infectious dose of *Cryptosporidium* as compared to the other pathogens. Total virus risks are ten-fold to 100-fold greater than the total protozoan risks depending on the beach (Table 6).

Table 6. Single Exposure Risk to Pathogens (bold face indicates <=).

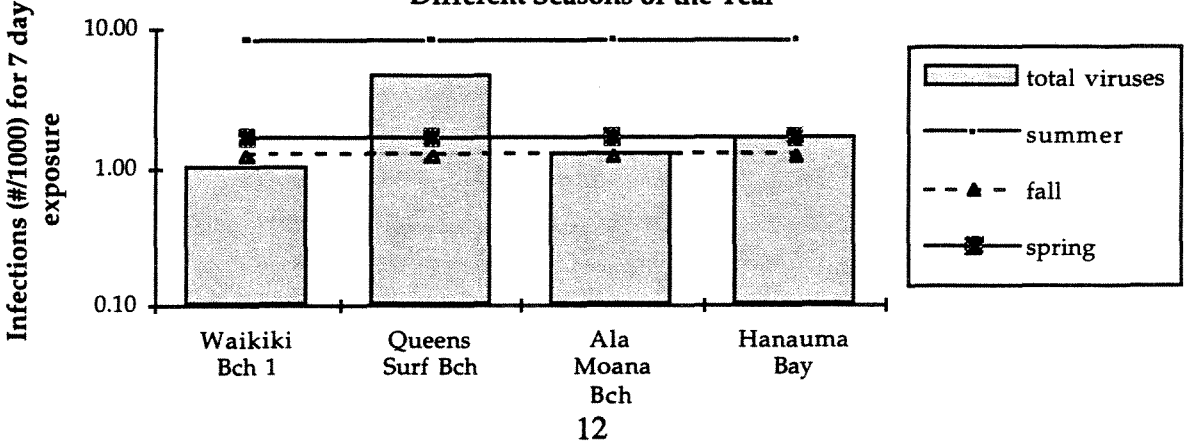
Organism	Waikiki Bch 1	Queens Surf Beach	Ala Moana Beach	Hanauma Bay
Enterovirus	3.40E-05	**1.02E-04**	7.39E-05	1.42E-04
Adenovirus	**1.13E-04**	5.88E-04	1.13E-04	**1.00E-04**
Cryptosporidium	2.31E-06	7.42E-07	1.65E-06	**5.03E-07**
Giardiaa	1.10E-05	7.53E-06	1.09E-05	2.39E-06

Risks for Swimming for Seven Days

Risk of acquiring a viral or protozoan infection were estimated for swimmers who were exposed for 7 days. Here it was assumed that throughout the year the average numbers of viruses or protozoa estimated in the Exposure section represent the best-estimate of a daily concentration.

The risks for viruses range from a low of 1/1000 at Waikiki Beach to a high of 5/1000 at Queens Surf Beach. Figure 4 shows the total viral risks at the four beaches compared to the risk of acquiring a viral infection in general in other ways (for example, contact with someone who is excreting a virus, person-to-person contact). Chances of acquiring a viral infection from swimming at these beaches is equivalent or greater than the chance of acquiring an infection from all other sources during the fall and spring. But is 2 to 10 times lower in the summer when virus infections are generally greater in the population (Payment, 1991).

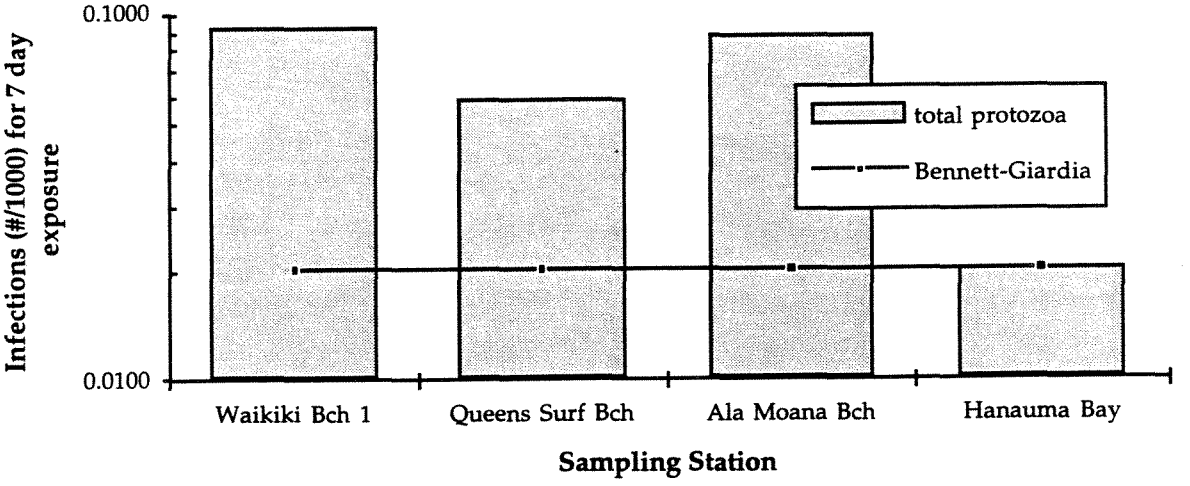
Figure 4. **Estimated Number of Viral Infections (Entero and Adeno) from Bathing at Beach Indicated vs. Background Levels Expected in the Community at Different Seasons of the Year**



The risks for protozoa range from a low of 2/100,000 at Hanauma Bay to a high of 9/100,000 at Waikiki Beach (Figure 5). In this case, swimming is a greater risk by as much as 4.5 times compared to other means of acquiring the infection. In fact, Center for Disease Control suggest that 60% of all cases of *Giardia*sis are waterborne (Bennet et al., 1987).

It should be kept in mind that these risks represent infections and not clinical illness. However, those individuals who do become infected can serve as a source for spreading the disease to others.

Figure 5. **Estimated Numbers of Protozoan Infections (Giardia and Cryptosporidium) vs. Background in the Community**



Risks Based on Concentrations Predicted from Transport Models

The following scenario was developed to assess recreational waterborne disease risks. Two groups of 100 people each visit Honolulu for one week. One group swims every day at Waikiki Beach while the other 100 do not go near the water. The potential risks of a virus and *Giardia* infection and subsequent illness were examined.

The group of tourists are visiting between May 18 through the 24th. These dates were used as this represents a time when the modeling predicts larger concentrations of contaminants at the beaches over the seven days.

Table 7. Concentrations of Viruses and *Giardia* at the Waikiki Beach Based on Transport Modeling Data

Date	Virus levels/L	*Giardia* levels/L
May 18	0.0288	0.02
May 19	0.1908	0.133
May 20	0.4064	0.283
May 21	0.332	0.231
May 22	0.139	0.097
May 23	0.236	0.164
May 24	0.188	0.131
Average for 7 days	0.2183	0.151

Exposure Issues and Risk Estimates for Swimmers

1. Exposures were set at 30mL for the swimmers at average concentrations for 7 days.

2. Assumptions used: All cysts detected were viable, and the Rotavirus model reflects risks from a variety of enteric viruses.

The models predict the probability of infection, however it will be assumed that 50% of those infected will become ill this is based on clinical data (Gerba and Rose, 1993 and Rose et al., 1991).

Table 8. Estimates of Risk Associated with Recreational Exposure at Waikiki Beach

Model	Average Exposure 30mL	Daily Risk	7-day risk	Illness Risk	No. ill per 100 people
Giardia $P=1-\exp(-0.0198 \times N)$	0.004538	9×10^{-5}	6.3×10^{-4}	3.1×10^{-4}	0.031
Rotavirus $P=1-(1+N/0.42)^{-0.24}$	0.006549	3.7×10^{-3}	2.6×10^{-2}	1.3×10^{-2}	1.3

The risk estimates shown in Table 8 suggest that at certain times of the year , viral risks could be as high as 1.3 /100 based on 7 days of exposure to levels predicted by the transport models. *Giardia* risks are much lower at 3.1/10,000.

Risks for the Non-swimmers

Enteric infections can be acquired from a variety of exposure routes including contaminated food and surfaces. It is unlikely that tourists will be exposed to day care centers while on vacation, but this is certainly another route of exposure for those individuals with young children.

According to Payment (1991), total virus enteric infections based on excretion of the virus in stool and not on illness ranges from a monthly rate of 7.5 per 1000 individuals in the spring (February through June), 36.1 per 1000 in the summer (July through August) and 5.2 per 1000 in the Fall (September through June). Seasonal distribution for *Giardia*sis infections has not been readily documented but the Center for Disease Control and Prevention estimate that the annual disease rate is 5 per 10,000 individuals (Bennett et al., 1987). These figures can be used to examine the risks to the non-swimming population of 100 visiting Hawaii from all other sources.

Table 9. Risks of Virus Infections in the General Population

Virus Infection Monthly Risk February-June	7 day Risk of Virus Infection	Infections per 100 people	Illnesses per 100 people
7.5 per 1000	1.7 per 1000	0.17	0.085

During the summer months when enteric virus infections are known to peak the 7 day risk would be 0.85 infections and 0.425 illnesses per 100 people. Compared to the risk from swimming in July if one assumes that the swimmer is exposed to only one day of contamination based on the monitoring data (0.2 viruses/ L, July 11, Waikiki Beach) the risk is 0.34 infections and 0.17 illnesses per 100 people. No illnesses would be expected in either group.

The comparison of risks for swimmers shows a risk which is 15 times greater than that for non swimmers for viruses and 6200 times greater for *Giardia* during certain parts of the year when modeling predictions suggest a greater level of contamination at the beaches. The greater level of infectivity of the viruses gives these microorganisms a higher

risk compared to the protozoa. One can predict that out of every 100 people, 1 person may become ill with a virus through recreational exposure during spring months.

Table 10. Risks of *Giardia* Infections in the General Population

Giardia Infection Annual Risk	7 day Risk of *Giardia* Infection	Infections per 100 people	Illnesses per 100 people
5 per 10,000	1 per 100,000	0.00001	0.000005

5 CONCLUSIONS

Based on the presence of pathogens detected at the beaches (see Figure 3, section on Exposure) it can be seen that Queen's Surf Beach and Ala Moana Beach and Waikiki Beach are more contaminated than the Hanauma Bay. The Waikiki Beach is east of the Ala Wai Canal and the Ala Moana Beach is west of the canal. According to the modeling data based on enterococci the Ala Moana Beach is most significantly impacted by the canal followed by Waikiki Beach and finally the Queens Surf Beach which is further east of Waikiki. The outfall most significantly impacts Waikiki Beach followed by Queens Surf Beach followed by Ala Moana. The monitoring data support this and thus the riskiest beaches can be identified.

Hanauma Bay was considered a beach that neither the canal nor the outfall would significantly impact. No Adenoviruses, *Cryptosporidium* were detected. One sample was positive for *Giardia*, however two samples were positive for Enteroviruses. This beach was very well protected with less circulation. One may hypothesize that Hanauma Bay may be frequented by more families with young children as it is more protected water and is calmer. Thus the Enteroviruses could be associated with young children using the water. These data point out that on occasion viruses can be detected in recreational sites with swimmers as the major source of the contamination.

The risk of acquiring a waterborne recreational infection range from 1/100 to 1/1,000,000. This is dependent on the time of the year, the beach which is used, the type of pathogen and the numbers of days one is exposed. Viral risks are much more significant than risks from the protozoa. It should be kept in mind that enterovirus infections have now been reported to be associated with insulin dependent diabetes and various forms of heart disease (Bowles et al., 1986; Toniolo et al., 1988). This could occur without the individual realizing that he or she was infected. Therefore not only are the virus risks greater but the consequences of the infection could be much more serious.

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