TABLE 6. SUMMARY OF THE MEAN ENTEROCOCCUS DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIPS OBTAINED FROM CLUSTERED TRIALS FOR ALL THE U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7a, TABLE 10)

Study	Beach	Year	Enterococcus Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock[5]	1973[8]	21.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		91.2	474	167	72	24	48*	46.4	18.0	28.4
		1974	3.6	1391	711	27	23	4	7.6	4.2	3.4
			7.0	951	1009	38	34	4	10.5	6.9	3.6
			13.5	625	419	42	17	25*	16.0	2.4	13.6
			31.5	831	440	43	23	20	18.1	—	18.1*
		1975	5.7	2232	935	63	55	8	18.8	19.3	-0.5
			20.3	1896	678	59	37	22*	14.8	7.4	7.4
			154	579	191	60	31	29	34.5	—	34.5*
Lake Pont.[3]	Levee	1977	44	874	451	86	51	35*	32.0	11.1	20.9*
			224	720	456	108	50	58**	31.9	8.8	23.1*
			495	895	464	108	54	54**	35.8	8.6	27.2**
	Levee	1978	11.1	1230	415	75	34	41**	36.6	14.5	22.1*
	Font.[7]		14.4	248	303	81	63	18	44.3	23.1	21.2
	Levee		142	801	322	112	50	62**	42.4	15.5	26.9*
Boston H.[4]	Revere	1978	4.3	697	529	83	66	17	23	11	12
	Nahant		7.3	1130	1099	71	67	4	33	28	5
	Revere		12.0	222	376	108	74	34*	41	13	28*

[1] Difference (swimmer rate minus nonswimmer rate).
[2] New York City, NY.
[3] Lake Pontchartrain, LA.
[4] Boston Harbor.
[5] Rockaways.
[6] Coney Island.
[7] Fontainebleau.
[8] Study population too small to cluster trials by similar indicator densities.

*$p<0.05$; **$p<0.01$

TABLE 7. SUMMARY OF THE MEAN ENTEROCOCCUS DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIPS OBTAINED FROM TRIALS GROUPED BY BEACH AND YEAR FOR ALL U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7b).

Study	Beach	Year	Enterococcus Density per 100 ml	N		Symptom Rates in Cases Per 1000 Study					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock[5]	1973	21.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		91.2	474	167	72	24	48*	46.4	18.0	28.4
	Rock.	1974	3.5	2767	2156	39	35	4	12.0	12.0	0.0
	C. Is.		16.4	1961	1185	42	26	16*	16.0	9.3	6.7
	C. Is.(1)	1975	17.9	1534	590	70	54	16	21.2	12.6	8.6
	(2)		27.7	1744	623	57	42	15	21.8	22.5	−0.7
	(3)		6.7	1131	475	50	44	6	13.7	8,5	**5.2**
	(4)		14.2	298	96	60	31	29	23.5	10.4	13.1
Lake Pont.[3]	Levee[8]	1977	44	874	451	86	51	35*	32.0	11.1	20.9*
			224	720	456	108	50	58**	31.9	8.8	23.1*
			495	895	464	108	54	54**	35.8	8.6	27.2**
	Levee[8]	1978	11.1	1230	415	75	34	41**	36.6	14.5	22.1*
			142	801	322	112	50	62**	42.4	15.5	26.9*
	Font.[7]		14.4	248	303	81	63	18	44.3	23.1	**21.2**
Boston H.[4]	Revere	1978	6.3	919	905	89	70	19	27.0	12.0	15.0
	Nahant		7.3	1130	1099	70	64	6	33.0	28.0	5.0

[1-7] See Table 6 for abbreviations.

[8] Data from Levee Beach were only clustered by trials with similar indicator densities for reasons explained in text.

*$p<0.05$; **$p<0.01$

TABLE 8. SUMMARY OF THE MEAN *E. coli* DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIP OBTAINED FROM CLUSTERED TRIALS FOR ALL THE U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS FIGURE 7c)

Study	Beach	Year	*E. coli* Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock.[5]	1973[8]	24.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		174.0	474	167	72	24	48*	46.4	18.0	28.4
		1974	**2.2**	2514	1641	25	34	−9	8.0	3.7	4.3
			13.3	1304	1045	38	29	9	14.1	5.7	8.4*
			30.5	600	425	65	33	32*	23.3	2.4	20.9
		1975	46.8	1945	1099	55	51	**4**	13.4	17.8	−4.4
			142	775	194	76	41	35	24.5	10.3	14.2
			278	1049	330	55	24	31*	21.0	3.0	18.0*
			514	937	271	68	55	13	24.5	7.4	17.1
Lake Pont.[3]	Levee	1977	44	372	222	132	45	87**	32.3	9.0	23.3
			161	910	306	120	65	**55****	52.7	22.8	29.9*
			497	574	307	85	45	40*	**32.8**	13.0	19.8
			3091	419	204	88	83	5	31.0	4.9	26.1
	Font.[7]	1978	9.0	248	303	81	63	18	44.3	23.1	**21.2**
	Levee		32.6	1123	382	78	44	**34***	38.3	20.9	17.4
			93.7	918	355	103	36	67**	39.2	8.5	30.7
Boston H.[4]		1978	5.5	541	874	72	63	9	39	29	10
			7.0	477	410	86	68	18	23	**10**	13
			17.5	589	225	70	67	3	27	27	0
			29.5	442	495	93	71	22	32	14	**18**

[1-8] See Table 6 for abbreviations.

*$p<0.05$; **$p<0.01$

TABLE 9. SUMMARY OF THE MEAN *E. coli* DENSITY—GASTROINTESTINAL SYMPTOM RATE RELATIONSHIP OBTAINED FROM TRIALS GROUPED BY BEACH AND YEAR FOR ALL U.S. STUDIES (INPUTS TO THE REGRESSION ANALYSIS, FIGURE 7d)

Study	Beach	Year	*E. coli* Density per 100 ml	N		Symptom Rates in Cases Per 1000					
						Total Gastrointestinal			Highly Credible GI		
				Swim	Nonswim	Swim	Nonswim	▲[1]	Swim	Nonswim	▲
NYC[2]	Rock.[5]	1973	24.8	484	197	81	46	35	30.4	15.2	15.2
	C. Is.[6]		174.0	474	167	72	24	48*	46.4	18.0	28.4
	Rock.	1974	2.4	2767	2156	39	35	4	12.0	12.0	0
	C. Is.		15.3	1961	1185	42	26	16*	16.0	9.3	6.7
	C. Is.(1)	1975	52.4	1534	590	70	54	16	21.2	12.6	8.6
	(2)		98.6	1744	623	57	42	15	21.8	22.5	−0.7
	(3)		61.3	1131	475	50	44	6	13.7	8.5	**5.2**
	(4)		157	298	96	60	31	29	23.5	10.4	13.1
Lake Pont.[3]	Levee[8]	1977	44	372	222	132	45	87**	32.3	9.0	23.3
			161	910	306	120	65	**55****	52.7	22.8	**29.9***
			497	574	307	85	45	40*	32.9	13.0	**19.8**
			3091	419	204	88	83	5	31.0	4.9	**26.1**
	Font.[7]	1978	9.0	248	303	81	63	18	44.3	23.1	**21.2**
	Levee[8]		32.6	1123	382	78	44	**34***	38.3	20.9	17.4
			93.7	918	355	103	36	67**	39.2	8.5	**30.7***
Boston H.[4]	Revere	1978	18.0	919	905	89	70	19	27.0	12.0	15.0
	Nahant		11.5	1130	1099	70	64	6	33.0	28.0	5.0

[1-8] See Table 7 for abbreviations and notations.

*$p<0.05$; **$p<0.01$

a mean indicator density limit to be used as a guideline. This requires the regression of X on Y. These lines along with their confidence limits, correlation coefficients and formulae are given in Figure 9. The 95 percent confidence limits for the mean enterococcus densities predicted for the observed swimming-associated rates are given in Table A52. The author favors the use of the criteria for HCGI symptoms because of the greater credibility of its data base and because it is more conducive to economic analysis. The 95 percent confidence limits for the regression lines as shown (Figure 9) are rather broad although the slopes are significantly different from zero. This was not unexpected since the relationships obtained are generalizations which may be altered by any of a number of temporal and spacial factors relative to the indicator, the pathogen, the relationship of the pollution sources to the bathing beach, the levels of the specific illnesses in the overall population, and the immune status of the swimmers. These will be discussed in the next two sections

Examination of the illness-indicator relationships by location and by year at a given location could provide some insight as to possible spatial and temporal effects. The latter was not attempted because of the small number of points available for analysis by year. The regression lines for the New York City study were compared to those obtained from

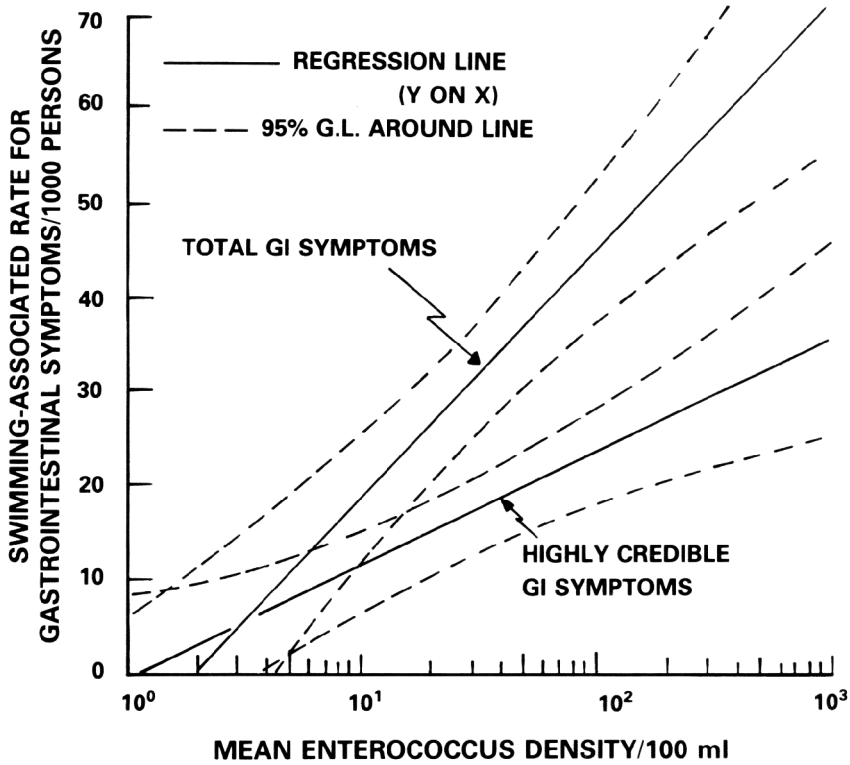

Figure 8. Regression lines for swimming-associated GI symptom rates (Y) against the mean enterococcus densities in water (X). Lines drawn from averages of slopes and intercepts from Figures 7a and 7b. Confidence limits are those for the regression lines shown in Figure 7a. Representation predicts the illness rates from the indicator densities and presents the 95% confidence limits of the former.

TABLE 10. REGRESSION FORMULAE AND CORRELATION COEFFICIENTS FOR SWIMMING-ASSOCIATED GI SYMPTOMS AGAINST ENTEROCOCCUS DENSITIES AND AGAINST *E. coli* DENSITIES IN THE BATHING WATERS (ALL U.S. STUDIES)

Indicator	Analysis by	N	Gastrointestinal Symptoms				HCGI Symptoms[1]			
			Slope	Intercept	r	p	Slope	Intercept	r	p
Enterococcus	Trials	18	24.19	−5.09	.82	<.001	12.17	0.20	.75	<.001
	Summers	16	27.37	−9.52	.86	<.001	11.53	−1.36	.72	<.005
Average			25.78	−7.31			11.85	−0.58		
E. coli	Trials	20	7.37	15.73	.25		6.30	5.88	.54	
	Summers	17	6.63	17.72	.20		7.30	2.79	.52	

[1] Highly credible gastrointestinal symptoms.

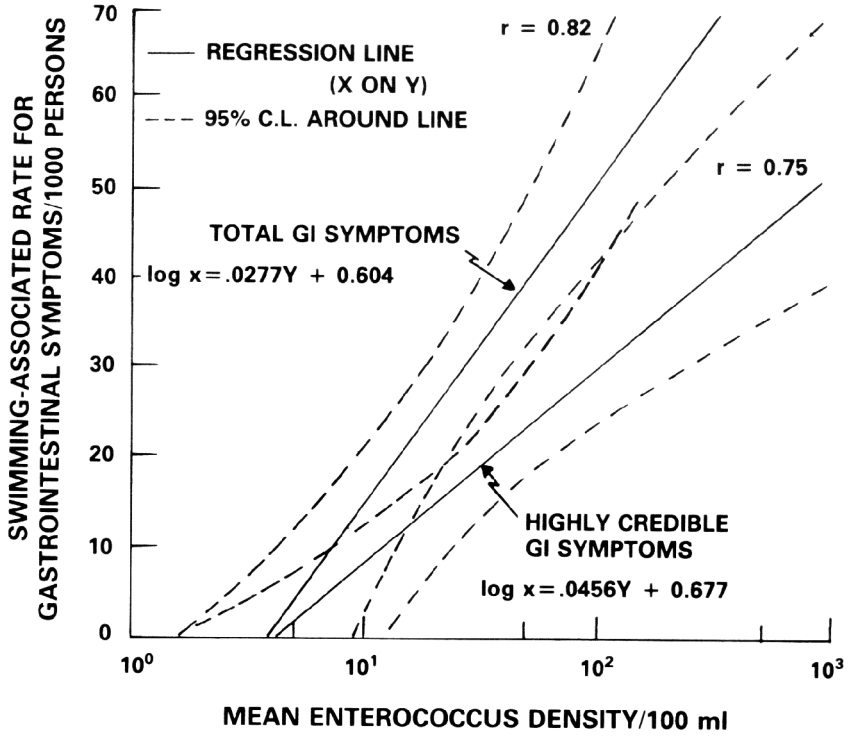

Figure 9. Health effects criteria for marine recreational waters developed by the USEPA epidemiological-microbiological program. Criteria are X on Y regression lines of the mean enterococcus density in the water against the swimming-associated rate of gastrointestinal symptoms. Lines drawn in the same manner as those shown in Figure 8. The 95% confidence limits around the lines are those for data given in Table 6.

the combination of the Lake Pontchartrain and the Boston Harbor studies; however, even for the trials clustered by similar indicator densities (Table 6), each line was defined by only nine points. Significant differences were obtained between the lines for highly credible but not total GI symptoms. The lines for total GI symptoms were not significantly different; however, those for HCGI symptoms were, although the two lines stay virtually within the 95 percent confidence limits of the total data. This provides some basis for the generalization obtained from the single regression line. This generalization may not be totally accurate in all situations. Thus, in the present case, the sources of pollution to the beaches in the Lake Pontchartrain and Boston Harbor studies were ill-defined and, presumably, more distant. This and the effect of the immune status of the swimming population could explain the significant differences between the regression lines for highly credible but not total GI symptoms. In any event, these results. emphasize the conclusion that guidelines derived from these criteria cannot be used without judgment; rather, they must be used in concert with good public health practice (e.g., taking into consideration changes in the incidence of enteric disease in the discharging population), an environmental (sanitary) survey, and judgment with regard to their limitations in time and space. In fact, the correlations obtained are remarkably

good when the sources of temporal and geographic variability are considered, and this has some interesting implications concerning the agent(s) and host population, i.e., ubiquity, infectivity, survival, immunity, etc.

THE ETIOLOGIC AGENT(S)

When the study design for the EPA program was being developed in 1969-1970, it was thought that swimming in sewage-polluted waters would constitute a relatively minor route of transmission for GI illness and that relatively high levels of pollution (as indexed by microbial indicator densities) would be required before GI illness could be detected. These assumptions were made on the basis of existing notions and available information (8,24). Both these assumptions were incorrect. If the nonswimming rates for GI symptomatology can be considered as those for the population at large, then swimming in sewage-polluted waters constitutes a significant route of transmission for the illnesses obtained, at least for individuals of "swimming age." This can be seen from the tabular (Table II) and graphic (Figure 10) representations of the ratios of the rates for

TABLE 11. RATIO OF SWIMMER TO NONSWIMMER GASTROINTESTINAL SYMPTOM RATES BY ENTEROCOCCUS DENSITY[1]

Enterococcus	Swim/Nonswim GI Symptom Rate	
Density/100 ml	Total Gastrointestinal	Highly Credible GI
3.6	1.17	1.81
4.3	1.26	2.09
5.7	1.15	0.97
7.0	1.12	1.52
7.3	1.06	1.18
11.1	2.21	2.52
12.0	1.46	3.15
13.5	2.47	6.67[2]
14.4	1.29	1.92
20.3	1.59	2.00
21.8	1.76	2.00
31.5	1.87	I[3]
44.0	1.69	2.89
91.2	3.00	2.58
142.0	2.24	2.74
154.0	1.94	I[3]
224.0	2.16	3.63
495.0	2.00	4.13

[1] Data taken from Table 6.
[2] Due to unusually low nonswimmer rate.
[3] Indeterminate because of no cases among nonswimmers.

swimmers divided by those for nonswimmers against the enterococcus densities for the clustered trials. In fact, at enterococcus densities of 70 and 10/100 ml, respectively, the rates for total and HCGI symptoms among swimmers were twice those for nonswimmers, and they are projected to be equal (a ratio of "I") at an enterococcus density of about 1/100 ml. This suggests that the etiologic agent(s) for the observed GI symptomatology is present in sewage in large numbers, that it is highly infective and/or that it survives sewage treatment, disinfection and/or transport better than the indicator.

One of the desired outputs from the program was an answer to the question: Does the swimming-associated illness rate increase with the levels of these specific illnesses in the population at large? This relationship was not observed for the types of illnesses obtained in this study (Table 12), probably because of the high level of immunity to the agent in the population.

Initially, it was thought that the Egyptian data could be used in the derivation of the final criteria. By the end of the first year of the Egyptian study, it was obvious that the data from the Alexandria residents could not be so used, and by the end of the third year, it was concluded that this was also true of data from the Cairo visitors. The regression lines for the rates of swimming-associated vomiting and diarrhea from these two groups along with those for GI and HCGI symptoms from the United States studies against the corresponding mean enterococcus densities are presented in Figure 11. It can be seen that, in the United States studies, gastrointestinal illness rates comparable to those obtained in the Egyptian study were associated with bathing in waters with much lower enterococcus densities. Part of the dissimilarity is probably due to differences in the nature (raw vs. treated) and proximity of the pollution sources in the United States and Egyptian studies. However, disparities in the immune state of the populations to the etiologic agent(s) probably accounts for most of the differences in the indicator-illness relationships obtained.

The importance of immunity in the epidemiology of the swimming-associated gastroenteritis is also supported by the age distribution of the attack rates. In most of the studies, children (<10 years of age) were found to have the highest symptom rates.

The following characteristics of the swimming-associated illness were obtained in or can be inferred from the findings of the EPA program: (i) The illness is a relatively benign gastroenteritis with a short incubation period (Figure 12), acute onset, short

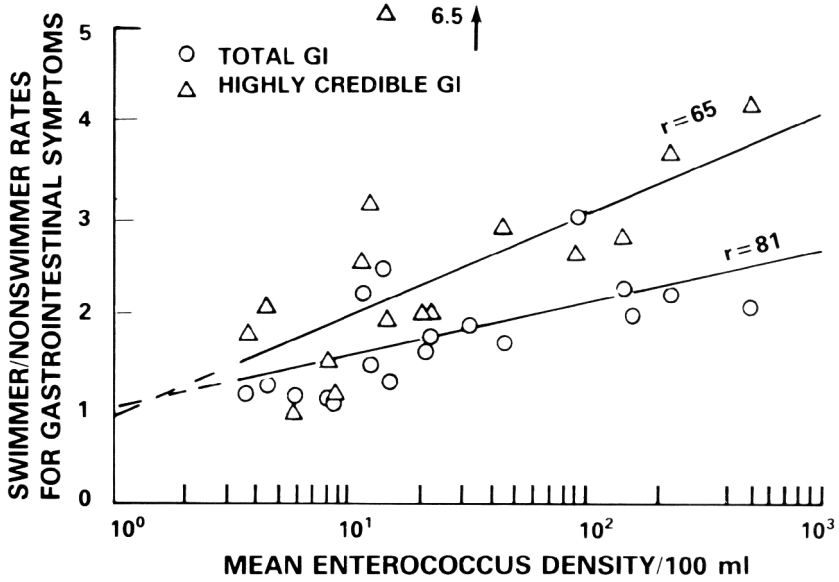

Figure 10. Ratios of swimmer to nonswimmer rates of gastrointestinal symptoms against the mean enterococcus density in the water. Data from Table 6. One value not used in the calculations.

TABLE 12. RELATIONSHIP OF SWIMMING-ASSOCIATED (▲) TO BACKGROUND (NONSWIM) RATES FOR GASTROINTESTINAL SYMPTOMS

Enterococcus Density[1]	Rates Per 1000 Persons			
	Total GI		Highly Credible GI	
	Nonswim	▲	Nonswim	▲
3.6-7.0	23	4	4.2	3.4
	34	4	6.9	3.6
	55	8	19.3	−0.5
	66	17	23.0	11.0
	67	4	28.0	5.0
11.1-21.8	17	25	2.4	13.6
	34	41	13.0	28.0
	37	22	14.5	22.1
	46	35	14.8	7.4
	63	18	15.2	15.2
	74	34	23.1	22.1
91.5-154	24	48	15.5	26.9
	31	29	18.0	28.0
	50	62	—[2]	34.5

[1] Values ordered according to the nonswimming rate within a density cluster. Only clusters of 3 or more reasonably close values used.
[2] Nonswimming rate "0."

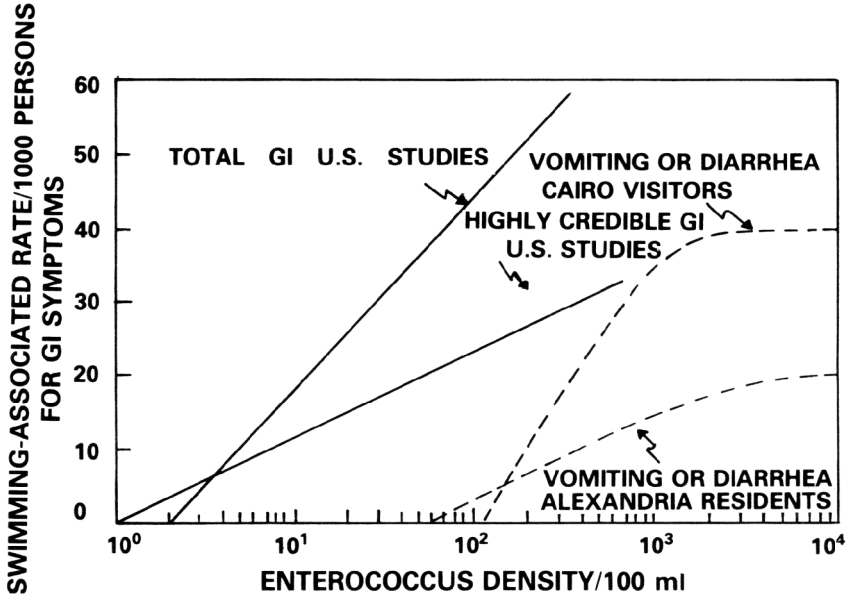

Figure 11. Comparison of the illness-indicator relationship obtained from the U.S. studies with those for the Cairo visitors and Alexandria residents in the Egyptian studies. Those for the U.S. populations taken from Figure 7 and those for the Egyptian study from Figure 4.

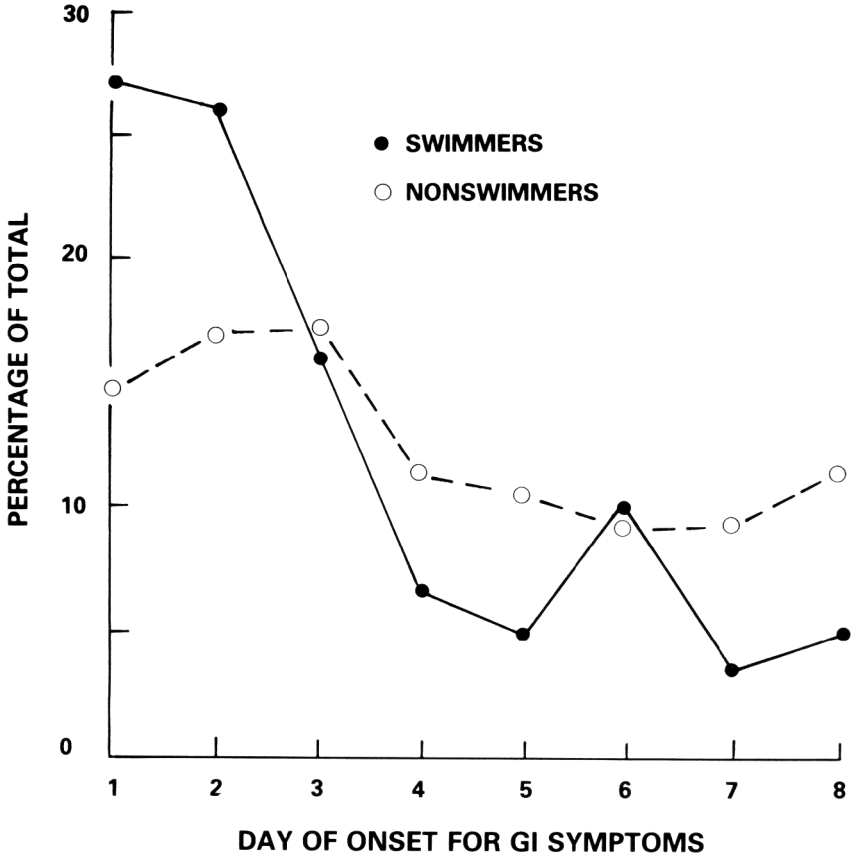

Figure 12. Day of onset of GI symptoms as obtained from the 1975 New York City trials.

duration (Table 13) and rare, if any, sequelae. (ii) It is widely distributed; most individuals are immune, and, in general, children have the highest attack rates. (iii) The etiologic agent is highly infectious, is present in sewage in large numbers, and/or survives sewage treatment disinfection and transport in the marine environment somewhat better than the indicators. These considerations suggest the human rotavirus and/or the parvo-like viruses as. the etiologic agents.

There are at least three explanations for the observations that individuals who swim during several days in a given week (from the Egyptian study) or for prolonged periods during a given day (from the New York City study) have low GI symptom rates. The obvious one is that these are "healthier" individuals. The second assumes that the extent of swimming is correlated with age, that is, individuals who swim regularly and extensively are more experienced and ingest less of the bathing water. However, it is commonly assumed that children are in the water the longest and also ingest the most water. The third explanation requires that the illnesses involved have short (< 3 days) incubation periods and that there may be a good immunity to the etiological agents The rationale for the Egyptian observations is that the susceptible individuals become ill within a day or so of the time they start swimming.

TABLE 13. DURATION OF GASTROINTESTINAL SYMPTOMATOLOGY: NEW YORK CITY, 1975 TRIALS

Symptom	Duration of Symptoms in Days for Swimmers		Nonswimmers	
	Number Reporting	Average Duration	Number Reporting	Average Duration
Total				
Vomiting	30	2.8	10	2.6
Diarrhea	73	2.6	26	2.7
Stomachache	101	2.7	36	2.4
Nausea	64	2.7	18	2.8
Disabling				
Vomiting	17	3.7	5	2.6
Diarrhea	22	3.0	11	3.2
Stomachache	36	3.5	12	3.0
Nausea	24	2.6	8	3.2

SECTION 7
LIMITATIONS IN THE USE OF THE RECOMMENDED CRITERIA

The criteria presented in this report (the enterococcus density in the bathing water against the swimming-associated rates for total and HCGI symptoms) are generalizations which have been found to apply in a number of situations. Nevertheless, a number of considerations, including the limitations in the indicator concept itself, impact on the use of the criteria as well as the guidelines and standards derived therefrom. More important, these considerations require that the findings from monitoring programs be interpreted in the light of good public health and environmental practice. They have been described elsewhere (49, 66) and several of the more important ones will be considered herein.

SMALL POINT SOURCES

The rationale for the use of guidelines and standards based on fecal indicator densities for indexing the health hazards in sewage polluted waters is that, under average conditions of illness in the discharging population, there is a reasonably constant indicator to pathogen ratio in the sewage and its receiving waters. Thereby, an acceptable probability of illness caused by the pathogen can be extrapolated to a given indicator density, which is then recommended as a guideline and promulgated as a standard. Such relationships appear to hold for waters receiving the discharges from relatively large municipal sewage treatment facilities. However, as the number of individuals who contribute to the source of the fecal wastes becomes smaller and smaller, the indicator-pathogen ratio will vary more and more from the average upon which the guideline or standard is based. In the extreme case where the fecal wastes of a single ill individual or carrier are discharged into the water, the number of pathogens may equal or exceed the number of indicator microorganisms. Routine examination of such waters for fecal indicators would be of no value. Furthermore, the routine examination for the pathogens would not be especially useful since the release of enteric pathogens will be sporadic. The solution is administrative action prohibiting such discharges into recreational waters.

ILLNESS RATES IN THE DISCHARGING POPULATION

Most epidemiologists and health officers recognize that, under epidemic conditions, the actual indicator-pathogen ratio may change sufficiently from that upon which a guideline was based so that the acceptable risk of illness will be exceeded unless the guideline is temporarily made more restrictive. The recent swimming-associated outbreak of shigellosis on the Mississippi River below Dubuque, Iowa (15) appears to represent an instance where, although the 200/100 ml fecal coliform guideline was probably exceeded, the outbreak did not occur until there was a large enough number of ill individuals and carriers in the discharging population. Conversely, if there is a significant and consistent decrease in the illness rate in the discharging population over a prolonged period of time, the rate for that specific illness associated with an existing indicator guideline or standard may be considerably less than predicted. The absence of recreational water-associated salmonellosis probably represents a case in point.

FECAL INDICATORS VERSUS PATHOGENS

The use of fecal indicators such as coliforms or portions of the coliform population, fecal streptococci, and *C. perfringens* for indexing the health hazards in drinking and recreational waters dates back to the late 1800s and early 1900s (32). This occurred shortly after these organisms were first isolated and associated with the fecal wastes of warm-blooded animals. Within the context of the limitations being discussed, such practices were and are sound both on theoretical and practical grounds since it is recognized that (i) there are large number of pathogenic bacteria and viruses potentially present in municipal sewage (67,68), each with its own probability of illness associated with a given dose; (ii) monitoring for each of the pathogens on a routine basis would be a herculean task; (iii) enumeration methods for some of the more important pathogens are unavailable and for the rest are difficult; (iv) pathogen density data are difficult to interpret because the methodology generally is imprecise and inaccurate and because of the meager dose-response data available; and (v) on theoretical grounds, the intent is not to index the presence of the pathogen but rather its potential to be there in sufficient numbers to cause unacceptable health effects.

By no means should the foregoing be construed as suggesting that recreational water quality criteria and the derived guidelines are unnecessary. To the contrary, criteria amenable to risk analysis are absolutely essential. It is evident from the nature of the illness indicator (Y on X) lines and the heavy usage of estuarine and coastal beaches in the United States that large numbers of individuals are becoming ill as a consequence of swimming in sewage-polluted waters. Furthermore, as seen from the Dubuque outbreak (15), the potential for more serious illness exists. Nevertheless, since the illnesses involved are relatively benign, there is undoubtedly a rate which is acceptable; however, *the acceptances of the risks involved should be deliberate decisions with consideration of all the factors involved and with local input*.

A temporary consequence of the application of the criteria may be the withdrawal of certain recreational resources from public use. However, the long range impact should be pollution abatement. This requires better technology for obtaining the data base needed for the translation of the target area criteria which have been developed into effluent guidelines on a case-by-case basis.

The findings from the EPA program have raised a number of questions. One is the nature of the etiologic agent for the gastrointestinal symptomatology. A second is the need for a more human fecal specific and environmentally resistant indicator. This relates to the difficult question of stormwater runoff and nonpoint sources. The third is need for separate criteria for fresh waters. Studies in progress which address these questions should be continued.

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