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Epidemiological studies on swimmer health effects associated with potential exposure to zoonotic pathogens in bathing beach water – a review

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11.1 INTRODUCTION

Humans, animals and birds discharge billions of tons of faecal material into the environment every year. Much of this faecal material reaches water bodies either indirectly through discharge after treatment or directly by being washed off the surface by rainfall or through defecation directly into water bodies. This faecal material can carry pathogenic microbes that may pose a risk to humans exposed to contaminated surface water.
Treated wastewater has been shown to be related to swimming-associated gastrointestinal illness in individuals in contact with contaminated water for recreation. Studies conducted in both marine and freshwater environments have shown that as the level of faecal contamination increases the frequency of gastrointestinal illness in swimmers also increases (Wade et al. 2003, Pruss 1998, Zmiou et al. 2003). Incidence is directly related to the densities of faecal indicator bacteria in the water. This relationship has provided regulators and water resource managers with a means to protect the health of swimmers. Faecal indicators have been used for many years to measure water quality and to maintain the safety of water for swimmers.

One of the shortcomings of faecal indicator bacteria is, however, that they are carried by all warm-blooded animals including birds. Thus, contamination of water by animal and bird faeces cannot be distinguished from contamination by human faeces when using these faecal indicator bacteria. Since the extent to which risk associated with the former differs from risk associated with the latter is unknown, it has been the practice of regulators and water resource managers to consider the risk as being the same for all waters regardless of the source. Current knowledge about the occurrence of pathogens in faeces leads us to believe that the risks related to human and animal faecal sources are not equivalent. A large proportion of the swimming-associated gastrointestinal illnesses (in unchlorinated water) are thought to be caused by viruses (Hendrikson et al. 2001). It is well-known that viruses commonly responsible for gastrointestinal illness (e.g. norovirus, rotavirus) tend to be species-specific. This relationship holds up quite well among the viruses that cause gastrointestinal infections, but less well for respiratory infections, where the species barrier is frequently crossed. The species barrier would imply that the risk associated with exposure to water contaminated by animals and birds would have to be lower than the risk associated with exposure to water contaminated by human faeces; however, this risk difference has not been well defined for individuals who swim in recreational waters.

The main challenge in defining the risk of infection associated with exposure to zoonotic pathogens that may be present in water contaminated with animal/bird faeces is the lack of a technique to determine the source of the faeces and to make quantitative attributions to different sources. Currently an extensive research effort is on-going to develop methods to identify sources of faecal contamination in water (USEPA 2005, Rochelle & De Leon 2006, Santo Domingo et al. 2007). Most of the efforts involve detecting and quantifying highly specific sections of DNA from the genome of bacteria that have established an ecological niche in the gastrointestinal tract of human and non-human species. None of the methods developed to date have the specificity
and sensitivity to be useful for characterizing the source of faecal contamination in recreational and other surface waters. In the absence of effective source identification methods, risk assessment remains confined to studies at sites where the dominant or only source of faecal contamination is known. This approach has been successful in studies of health effects in swimmers exposed to point sources of pollution, such as treated wastewater effluents.

Few studies have addressed the health effects in swimmers exposed to bathing beach waters contaminated by animal and bird faecal wastes. Only two studies in the literature were specifically designed to answer the question whether an excess of swimming-associated health effects can be related to exposure to waters affected by non-point source faecal wastes (Calderon et al. 1991, Colford et al. 2007). In a third study, which was designed to look at health effects associated with polluted waters, two of nine beaches were contaminated with animal faecal wastes and swimmer exposure at these beaches may be helpful in answering our question (Cheung et al. 1990). One other study was designed to determine if health effects were different in populations exposed to rural run-off conditions as opposed to exposure to beach waters affected by human wastewaters treated with oxidation pond processes (McBride et al. 1998). These studies will be described in some detail to determine if a general conclusion can be drawn from their results with regard to health effects associated with exposure to animal and bird faecal contamination of bathing waters.

11.2 HONG KONG STUDY

In 1990, Cheung et al. reported the results of epidemiological studies undertaken in 1986 and 1987 in Hong Kong. Although these studies were not conducted to determine the effect that animal faeces-contaminated beach waters might have on exposed swimmers, the data generated by these studies may have a bearing on the issue. The group studied water quality and swimmer health effects at nine beaches. Six of the beaches were contaminated by human sewage discharged through sea water outfalls or storm water drains. Two of the beaches (“Old Cafeteria” and “New Cafeteria”) were contaminated by livestock waste (mainly pig excreta) discharged from the mouth of a river. One of the nine beaches had mixed human and animal faecal wastes as the source of contamination of the beach.

Water samples were obtained from beaches on weekend days every two hours between 9 a.m. and 5 p.m. The water samples were tested for nine water quality indicators, including E. coli and enterococci.

The study population included beachgoers recruited at the beach. Follow-up telephone interviews were conducted the next day to collect demographic and swimmer activity information. A second telephone interview was conducted
seven to ten days later to determine if any episode of illness had occurred since the swimming experience.

Swimmers were defined as individuals having significant exposure of upper body orifices to beach water. Non-swimmers were beachgoers who did not immerse their heads in beach water. The health endpoint, highly credible gastrointestinal illness (HCGI), was defined as any one of the following: vomiting; diarrhoea with fever or a disability condition; and nausea or stomachache accompanied by a fever.

We re-analyzed the data reported in Cheung et al. (1990) with respect to source and swimmer illness. The data for both beach water quality and health status from the two animal waste-affected sites were combined. This was also done with the data from six of the seven remaining beach sites that were contaminated by faecal wastes from sewer outfalls or stormwater drains. The seventh site was not included in the analysis because the faecal sources were described as being from a sewage outfall and a river, a mix of both human and animal faecal contamination. The data extracted from Table 11.3 of the Cheung study are shown in Table 11.1. The average E. coli density at the animal faeces-contaminated beach sites was 978 per 100 ml, with a range of 243 to 1714 E. coli per 100 ml. At the human sewage contaminated beach sites the average E. coli density was 187 per 100 ml, with a range of 69 to 269 per 100 ml.

### Table 11.1 Analysis of data from Cheung et al. (1990) for swimmer-associated gastroenteritis among beaches grouped by source of faecal contamination.

<table>
<thead>
<tr>
<th>Faecal source</th>
<th>E. coli density (range, per 100 ml)</th>
<th>Category</th>
<th>Total number</th>
<th>Number ill</th>
<th>p Value&lt;sup&gt;1&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Animal</td>
<td>243–1714</td>
<td>Swimmers</td>
<td>960</td>
<td>2</td>
<td>0.5246</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-swimmers</td>
<td>366</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Human</td>
<td>69–269</td>
<td>Swimmers</td>
<td>11,748</td>
<td>25</td>
<td>0.0418</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Non-swimmers</td>
<td>3368</td>
<td>2</td>
<td></td>
</tr>
</tbody>
</table>

<sup>1</sup>p-value for the difference between swimmer/non-swimmer illness rates based on Fisher’s exact test.

The total number of individuals who swam at the animal faeces-contaminated beach waters was 960; 366 non-swimmers at these beaches participated in a study. In these two groups HCGI illness was observed in two of the swimmers while no HCGI illness was observed among the non-swimmers. At the beaches with bathing waters contaminated by human sewage 11,748 swimmers
participated in the study and 3,368 individuals participated as non-swimmers. Twenty-five of the swimmers and two of the non-swimmers suffered HCGI illness. Fisher’s Exact test was used to determine if it was reasonable to assume that illness rates in swimmers and non-swimmers were identical.

The results of this statistical analysis indicate that the illness rate in swimmers was different from that of non-swimmers only at beaches that were contaminated by human sewage. At beaches contaminated by animal faecal wastes no significant difference in illness rate was observed between swimmers and non-swimmers despite much higher levels of faecal contamination at the animal impacted sites. The mean *E. coli* densities in the beach waters contaminated with animal faeces were about five times greater than those in the beach waters contaminated with human sewage. Enterococci densities in the animal contaminated waters were about twice the mean density observed in the waters contaminated with human sewage. Although the resolution to detect a difference was low, since very few respondents reported episodes of gastroenteritis, one interpretation of these analyses is that exposure to animal faeces carries less risk than exposure to human sewage.

### 11.3 CONNECTICUT USA STUDY

In 1991 Calderon *et al.* published a prospective epidemiological study at a beach on a small freshwater pond located in central Connecticut, USA. The pond, an impoundment with a surface of about three acres, was formed by damming a river. The pond was fed by two small streams. One side of the pond had a sandy beach about 200 yards in length. There were no human sources of faecal contamination in the surrounding watershed contributing to the streams that fed the pond. There were no direct sources of human faecal contamination of the pond either, such as leaking septic tanks. The main source of the faecal contamination of the pond was associated with bird or animal faeces from the surrounding forestland.

The study participants consisted of families from the local community who had exclusive use of the beach area. Community members frequently used the pond for swimming. Attendance at the beach was recorded daily by study participants. The health status of beach attendees was recorded in a daily diary kept by each family member. Gastrointestinal illness was defined as having experienced any of the following symptoms: vomiting, diarrhoea, stomachache or nausea. If symptomatic illness occurred one, two or three days after a swimming experience it was designated as swimming-associated. Exposure was defined as complete immersion of the head and body beneath the surface of the water. Individuals who swam at other locations were excluded from the study.
Multiple indicators of water quality were measured daily over the course of the bathing season. Water samples were collected daily at two sites at the beach at 10 a.m., 2 p.m. and 5 p.m. Water samples were analyzed for *E. coli*, enterococci, faecal coliforms, *Staphylococcus* and *Pseudomonas aeruginosa*. Rainfall also was measured daily.

The results of the study indicated that there was significant swimming-associated gastrointestinal illness, but that it was not related to the level of faecal indicator bacteria in the water. A strong relationship between illness and swimmer density or *Staphylococcus* density led the authors to conclude that the excess gastrointestinal illness in swimmers was probably caused by swimmer-to-swimmer transmission and not by exposure to faecal contamination of animal or bird origin.

11.4 NEW ZEALAND STUDY

In 1998 McBride *et al.* reported the results of epidemiological studies conducted at beaches contaminated by human faecal contamination from oxidation ponds (three beaches) and beaches contaminated by run-off from rural areas contaminated by animal faeces (two beaches). Two control beaches with minimum faecal contamination impact were also included in the study.

Water samples were collected twice daily at 11 a.m. and 3 p.m. on weekends and holidays at three locations along each beach. Each sample was tested for *E. coli*, faecal coliforms and enterococci using membrane filter techniques.

Multiple health endpoints were used, including highly credible gastrointestinal illnesses (HCGI). The symptoms associated with HCGI in this study included vomiting; loose bowels with fever; loose bowels with disability (one or more days away because of illness or days unable to do normal activity, medical advice sought or hospitalization); nausea with fever; or, indigestion with fever.

Three categories of exposure were defined: swimmers, paddlers, and non-exposed individuals. Exposure was defined as the act of entering the water. Swimmers were individuals who immersed their head beneath the surface of the water. Paddlers were individuals who entered the water but did not immerse their head beneath the surface.

Potential study participants were recruited at the beach. The initial contact was used to gather information about swimming exposure and demographic information. At this time questions were also asked about the types of food recently eaten and details were collected for a follow-up contact.

The follow-up questionnaire was designed to determine types of swimming activity, symptoms that were evident since a swimming experience or food types ingested since the swimming event. Questions were also asked to determine if
other family members had become ill or if the participants had had contact with animals.

None of the faecal indicator bacteria occurred at very high densities. The median values for enterococci at all beaches were less than 10 MPN per 100 ml. The median values for \textit{E. coli} at the control and rural beaches were approximately 10 per 100 ml or less. \textit{E. coli} median values in beach waters with possible human faecal contamination ranged from less than 10 to about 30 per 100 ml. All of the median faecal coliform densities were less than 30 per 100 ml.

The relationship between health effects associated with swimming and \textit{E. coli} or faecal coliforms did not show any increase in illness risk through increasing quartiles. Similarly, the risk for exposed groups (those entering the water or entering the water and immersing their heads beneath the surface of the water) did not show a statistically significant association with increasing quartiles of enterococci concentrations.

These findings were associated with swimmers from all of the beaches regardless of the source of the faecal contamination. The combination of data from beaches where swimmers were exposed to faecal contamination from different sources made it difficult to determine if exposure to one source or the other posed a greater risk to swimmers. The authors indicate that, “No evidence has been found to suggest any merit in separating beachgoers illness risk on the basis of the types of faecal material present (i.e., from rural areas versus from oxidation ponds treating human wastes).” They also concluded, “Illness risks at control beaches were significantly lower than at beaches believed to be impacted by oxidation pond effluent and by rural runoff.” The authors indicated that because of very low densities of faecal indicator bacteria in the waters at the beaches, exposure may not have been great enough to elicit an observable health effect in swimmers.

11.5 SAN DIEGO, CALIFORNIA STUDY

In January 2007, Colford \textit{et al.} published a report describing epidemiological studies expressly conducted to examine health effects experienced by swimmers and the relationship of these effects to water quality indicators in water predominantly contaminated by non-human faecal sources. Their study was conducted on Mission Bay in San Diego, a 2,287 acre man-made estuary. The Bay has 27 miles of shoreline, 19 of which are sandy beaches. Multiple beach sites were used for the epidemiology study. This study was unique, in that the year before the epidemiology study was initiated, a source identification project (Gruber \textit{et al.} 2005) had been conducted to determine the sources of faecal contamination of the Bay. Faecal sources were identified with two separate source identification methods, one a library method (ribotyping) and the other a
non-library method (Polymerase Chain Reaction). The results of this bacterial source identification study showed that the major source of faecal contamination to the Bay were birds and that less than 9% of the faecal contamination was from human sources.

In this prospective cohort study participants were recruited each sampling day and their current health and degree of exposure to the water were recorded. Six beaches in the Mission Bay were used for the study, which was conducted on weekends and holidays.

Water quality samples were collected at the six selected beaches at eighteen sites, with the number of sites per beach ranging from two to five depending on the beach length and anticipated swimming activity. The quality of beach waters was measured using three traditional faecal indicator bacteria, enterococci, faecal coliforms and total coliforms.

Study participants recruited at the beach had to meet certain criteria including (1) they could not have participated previously in the study; (2) at least one family member had to be 18 years old or older; (3) they had to have a home address in the United states, Canada or Mexico; and (4) they had no history of swimming in the previous seven days. Participants were asked to complete a questionnaire about possible exposures at the beach and illnesses experienced in the previous two to three days, prior to departure from the beach. Follow-up telephone interviews were conducted about 14 days after the beach visit. Participants were interviewed about demographic information; swimming and other exposures since the beach day; pre-existing health problems and health problems experienced since the beach visit.

Health outcomes included gastrointestinal (GI) illness, respiratory symptoms, dermatologic symptoms and other non-specific symptoms. GI symptoms included nausea, vomiting, diarrhoea and stomach cramps. Grouped symptoms were defined as HCGI-1 (vomiting, diarrhoea and fever or cramps and fever) and HCGI-2 (vomiting plus fever).

Eight thousand seven hundred and ninety-seven of the enrolled participants completed the follow-up telephone interview. The results of the study showed that there was a significant excess of diarrhoea among swimmers. However, there was no correlation between traditional water quality indicators (Enterococcus, faecal coliforms or total coliforms) and the risk of illness. Although swimmers experienced more diarrhoea than non-swimmers and the incidence of symptoms increased with increased exposures, an increased risk was not observed for the more severe symptoms, such as fever, vomiting or HCGI-1 or HCGI-2. The authors concluded, “Our findings do not agree with earlier studies reporting association between bacterial indicators of water quality and illness. We believe these results are due to a lack of human sources of
traditional faecal indicator bacteria, supported by our lack of virus detection and an independent microbial tracking survey.”

11.6 OTHER STUDIES

Outbreaks of human infection with zoonotic pathogens have often been linked to bathing or incidental contact with untreated surface water (Kramer et al. 1998, Ackman et al. 1997). Most, however, have identified other bathers rather than animals as the likely source of contamination. A large outbreak of E. coli 0157: H7 in Swaziland (Effler et al. 2001) and several cases of E. coli 0157:H7 (Ihekweazu et al. 2006) were associated with contact with untreated surface waters where cattle were implicated as the likely source, but definitive linkages to cattle faeces were not established. Outbreaks in drinking-water systems, such as the outbreak in Ontario (PPHB 2000, Hrudey 2003) of E. coli 0157: H7 and Campylobacter spp., also provide indirect evidence of the transmission of zoonotic illnesses through contact with untreated water. The municipal water supply was contaminated by nearby livestock demonstrated to be infected with genetically identical strains of the responsible pathogens. While the Walkerton example is not directly applicable since, for obvious reasons, drinking-water exposures would be considerably greater than bathing water exposures, it does support the general plausibility of similar situations involving contamination of untreated surface waters and recreational or other incidental contact.

Case-control studies of the five zoonotic pathogens addressed in this book: Campylobacter spp. (Denno 2009, Schoneburg-Norio 2004), Cryptosporidium parvum (Pintar 2009, Roy 2004), E. coli 0157:H7 (Denno 2009, Slutsker 1998, Werber 2007), Salmonella spp. (Denno 2009) and Giardia spp. (Stuart 2003) have identified exposures to untreated surface waters as a significant risk factor for infection and illness. In the northwestern United States, a recent study attributed 10 per cent of sporadic Campylobacter and 21 per cent of sporadic Salmonella infections to swimming in or contact with a natural source of water (Denno 2009). None of these studies, however, made an attempt to characterize the sources of contamination affecting the water bodies. Since case-control studies investigate many different exposures, detailed investigations of all exposures are usually not feasible.

11.7 CONCLUSIONS

The epidemiological studies reviewed in this chapter do not provide evidence for associations between swimming-associated gastrointestinal illness and exposures to bathing waters contaminated with faeces from animals or birds. Other studies,
such as outbreak investigations and case-control studies, have provided logical linkages to human infections with zoonotic pathogens and recreational or occupational exposures to water, but they have not established a definitive link between water contamination and specific animal sources.

Three of the prospective studies were conducted in marine or estuarine waters and one was conducted at a freshwater beach. Two of these studies were specifically designed to answer the question “Do bathing waters contaminated by non-human faeces pose a risk to swimmers?” In both studies efforts were made to ensure that animals and birds were the dominant source of the faecal contamination. This was done in one case by surveying the watershed and determining human wastes were not being discharged into streams that fed into the bathing water area (Calderon 1991). In the second case (Colford 2007) an extensive pre-study was performed using microbial source identification methods to determine if human faeces were contaminating the beaches. In the third case (Cheun 1990) the dominant source of non-human faecal contamination was identified as being related to wastes that were discharged to a river that contaminated the water at two of the beaches that were studied. In the fourth case (McBride 1998) the beaches studied were characterized as receiving discharges from oxidation ponds, rural runoff or not receiving known sources of faecal contamination. The sources of faecal contamination were not specifically identified.

The levels of faecal contamination at the study locations, as measured by faecal indicator bacteria, were not unusual. Enterococci and E. coli were measured at three of the study locations while only enterococci was measured at the San Diego location. The enterococci geometric mean densities per 100 ml at all four locations ranged from 17 to 144. The geometric mean E. coli densities per 100 ml at the Hong Kong, Connecticut and New Zealand locations ranged from 25 to 1,705. In similar epidemiological studies conducted at beaches contaminated by human sources (sewage effluents) with indicator densities at these same levels, swimming-associated gastroenteritis has been frequently observed. In the study where the beaches were characterized as receiving rural run-off, the densities of faecal indicator bacteria were very low, averaging eight per 100 ml at 1 beach and 30 per 100 milliliters at a second beach. This unusually low level of faecal contamination is likely to have hindered the ability to draw clear-cut conclusions about whether rural run-off is related to swimming-associated gastroenteritis.

All of the studies used a health endpoint similar to or identical to the Highly Credible Gastrointestinal (HCGI) symptomatology first described by Cabelli (1983), in which combinations of the symptoms (vomiting, diarrhoea, nausea and fever) were used to confirm a case of gastroenteritis.

After observing the results of these studies the question can be asked, can the lack of excess faecal-associated illness in swimmers be taken as evidence that animal
contaminated waters do not pose a health risk to swimmers? One possible explanation for the observations is that the conditions under which these studies were conducted prevented finding a health effect in swimmers. These studies were conducted at sites where water quality standards were based on data gathered from human epidemiological studies at sites impacted by human sewage. This is important because human sewage is usually treated and disinfected before being discharged into surface waters. Treatment and disinfection significantly re-arrange the relationship between faecal indicator organisms (FIO) and potentially present pathogens. The ratio of FIO to pathogens is changed from a very high ratio to a very low ratio after disinfection and it is the latter ratio which is used to develop bathing beach standards. On the other hand, faeces from animals are not treated and the high ratio of FIO to pathogens that are observed at the animal source may be maintained for long periods. This could result in levels of FIO that would not be allowed at a beach, or if distances were involved, dilution or die-off of pathogens would take place before faecal wastes reached a beach. In light of this scenario, the conclusions drawn above do not completely answer the question whether exposure to animal-contaminated waters poses a health risk to swimmers. The exposure to zoonotic pathogens is unlikely to have occurred at beaches meeting local beach water quality standards.

The real question remains whether epidemiological studies to determine if swimming associated gastroenteritis is related or not to exposure to waters contaminated by animal or bird faeces can be properly conducted under conditions where animals or birds are the source of recreational water contamination. There are many impediments to carrying out the necessary prospective cohort types of studies.

First, because of the putative low frequency of illness associated with zoonotic pathogens, large populations would need to be recruited. Such large study populations might not be available at rural beaches where animal exposures might take place.

Second, faecal indicator bacteria may occur in very high numbers, relative to the numbers of zoonotic pathogens. Unlike the wastewater treatment plant systems used for eliminating faecal indicator bacteria and pathogens from human faecal wastes, the wastes from animals are normally not treated, except in the case of confined animal feedlot operations. This means that a waiver from local regulations would have to be obtained to allow high indicator counts at animal-impacted beaches.

Third, some zoonotic pathogens can cause serious disease, especially in children. For example, *E. coli* 0157:H7 not only causes severe gastrointestinal disease, but also haemolytic uremic syndrome which frequently results in the death of children. Therefore, a study at a site which is contaminated enough to
elicit health effects may pose too great a health risk for severe illness and infection and thus raise ethical issues related to conducting an epidemiological study.

A fourth challenge is that traditionally designed epidemiology studies may be poorly suited to quantifying and understanding the sporadic and episodic risks associated with animal wastes. Furthermore, a single study cannot encompass risks from all types of animal waste (cattle, birds, pigs, etc.) and no study to date has addressed the risks associated specifically with cattle waste. While the studies considered here had no obvious major flaws in design, the impact at the specific sites may not have been enough to allow for a risk to be detected, or a risk may only have been present following a heavy rain or specific sporadic contamination events, which also in turn, could affect swimming behaviour and decrease the likelihood of exposure.

In the presence of all these formidable barriers and confounding factors, which severely hinder our ability to quantify the relationship between water quality and swimming-associated disease caused by zoonotic pathogens, some other means will have to be used to measure the risk posed by the presence of zoonotic pathogens in bathing beach waters. Alternate approaches to traditional prospective (observational or randomized/intentional exposure) epidemiology studies should be explored to better quantify and understand health risks from waters contaminated with animal faeces. Approaches could include case-control studies targeting specific zoonotic pathogens combined with detailed source characterizations, or studies targeted at highly exposed populations. Incorporation of human-specific indicators into studies can help better define exposure. Future epidemiological studies designed to address this issue must carefully consider site selection, sample size, exposure and measurement of appropriate indicators.

Quantitative microbial risk assessment has been proposed as another alternative approach to defining risks associated with zoonotic pathogens. However, it is generally agreed that this is not an ideal substitute for epidemiological evidence. Until the time when information is available to regulators for developing water quality criteria for waters contaminated by non-human faecal wastes, they may be left with no other choice than regulating animal-polluted bathing water as if it poses the same risk as human-contaminated bathing water.

REFERENCES


