Epidemiology

Illness associated with contamination of drinking water supplies with phenol

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Abstract

In January 1984 the River Dee in north Wales was contaminated with phenol, with subsequent contamination of the tap water received by about two million consumers. A retrospective postal survey of 594 households was undertaken to determine whether consumption of this contaminated water was associated with illness. Subjects in areas that received contaminated water reported significantly more gastrointestinal illness than those in a nearby unexposed area (32·6% v 8·7%, p<0·00001) as well as reporting a higher incidence of any symptoms (43·6% v 18·4%, p<0·00001). Symptoms were consistent with phenol poisoning and bore a strong temporal relation to the pollution of the supply, but they developed at concentrations of phenols previously considered to be safe by the water authorities concerned. Chlorophenols produced during the treatment of water may have aggravated the problem.

Introduction

Water extracted from the River Dee in north Wales is distributed to about two million consumers in north western England, including Liverpool and Chester, and north eastern Wales. Although the main catchment area of this supply is the sparsely populated Welsh uplands, there are chemical industries upstream of the major extraction points. On about 25 January 1984 the river became polluted with an unknown quantity of chemicals, consisting of phenol and a much smaller amount of 2-ethyl-hexanol, a relatively inert plasticiser.[1] Subsequent chlorination of the water during routine treatment converted much of the phenol to monochlorophenols, dichlorophenols, and trichlorophenols, which imparted a strong medicinal taste to the water.

The water authorities were not aware that their supply had been badly contaminated until after the polluted water had reached some consumers. Initial assessment of the concentrations of phenols in tap water and the belief that the foul taste would limit the amount of water drunk led the water authorities to reassure the public and public health officials that although the water was unpleasant, it posed no important health risk. During the next week, however, anecdotal reports of an increase in gastrointestinal complaints were

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noted. This study was undertaken to assess whether there was an actual increase in incidence of illness during the period of the pollution and, if so, whether this increase was related to consumption of polluted water.

Subjects and methods

Households from three geographical areas were sampled. We approached 250 households in an area that received unpolluted water, which was not from the River Dee (unexposed area), 250 households that received water from the River Dee diluted in reservoir A (high exposure area), and 94 households that received water from the River Dee after dilution in reservoir B (low exposure area).

Information was obtained by a postal questionnaire, which was sent on 10 February. A second questionnaire and covering letter were sent to all those who had not returned the original questionnaire within two weeks. Questionnaires were sent to every 100th name on the electoral roll in the exposed area and every 25th name in the unexposed area. Information was requested for all members of the household and included age, sex, normal source of drinking water, whether tap water was drunk during the incident (drunk alone or mixed with soft drinks, baby foods, soup, tea, coffee, etc), sources of water drunk outside the home during the incident, symptoms experienced during 26 January to 1 February, and whether medical advice was sought for these symptoms.

Estimated concentrations of phenols in the two exposed areas were provided by the relevant water authority. The concentration of the pollutants in the low exposure area was roughly half that in the high exposure area for the first 24 hours, but after that time they were similar (table I). The unexposed area was supplied by a private water company that provided half of its customers with water from the River Dee and half with unpolluted upland water. The local press had reported that the tasteless phenol had contaminated local water without specifying which portions of the distribution system were implicated. Thus subjects in the unexposed area were unsure whether they had received polluted water even though they did not have bad tasting water.

Results were analysed statistically by the standard χ^2 test or by estimating two tailed p values by doubling Fisher's exact one tailed probability.[2] The use of the χ^2 test was an oversimplification as observations within households were not truly independent. Adjustment for this, however, would not have appreciably affected the results as the relevant χ^2 values were large.

The possibility of a concurrent localised outbreak of infectious gastroenteritis biasing the results was investigated by recording the number and geographical distribution of all people from whom pathogenic organisms were identified in stool specimens submitted to the Public Health Laboratory Service microbiology laboratories at Rhyl, Liverpool, and Chester. These laboratories examine most of such specimens in the areas studied.

The water authorities were approached for details of the incident and further information on chemical analyses performed.

Results

Half of the initial questionnaires were returned within two weeks. After the letter of reminder had been sent we received a total of 172 questionnaires (69%) from the unexposed area, which provided information on 448 people; 73 questionnaires (77%) from the low exposure area, which provided information on 213 people; and 181 questionnaires (72%) from the high

exposure area, which provided information on 541 people. Of the possible initial comparisons between the three groups, that of the high and low exposure areas was the first choice because during the incident the likely concentrations of chemicals were similar (table I). This analysis did not show a significant difference for any of the reported outcomes between the two exposure areas and therefore results for these areas were combined (exposed areas).

TABLE I—*Estimated chlorophenol and phenol concentrations in two service reservoirs (μmol/l) (all chlorophenols were assumed to be in the form of trichlorophenols)*

| Date | Reservoir A | | Reservoir B | |
	Chlorophenol	Phenol	Chlorophenol	Phenol
26 January	0·43	0·11	0·2	0·05
27 January	0·25	0·05	0·25	0·05
28 January	0·13	<0·01	0·15	<0·01
29 January	0·08	<0·01	0·08	<0·01
30 January	<0·03	<0·01	<0·03	<0·01

Conversion: SI to traditional units—Chlorophenol: 1 μmol/l 197 μg l. Phenol: 1 μmol l = 94 μg l.

A broadly similar sex distribution (213 men out of 445 (48%) people in the unexposed area and 379 out of 748 (51%) in the exposed areas), age distribution (mean age was 43·8 in the unexposed area and 39·1 in the exposed areas), and usual water source (97% use of mains in the unexposed area and 99·7% in the exposed) were found in both study areas. During the incident, however, as might be expected with the abnormal taste, the number of people drinking tap water was significantly lower in the exposed areas (522 out of 736 (71%)) than in the unexposed area (397 out of 439 (90%)) ($\chi^2 = 61·4$ with 1 df, $p < 0·00001$).

Respondents were questioned about whether they had experienced any one of seven specified symptoms. A significant increase in the reporting of each of the specific symptoms was noted in subjects in the exposed areas compared with subjects in the unexposed area (table II). Highly significant differences were also noted in the number of subjects reporting gastrointestinal illness (defined as nausea, vomiting, diarrhoea, or abdominal pain) as well as those reporting any symptom. The distribution of illness by age was fairly uniform (figure).

Separate analysis of the data according to whether or not respondents claimed to have drunk the water during the incident (table III) strengthened the associations of exposure with symptoms, although people in exposed areas who did not drink any water may have absorbed polluted water by other means—for example, from cooked food and ice.

To explore the possibility that symptoms reported might have been biased by the presence of bad tasting water the analysis was repeated comparing

TABLE II—*Number (%) of people reporting symptoms*

| | Study area | | χ^2 | p |
	Unexposed (n = 448)	Exposed (n = 754)		
Diarrhoea	24 (5·4)	124 (16·4)	32	<0·00001
Nausea	26 (5·8)	185 (24·5)	68	<0·00001
Vomiting	7 (1·6)	39 (5·2)	10	0·00500
Abdominal pain	32 (7·1)	130 (17·2)	24	<0·00001
Headache	36 (8·0)	161 (21·4)	36	<0·00001
Rash	13 (2.9)	42 (5·6)	4·6	<0·05000
Malaise	28 (6·2)	131 (17·4)	30	<0·00001
Other	23 (5·1)	115 (15·3)	28	<0·00001
≥1 symptom	84 (18·4)	329 (43·6)	77	<0·00001
Gastrointestinal symptoms	39 (8·7)	246 (32·6)	88	<0·00001
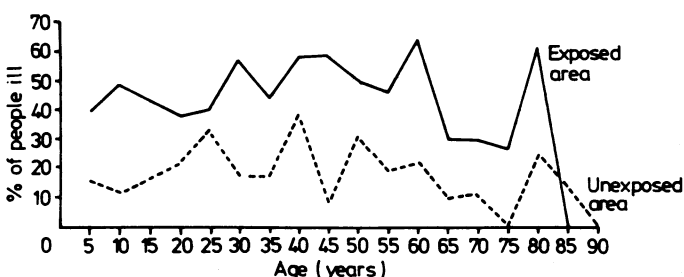
Proportions of people ill in exposed and unexposed areas by age.

respondents in the two areas who had bad tasting water with those who had not noticed a bad taste (table III). In the exposed area the presence of a bad taste did not significantly increase the incidence of reporting symptoms (Fisher's exact test, $p = 0·06$ for any symptoms, $p = 0·46$ for gastrointestinal symptoms). Contaminated water, however, would not necessarily taste abnormal.

The day of onset of illness among subjects in the unexposed area was fairly uniformly distributed over time, whereas the onset among those in the exposed area suggested a "point source" outbreak (table IV). The illness reported appeared to be self limiting, lasting one to 48 hours after onset. Only 0·8% of those who reported symptoms sought medical help for their symptoms.

TABLE III—*Illness among subgroups within study areas expressed as number ill per total in group (and %)*

| | Study area | | Probability* |
	Exposed	Unexposed	
Drank water†:			
Any symptoms	289 522 (55·4)	81 397 (20·4)	0·0001
Gastrointestinal symptoms	224 522 (42·1)	38 397 (9·6)	0·0019
Did not drink water:			
Any symptoms	40 214 (18·7)	2 42 (4·8)	0·0307
Gastrointestinal symptoms	22 214 (10·3)	0 42	0·0321
Bad tasting water‡:			
Any symptoms	317 712 (44·5)	22 90 (24·4)	0·0003
Gastrointestinal symptoms	235 712 (33·0)	18 90 (20·0)	0·0142
No taste to water:			
Any symptoms	12 42 (28·6)	62 355 (17·5)	0·1331
Gastrointestinal symptoms	11 42 (26·2)	21 355 (5·9)	0·0003

*Double Fisher's exact one tailed test.
†Eighteen subjects in exposed area did not indicate whether they had drunk water or not, but none were ill; nine in the unexposed area failed to indicate whether they had drunk water, but one had gastrointestinal symptoms.
‡None in the exposed area and only three in the unexposed area (none ill) failed to indicate whether their water tasted "funny."

TABLE IV—*Date of onset of symptoms*

| Date | Study area | |
	Exposed	Unexposed
20 Jan	–	2
21 Jan	3	4
22 Jan	–	–
23 Jan	1	1
24 Jan	1	1
25 Jan	8	5
26 Jan	47	2
27 Jan	57	5
28 Jan	42	3
29 Jan	22	2
30 Jan	19	6
31 Jan	14	5
1 Feb	7	2
2 Feb	3	1
3 Feb	–	3
4 Feb	–	2
5 Feb	–	1
6 Feb	2	3
7 Feb	–	2
8 Feb	–	1
9 Feb	–	–
10 Feb	1	1
11 Feb	–	–
Total	**227**	**52**

Discussion

Several potential biases must be considered when interpreting the results of this survey. The method we used to select the households contacted might have led to overrepresentation of large households. As the same method was used in all study areas this was unlikely to create a significant bias, although the extent that the results can be generalised may be decreased.

The overall response to the questionnaire was only 72%, but there was little difference in response between the exposed and non-exposed areas. Furthermore, a worst case analysis of non-responders (assuming that all unexposed non-responders were ill and all exposed non-responders not ill) showed that in this improbable event the difference between the two groups in the incidence of gastrointestinal illness would remain at a borderline significance ($p = 0·06$, calculated assuming that a non-responding and a responding household had the same average number of occupants).

A few of the respondents may have misunderstood the questionnaire in so far as they gave dates of onset after 1 February despite the fact that the inquiry concerned symptoms during the week 26 January to 1 February (table IV). Exclusion of these cases (largely from the unexposed area), however, would serve only to exaggerate further the effect of exposure.

Although the bad taste of the water itself may have led to psychosomatic symptoms, this effect was insufficient to produce significant increases in the reporting of symptoms, even in the exposed area. Analysis of laboratory data on specimens submitted suggested that people in the study areas experienced some gastrointestinal infections but that this was uniform throughout the study areas.

The principal pollutants in the tap water during this incident were phenol and chlorophenols (about 80% were 2,4,6-trichlorophenol in the only detailed analysis available from the exposed area). Phenol itself is highly miscible with water, almost tasteless in pure solution, readily reactive with other chemicals, rapidly absorbed by man through the gastrointestinal tract or skin, and mostly cleared by the kidneys within 24 hours.[4,5] Poisoning at high concentrations produces corrosive local damage and subsequent systemic shock, with a fatal dose being 80-1300 mg/kg.[6,7]

In an incident of contamination of groundwater in Wisconsin in 1974 subacute poisoning, characterised by nausea, vomiting, diarrhoea, burning sensation, and sores in the mouth, together with dark urine was shown to occur at concentrations of phenol above $1 \cdot 1$ μmol/l ($0 \cdot 1$ mg/l) in drinking water,[8] and this finding was subsequently reinforced by an episode of minor gastrointestinal illness in Georgia after the contamination of drinking water with a mixture of phenols from the lining of a solar water tank at concentrations of $3 \cdot 7$ μmol/l ($0 \cdot 35$ mg/l).[3] These symptoms appear to represent an irritant effect on the gastrointestinal mucosa related to local concentrations as much larger systemic doses (70 mg) can be tolerated when absorbed from aerosols.[4] The United States of America Environmental Protection Agency, when deriving its maximum permissible concentration of 37 μmol/l ($3 \cdot 5$ mg/l) for phenol in ambient water, used just such data on systemic toxicity and did not discriminate the separate influences of concentration and portal of entry.[9]

Treatment of water for drinking is intended essentially to clarify and then disinfect the water. Chemical pollutants are probably affected primarily by the excess of highly reactive chlorine that is added as a disinfectant. In the absence of other reagents or high temperatures the chlorination of phenol proceeds largely to 2,4,6-trichlorophenol with smaller amounts of lower chlorophenols[10]; in reports these chlorophenols tend to be subsumed under the generic term phenols.[1] Some authors suggest, however, not only that chlorination of phenol increases its toxicity[11] but also that the individual chlorophenols have different properties, 2,4,6-trichlorophenol being the most toxic isomeric form and having appreciable carcinogenic properties in rats and mice.[10] There is also potential for further chemical alterations of these chlorophenols before consumption—for example, when polluted water is used for cooking.

The most noticeable result of chlorinating water containing phenol, however, is to confer a very unpleasant taste to the water due to the production of monochlorophenols and dichlorophenols; this is the principal criterion on which international and local standards for the permitted concentrations of phenol in treated drinking water are based (recommended limit $0 \cdot 01$ μmol/l (1 μg/l).[12] The final product, 2,4,6-trichlorophenol, however, is relatively tasteless so that over a few hours superchlorination may effectively remove the unpalatable taste while increasing the toxicity of the results.[13]

During this incident in an area adjacent to those studied a concentration of phenols of about 21 μmol/l (2 mg/l) was measured in a bulk supply system before further dilution with uncontaminated water (North West Water Authority, personal communication, 1984). In view of this and the relatively short duration of the peak contamination and the small number of samples taken, actual concentrations in the study area were probably higher than the estimates given in table III, even allowing for dilution in the reservoirs. The findings of this study, therefore, are not only compatible with previously documented incidents[1,8] in terms of clinical features but may also be compatible in terms of the concentrations to which consumers were exposed.

The consumers were exposed during this incident to a mixture of phenol and several chlorophenols. The most extensive experience of exposure to phenols in man has been from industrial exposure to pure phenol in aerosols, and these data may underestimate the potential toxicity of phenol pollution in which different routes of absorption are implicated and other compounds are produced during intermediate processing.

The data presented strongly support the hypothesis that consumers who drank tap water that had been contaminated by phenols developed illness. Insufficient data were available to determine whether this was a result of consumers receiving concentrations of phenol known to be toxic or a result of the complex mixture of phenol and chlorophenols, each of which was below the previously reported toxic range. Appropriate epidemiological investigations may provide the only relevant information on the effects of actual pollution (as opposed to industrial exposure) on public health and should be routine in major incidents such as this.

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A healthy active young man is awakened several times a night by painful erections. On each occasion he gets up and passes urine (usually only a small volume) after which his erection subsides. He has a regular and satisfactory sexual relationship with his girlfriend. Physical examination is normal and although rather preoccupied with his physique he seems psychologically well adjusted. What could be the basis for his symptoms and what treatment is advised?

Nocturnal erections are normal and occur during periods of so-called REM sleep. Nocturnal penile tumescence studies have shown that in men aged between 30 and 70 erections occur for periods totalling about 100 minutes in a period of 400 minutes sleep. These do not usually cause the sleeper to awake, but if sleep is shallow, restless, or disturbed for some other reason then the person is made aware of their presence. In the case in question the patient may well be aroused by an uncomfortable erection associated with a full bladder. The fact that he apparently voids what is described as a small volume of urine does not discount this possibility. The bladder neck usually fails to relax sufficiently in these circumstances to allow the bladder to empty completely. This possible explanation should be given to the patient with advice to restrict his fluid intake, particularly alcohol, before retiring to bed. Sometimes priapism, which is a persistent painful erection of the penis, is preceded by somewhat prolonged uncomfortable erections. There is an increased incidence of this condition in patients with sickle cell trait.—J C GINGELL, consultant urologist and lecturer in urology, Bristol.