THE RELATIONSHIP BETWEEN THE HARDNESS OF POTABLE WATER AND CARDIOVASCULAR AND ISCHAEMIC HEART DISEASE MORTALITY IN SOUTH AFRICAN URBAN AREAS

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Dissertation submitted in fulfilment of the requirements for the M Sc (Medicine) degree in the Department of Community Health of the University of Cape Town.

1987

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ACKNOWLEDGEMENTS

I would like to sincerely thank the following for their most valuable and appreciated contributions, as listed:

David Bourne ..... Sound advice, Fortran and pub lunches.

Rauf Sayed ..... Statistics sessions and emergency computer aid.

Aneleh Midgley ..... Hard and floppy copies.

Edie, my wife ..... Backup support.

Water Research Commission .. Indirect but generous financial aid.

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ABSTRACT

Studies carried out in a number of countries have revealed statistically significant negative correlations between death rates from cardiovascular disease (CVD) or ischaemic heart disease (IHD), and the hardness of local water supplies, a phenomenon which is known as the "water story".

These findings have not, however, been universal and it was decided that a study carried out in South Africa with its high CVD and IHD death rates, might yield meaningful results to contradict or support existing findings.

In 1983 a pilot study was thus initiated using a spatial model and a more detailed study began in 1984.

This study ultimately involved the correlation of standardized mortality ratios (SMRs) for CVD and IHD with total water hardness and with a number of contributory and associated water quality factors.

The study supported the hypothesised "water story", showing the existence of negative correlations between standardized mortality ratios (SMRs) for both CVD and IHD, and the hardness of potable water, whether measured as total hardness or as its two major contributory cations, calcium and magnesium.

The level of statistical significance at which this correlation occurred, however, varied with differences in methodological approach.

A "population-unweighted" methodology, which was applied to enable comparison with a number of previously published studies, pointed to potassium (a known hypertension normalisor) in permanently hard water as being an important factor.

Problems inherent to each methodological approach have been discussed as has the need for improved data. In this regard, the need for a National water quality data bank has been emphasised.

1. INTRODUCTION

1.1 RETROSPECTIVE OVERVIEW

The existence of high mortality rates for cardiovascular disease (CVD) and ischaemic heart disease (IHD) in the white South African population group is a well recorded phenomenon (1,2). Relatively little has been published, however, regarding local variations in mortality rates, and the possible causes of such variations.

A number of studies carried out in other countries with regard to local variations have revealed statistically significant negative correlations between CVD or IHD death rates and the hardness of local water supplies, a phenomenon generally known as the "water story".

Such correlations were recorded, *inter alia*, by Crawford *et al.* who studied changes in the death rates of 11 county boroughs in England and Wales where the hardness of the water supply had been substantially changed during the previous 30 years (3); by Morris *et al.*, who obtained negative correlations of hardness with all CVD, coronary heart disease, myocardial disintegration, hypertensive heart disease and stroke mortalities (4); by Lacey, who found that the death rate from CVD in fixed cohorts decreased by about 8% for every 100mg/l increase in hardness in the range up to

170mg/l equivalent CaCO3 (5); by Anderson et al., who proposed that the magnesium component of hard water might play a role in IHD mortality by virtue of its relationship to myocardial magnesium (6); by Neri et al., who not only recorded negative correlation with CVD mortality, but with deaths in general (7); and by Dudley et al., who suggested that water hardness was only one of a number of important variables which should be simultaneously analysed in any study of IHD mortality (8).

The British Water Research Centre/Royal Free Hospital Study carried out by Powell et al. (9) warrants special comment in that it did not adhere to the simple correlation model found in most studies but tested the negative correlation obtained between total water hardness and CVD in terms of a broad spectrum of confounding variables of a climatic, geographic, environmental, genetic and socio-economic nature by means of a multiple regression model.

In South Africa a regional study carried out by Leary et al. in 1983 noted an inverse relationship between crude deaths from IHD in white males and the magnesium content of "drinking water" (10). The study should, however, be treated with circumspection as death rates were unstandardized and this has been shown to be a major fault in similar studies (11); the districts selected were mismatched for population, which ranged from 260 to 114 420; six of the 12 districts

were too small to produce realistic rates, having white male populations of less than 5 000; the study was only based on a one-year period (1978) which would not take into account annual variations in mortality, and a later report from the relevant water data bank shows appreciable differences between the raw water data used in the study and the data for treated water as supplied to the consumer (12).

Support for the "water story" has not been universal. For example, Lindemann et al. showed no correlation in their Oklahoma study (13); Mackinnon et al. showed no correlation between sudden cardiac deaths and hardness or magnesium contents of the drinking water in five Yorkshire cities (14); Bierenbaum et al. reported the highest CVD mortalities in hard water areas in Kansas (15); Allwright et al. claimed that the lowest death rates were found in areas with the softest drinking water and argued that previous studies had not assessed the hardness of the water actually consumed (16); Heyden in a review article questioned the very existence of a "water factor" (17) and Punsar, after thorough review, believed the association of CVD deaths with soft water to be "spurious" (18).

In Holland, research carried out by Zielhuis et al., which ultimately contributed to a Government report on health aspects of water softening (or potential hardening) showed that the negative correlation which had initially been

observed between water hardness and CVD mortality in females became weaker as the study was refined by increasing the sample size and period, until the results were no longer statistically significant at the 0,05 level (19).

1.2 STUDY OBJECTIVES

These can be summarised as follows:

(i) To carry out a South African study with regard to the hypothesised inverse relationship between water hardness and CVD and IHD, which would take advantage of the high CVD and IHD death rates observed in South Africa, to yield results which might be used to supplement existing knowledge of the "water story" phenomenon.

(ii) To establish a limited data base for the chemical quality of potable water in South Africa which would not only facilitate the study but which would also provide a base for future epidemiological research.

(iii) To investigate contemporary trends in cardiovascular disease mortality in South Africa as would be necessary to enable the study.

1.3 METHOD OF INVESTIGATION

A retrospective and current literature review was carried out with the aid of Waterlit.

The review showed that most existing studies are based on one of two models, ie. :

(i) The temporal model, which involves the study of changes in cardiovascular death rates of a cohort or of a fixed-age class with changing membership, occupying a specific area in which the water supply has undergone a dramatic and sudden change in hardness (either an increase or decrease) at some known point in time.

(ii) The spatial model, which involves the study of regional or urban differences in water hardness related to differences in cardiovascular death rates over the same area.

Whilst the former model is less effected by confounding variables of a geographic and socio-economic nature than the latter, it was not suited to the present study for the following reasons:

(i) The smallest well-urbanized areas suitable for study by virtue of the accuracy of their demographic data were found to be magisterial districts, the urban component

of which was often supplied with water from a number of authorities or sources. Thus any marked change in the water quality from one supply source or authority could not be statistically related to change in CVD mortality rate for the whole magisterial district.

(ii) Cohort studies are difficult to carry out in South Africa because of population movement to new urban areas and the lack of demographic data for urban areas prior to 1980.

In 1983 a pilot study was thus initiated using the spatial model and a more detailed study began in 1984. This study ultimately involved the correlation of standardized mortality ratios (SMRs) for CVD and IHD with total water hardness and with a number of contributory and associated water quality factors.

The following two correlation methods were used, each with intrinsic advantages and disadvantages, as outlined:

(i) Correlation with statistical weighting for population differences between study districts, which effectively compensated for the wide population range between the districts, but which simultaneously caused geographical clusters of districts to attain prominence, thus

weighting the effects of potentially confounding variables associated with each geographical region.

(ii) Correlation without population weighting, which removed the effects of clustering and rendered the results more comparable with the majority of correlation studies from other countries. This method, however, did not make allowance for the wide local population range between magisterial districts referred to above.

The results of the two correlation methods were then related to scatter plots and demographic base maps which enabled a set of conclusions to be drawn.

2. THE DATA

2.1 SELECTION OF STUDY AREAS

In South Africa, water quality data can be obtained for most cities and larger towns, but the smallest areas for which demographic data are available are "magisterial districts" as defined in the 1980 Census (20). These districts may consist of only part of a large city or may consist of a number of discrete towns surrounded by a non-urban area.

Accurate water data are not available for non-urban areas where water may be obtained from small impoundments, wells, springs or rain-butts.

Districts with a large non-urban component were eliminated as far as possible from the study by setting a minimum population requirement of 10 000 whites for each study area. After selection, mortality and demographic data for the remaining small non-urban population in each selected district was removed by subtraction.

In this way 56 "urban" magisterial districts listed with their magisterial district codes as shown in Table 1 were isolated as potential study areas. For the sake of uniformity, all codes used are for 1980, as eight of the 56 area codes changed between 1978 and that year, although changes did not occur between 1980 and 1982.

TABLE 1: NAMES AND 1980 CENSUS CODE NUMBERS OF 56 URBAN MAGISTERIAL DISTRICTS ELIGIBLE FOR INCLUSION IN THE STUDY BY VIRTUE OF POPULATION NUMBER

PROVINCE	NAME OF MAGISTERIAL DISTRICT	CODE
CAPE	Cape Town	0101
	Wynberg	0102
	Simonstown	0103
	Goodwood	0104
	Bellville	0105
	Stellenbosch	0201
	Kuils River	0202
	Somerset West	0203
	Strand	0204
	Paarl	0205
	Oudtshoorn	0501
	George	0602
	Worcester	0801
	Port Elizabeth	1401
	Uitenhage	1402
	Kimberley	2201
	East London	2801
	Queenstown	2907
NATAL	Durban	3501
	Pinetown	3502
	Pietermaritzburg	3801
	Port Shepstone	3901
	Newcastle	4301
	Kliprivier	4306
	Vryheid	4403
	Lower Umfolozi	4502
TRANSVAAL	Pretoria	4601
	Wonderboom	4602
	Johannesburg	4701
	Germiston	4801
	Alberton	4802
	Boksburg	4803
	Kempton Park	4804
	Benoni	4805
	Brakpan	4901
	Springs	4902
	Nigel	4903
	Highveld Ridge	4907
	Krugersdorp	5001
	Westonaria	5003
	Oberholzer	5005
	Vereeniging	5201
	Vanderbijlpark	5202
	(Sasolburg, OFS)	5301
	Pietersburg	5601
	Nelspruit	5701
	Witbank	5801
	Middelburg	5802
	Bethal	5901
	Klerksdorp	6001
	Potchefstroom	6102
ORANGE FREE STATE	Kroonstad	6204
	Bethlehem	6301
	Welkom	6402
	Virginia	6403
	Bloemfontein	6501

The selected districts can be spatially related by means of the outline map of South Africa (Figure 1), although it should be noted that the magisterial district boundaries indicated include both urban and non-urban components.

Twelve of these magisterial districts had to be removed from the study and one composite area added, for the following reasons :

(i) Reliable and comprehensive water data could not be obtained for Vryheid, Potchefstroom, Bethlehem and Kimberley.

(ii) The presence of a data coding error has been hypothesised for the Cape region, which includes the magisterial districts of Cape Town, Wynberg, Simonstown, Goodwood and Bellville (21). The contiguous districts of Cape Town and Wynberg are of particular concern; in the present study their SMRs for white urban CVD were found to be 37% higher and 37% lower respectively than the SMR for the overall Cape region. The five individual magisterial districts were thus removed from the study, and pooled data for the Cape region were included.

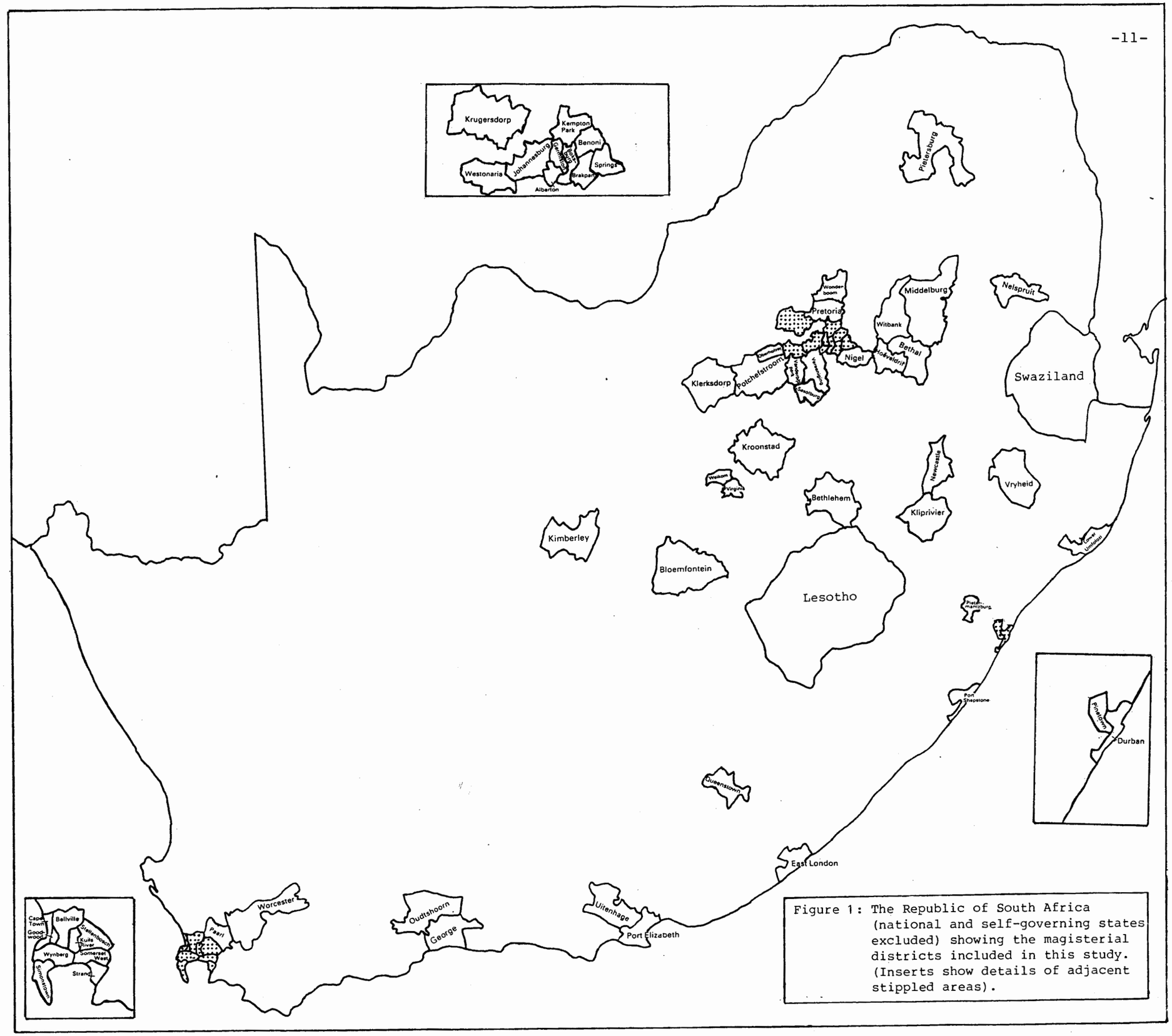

Figure 1: The Republic of South Africa (national and self-governing states excluded) showing the magisterial districts included in this study. (Inserts show details of adjacent stippled areas).

(iii) No CVD and IHD related deaths were reported for the Wonderboom district and extremely few for Highveld Ridge.

It is possible that mortality data for Wonderboom is being miscoded to the contiguous Pretoria area, although this could not be proved. In view of the fact that the white urban population of Pretoria was seven times that of Wonderboom, which would greatly reduce the impact of the hypothesised coding error on mortality rates produced for Pretoria, it was decided that Pretoria data would be included in the study without modification, whilst the Wonderboom district was excluded from the study.

Highveld Ridge, on the other hand, is close to Bethal for which an excessively high mortality rate was calculated. The hypothesis that miscoding between these two areas had occurred could not, however, be supported by reference to data sources or existing literature and both areas were thus eliminated from the study.

(iv) Excessively high mortalities for CVD and IHD were noted for Queenstown and Middelburg, although these could not be related to under-recording for contiguous areas because they had not been included in the study owing to insufficient population numbers for the production

of meaningful rates. The two districts were thus eliminated from the study.

This gave 42 urban magisterial districts and one urban region (Cape) for which meaningful data were available for statistical analysis.

2.2 MORTALITY DATA FOR CVD AND IHD

"Cardiovascular disease" was taken to imply diseases of the circulatory system as listed in Section VII of the South African Statistical Classification of Diseases, Injuries and Causes of Death (22), but excluding rheumatic heart disease (Table 2). Ischaemic heart disease is included as rubric 410-414 within the tabulation.

Mortality data for these diseases were obtained for 1978 to 1982 from the Department of Central Statistical Services' computerised records of unpublished mortality data.

The data extracted were related to place of usual residence as opposed to place of death and were further broken down by race, sex and five 10-year age categories between 25 and 74 years.

Table 2: DISEASES OF THE CIRCULATORY SYSTEM (WITH CODES) INCLUDED AS "CARDIOVASCULAR DISEASE" IN THIS STUDY

Hypertensive disease (401-405)

401	Essential hypertension
402	Hypertensive heart disease
403	Hypertensive renal disease
404	Hypertensive heart and renal disease
405	Secondary hypertension

Ischaemic heart disease (410-414)

410	Acute myocardial infarction
411	Other acute and subacute form of ischaemic heart disease
412	Old myocardial infarction
413	Angina pectoris
414	Other forms of chronic ischaemic heart disease

Diseases of pulmonary circulation (415-417)

415	Acute pulmonary heart disease
416	Chronic pulmonary heart disease
417	Other diseases of pulmonary circulation

Other forms of heart disease (420-429)

420	Acute pericarditis
421	Acute and subacute endocarditis
422	Acute myocarditis
423	Other diseases of pericardium
424	Other diseases of endocardium
425	Cardiomyopathy
426	Conduction disorders
427	Cardiac dysrhythmias
428	Heart failure
429	Ill-defined descriptions and complications of heart disease

Cerebrovascular disease (430-438)

430	Subarachnoid haemorrhage
431	Intracerebral haemorrhage
432	Other and unspecified intracranial haemorrhage
433	Occlusion and stenosis of precerebral arteries
434	Occlusion of cerebral arteries
435	Transient cerebral ischaemia
436	Acute but ill-defined cerebrovascular disease
437	Other and ill-defined cerebrovascular disease
438	Late effects of cerebrovascular disease

Diseases of arteries, arterioles and capillaries (440-448)

440	Atherosclerosis
441	Aortic aneurysm
442	Other aneurysm
443	Other peripheral vascular disease
444	Arterial embolism and thrombosis
445	Gangrene
446	Polyarteritis nodosa and allied conditions
447	Other disorders of arteries and arterioles
448	Diseases of capillaries

Diseases of veins and lymphatics, and other diseases of circulatory system (451-459)

451	Phlebitis and thrombophlebitis
452	Portal vein thrombosis
453	Other venous embolism and thrombosis
454	Varicose veins of lower extremities
455	Haemorrhoids
456	Varicose veins of other sites
457	Non-infective disorders of lymphatic channels
458	Hypotension
459	Other disorders of circulatory system

2.3 CHEMICAL QUALITY DATA FOR POTABLE WATER

South Africa does not possess a comprehensive data bank for the chemical quality of potable water and thus the establishment of a limited data base for the purposes of this study was necessary.

The relevant data were obtained for the years 1978 to 1982 from the Rand Water Board, which supplied 19 of the selected urban magisterial districts, from the Orange Free State Goldfields Water Board, the Umgeni Water Board, the Lower South Coast Regional Water Services Corporation, the Hydrological Research Institute of the Department of Environment Affairs and from 22 local authorities supplying local areas.

The local authorities of Potchefstroom, Vryheid, Bethlehem and Kimberley were unable to supply adequate data and were removed from the study.

The effect of potential changes in potable water quality with passage through the water reticulation system were minimized wherever possible by requesting data for water sampled at representative standpipes.

Correction for variations in individual water usage patterns in the home are known to be desirable but would have

involved an extensive National survey beyond the constraints of the present study (23).

In view of the long latency period of about 20 years associated with fatal atheromatous degeneration (24), the use of contemporaneous water data in this and previous studies could be criticised, but until the biochemical nature of the water-related mortality phenomenon has been described, the assumption of a specific latency period would be dangerous. In addition, a number of rapidly growing urban districts have poor retrospective records of water quality.

During the establishment of the limited data base the following problems were identified and reported on at the first South African Symposium on Water Quality Assessment: (25)

(i) The incompatability of certain data kept by water boards, research institutes and local authorities, owing to variations in sampling period, factors analysed and analytical technique.

(ii) The inaccuracy or inavailability of data produced by some authorities because of the lack of trained sampling or analytical staff, or the existence of administrative shortcomings.

(iii) The lack of a National data bank for potable water on the lines of the raw water data bank at present kept by the Hydrological Research Institute, Pretoria, and the Computerized Centre for Water Research, Pietermaritzburg.

Recommendations for rectification of these shortcomings were made as follows:

(i) That a National data bank for potable water be established on the lines of the data bank for raw water at present operated by the Hydrological Research Institute.

(ii) That suitable guidelines or standards be produced relating to the frequency and method of collection of samples, the method of analysis, the submission of data to the central repository and the subsequent coding, storage, representation and availability of data.

At this stage attention must be focused on a problem which haunts "water story" studies in general, viz: the existence of a number of definitions for "water hardness" and thus the existence of a number of acceptable measuring techniques, each of which will yield a slightly different result.

The definition of hardness has changed with time, originally signifying the ability of water to waste soap by forming scum, or its ability to form scale on boiling. Today chemists tend to refer to "total hardness", which is the sum of the temporary (carbonate) hardness and the permanent (non-carbonate) hardness, expressed as "equivalent calcium carbonate" in mg/l of water (26).

Temporary hardness can be removed by boiling, which drives off dissolved carbon dioxide, eliminating the carbonic acid in which the carbonates were dissolved and resulting in their precipitation.

Total hardness can be assessed by means of spectrophotometric analysis followed by the necessary stoichiometry to give highly accurate results. Inclusion of salts other than those of calcium and magnesium is, however, arbitrary and can influence the final result of such assessment.

A simpler method for smaller sample numbers involves the titration of the water sample against a standard solution of the disodium salt of ethylenediaminetetraacetic acid (EDTA). This method, however, lacks the accuracy of spectrophotometric analysis.

Because of differences in definition and analytical technique, standardized methodology for the analysis of water requires the reporting of analytical method used alongside the results obtained (26), but this was found to have been omitted in all the South African data sets for total hardness.

The problem was controlled in the present study by including data not only for total hardness but also for 16 chemical sub-factors, six of which are known to contribute directly to the phenomenon of hardness, and 10 of which are indirectly associated with hard water (27). The six "contributory factors" were further divided to isolate two "major contributory factors" (calcium and magnesium) and four "minor contributory factors" (see Table 3).

All contributory and associated factors were inter-correlated, using Spearman's Rank Correlation (r_s) (for non-parametric data) and the relevant two-tailed test for statistical significance applied, in order to assess the relationships between total hardness and the factors contributing to and associated with that phenomenon. The results of this data testing are shown in Table 4.

TABLE 3 : WATER QUALITY FACTORS INCLUDED IN THE STUDY CLASSIFIED BY THEIR USUAL RELATIONSHIP TO HARDNESS

A. FACTOR SUMMATING ALL CARBONATE AND NON-CARBONATE HARDNESS:

Total hardness (as $CaCO_3$)

B. MAJOR FACTORS CONTRIBUTING DIRECTLY TO THE PHENOMENON OF HARDNESS:

Calcium
Magnesium

C. MINOR FACTORS CONTRIBUTING DIRECTLY TO THE PHENOMENON OF HARDNESS:

Iron
Aluminium
Silicon dioxide

D. FACTORS INDIRECTLY ASSOCIATED WITH HARDNESS:

Sodium
Potassium
Manganese
Sulphate
Fluoride
Chloride
Conductivity
pH
pHs
Langelier Saturation Index (LSI)
Alkalinity

TABLE 4:SPEARMAN'S RANK CORRELATION COEFFICIENTS (r_S) FOR WATER HARDNESS AND RELATED FACTORS FOR 55 URBAN MAGISTERIAL DISTRICTS

	Tot hardness	Calcium	Magnesium	Iron	Aluminium	Silicon dioxide	Sodium	Potassium	Manganese	Sulphates	Fluorides	Chlorides	Conductivity	pH	pHs	LSI	Alkalinity
Tot hardness	+1,00xx																
Calcium	+0,94xx	+1,00xx															
Magnesium	+0,84xx	+0,82xx	+1,00xx														
Iron	-0,65xx	-0,65xx	-0,63xx	+1,00xx													
Aluminium	-0,70xx	-0,64xx	-0,73xx	+0,62xx	+1,00xx												
Silicon dioxide	+0,26NS	+0,11NS	+0,26NS	-0,60xx	-0,25NS	+1,00xx											
Sodium	+0,82xx	+0,76xx	+0,78xx	-0,53xx	-0,68xx	+0,02NS	+1,00xx										
Potassium	+0,92xx	+0,83xx	+0,85xx	-0,76xx	-0,77xx	+0,42x	+0,75xx	+1,00xx									
Manganese	-0,64xx	-0,61xx	-0,72xx	+0,91xx	+0,66xx	-0,79xx	-0,61xx	-0,80xx	+1,00xx								
Sulphates	+0,94xx	+0,85xx	+0,82xx	-0,56xx	-0,68xx	+0,11NS	+0,90xx	+0,87xx	-0,69xx	+1,00xx							
Fluorides	+0,85xx	+0,76xx	+0,78xx	-0,71xx	-0,53xx	+0,27NS	+0,78xx	+0,74xx	-0,78xx	+0,81xx	+1,00xx						
Chlorides	+0,63xx	+0,50xx	+0,55xx	-0,41xx	-0,74xx	+0,05NS	+0,85xx	+0,61xx	-0,58xx	+0,78xx	+0,70xx	+1,00xx					
Conductivity	+0,76xx	+0,66xx	+0,56xx	-0,38xx	-0,68xx	+0,24NS	+0,70xx	+0,76xx	-0,61xx	+0,73xx	+0,73xx	+0,75xx	+1,00xx				
pH	-0,15NS	-0,15NS	-0,30x	+0,04NS	+0,41x	-0,22NS	-0,41xx	-0,33x	+0,29o	-0,40xx	-0,45xx	-0,18NS	-0,27o	+1,00xx			
pHs	-0,82xx	-0,64xx	-0,65xx	+0,25NS	+0,39o	-0,34NS	-0,53xx	-0,79xx	+0,16NS	-0,77xx	-0,47xx	-0,56xx	-0,79xx	-0,22NS	+1,00xx		
LSI	+0,54xx	+0,48xx	+0,27NS	-0,45x	-0,11NS	+0,23NS	+0,28NS	+0,42x	-0,33NS	+0,32o	+0,55xx	+0,20NS	+0,37x	+0,86xx	-0,56xx	+1,00xx	
Alkalinity	+0,84xx	+0,85xx	+0,79xx	-0,53xx	-0,72xx	+0,36x	+0,61xx	+0,74xx	-0,56xx	+0,73xx	+0,72xx	+0,39xx	+0,68xx	-0,18NS	-0,78xx	+0,49xx	+1,00xx

Note:
Correlation coefficient is in each case followed by a code indicating the level of significance (two tail test), viz:

xx indicates significant at α=0,01
x indicates significant at α=0,05
o indicates significant at α=0,10
NS indicates not significant at α=0,10

where α is the level of statistical significance

With the exception of silicon dioxide and pH, all sub-factors show highly significant correlation with total hardness ($p < 0,01$ by two-tailed test).

After statistical weighting of data as discussed in Chapter 3 had been carried out, the data were distributed by magisterial district as shown in Figures 8 to 24, Appendix A. Bimodality is apparent for several factors, including hardness, calcium and magnesium (Figures 8 to 10, Appendix A).

This bimodality can be related to the demographic base map of water hardness by urban magisterial district (Figure 7 as observed with Figures 1 and 4) which shows that the main coastal towns receive very soft water which is obtained from upland surface reservoirs in adjacent mountain ranges, but that the large Pretoria-Witwatersrand-Vereeniging (PWV) grouping of districts in the interior of the sub-continent is typified by moderately hard water, obtained mostly from riverine impoundments or large dams where the water, which may have flowed over long distances through mineralized areas, is often held for considerable periods of time to compensate for "dry years". During this storage, much evaporation of water and thus concentration of salts occurs, to produce relatively hard water.

(Comments regarding the use and interpretation of the demographic base maps referred to in this section are included in section 3.3.4.)

2.4 DEMOGRAPHIC DATA

The data were obtained from the 1980 census and the urban population obtained for each magisterial district by subtraction of the non-urban component.

Only data for White males and females in the 25 to 74 age category were included; Blacks being excluded from the study as demographic data for this group is known to be inaccurate (28).

Coloured and Asian groups were also excluded as ischaemic heart disease rates for these groups are known to be substantially different from those for Whites (29).

Comments regarding inadequacies in the demographic data, which necessitated the removal of a number of magisterial districts from the study and the addition of one composite area, are included in section 2.1 ("Selection of Study Areas").

3. STATISTICAL APPROACH

3.1 STANDARDIZATION OF MORTALITY RATES

Whilst the use of specific rates in epidemiological analysis is desirable (30), indirect standardization to yield standardized mortality ratios (SMRs) for CVD and IHD was used in this study for the following reasons:

(i) The inclusion of deaths for both sexes in a wide age range (25 to 74 years) resulted in some of the age/sex strata being composed of relatively small numbers which would have not lent themselves to the production of comparable specific rates.

(ii) The production of a single summary index for each population facilitated ease of comparison between the relatively large number of populations involved in the study. In addition, comparison of entire schedules of specific rates would have been difficult bearing in mind the number of populations involved.

To obtain SMRs, death rates were calculated for each sex and 10-year age category of the standard population, which was taken to be the total South African urban White population.

These rates were related to the population structure of each of the 43 urban magisterial districts to produce expected numbers of deaths.

SMRs were then calculated as the ratio of the observed deaths to the expected deaths for each district, expressed as a percentage.

The resultant SMRs for CVD and IHD are listed by urban magisterial district in Table 5 and are also shown distributed by the 43 urban magisterial districts in Figures 2 and 3.

A point of interest is that the majority of SMRs for both CVD and IHD are well above 100. A possible explanation is that the study districts are mostly large urban centres whereas the standard population includes all urbanized areas, from large centres to very minor settlements. A tendency to code place of residence at time of death as being the largest sizeable centre proximate to the actual place of death might well exist, and this would have accounted for the production of inflated SMRs during the standardization process.

TABLE 5: STANDARDIZED MORTALITY RATIOS (SMRs) FOR CVD AND IHD BY URBAN MAGISTERIAL DISTRICT

PROVINCE	URBAN MAGISTERIAL DISTRICT	SMR FOR CVD	SMR FOR IHD
CAPE	Cape (grouped*)	155	142
	Stellenbosch	168	143
	Kuils River	142	129
	Somerset West	180	153
	Strand	194	175
	Paarl	205	188
	Oudtshoorn	245	216
	George	197	169
	Worcester	232	193
	Port Elizabeth	162	136
	Uitenhage	206	182
	East London	194	170
NATAL	Durban	158	135
	Pinetown	117	89
	Pietermaritzburg	192	157
	Port Shepstone	151	113
	Newcastle	184	165
	Kliprivier	178	154
	Lower Umfolozi	134	142
TRANSVAAL	Pretoria	161	136
	Johannesburg	154	131
	Germiston	138	121
	Alberton	127	103
	Boksburg	167	151
	Kempton Park	117	103
	Benoni	148	125
	Brakpan	176	144
	Springs	202	160
	Nigel	244	205
	Krugersdorp	244	224
	Westonaria	124	100
	Oberholzer	176	181
	Vereeniging	158	150
	Vanderbijlpark	169	158
	(Sasolburg, OFS)	149	145
	Pietersburg	233	221
	Nelspruit	245	246
	Witbank	248	243
	Klerksdorp	185	177
ORANGE FREE STATE	Kroonstad	209	199
	Welkom	135	114
	Virginia	163	173
	Bloemfontein	165	147

* Includes the urban magisterial districts of Cape Town, Wynberg, Simonstown, Goodwood and Bellville.

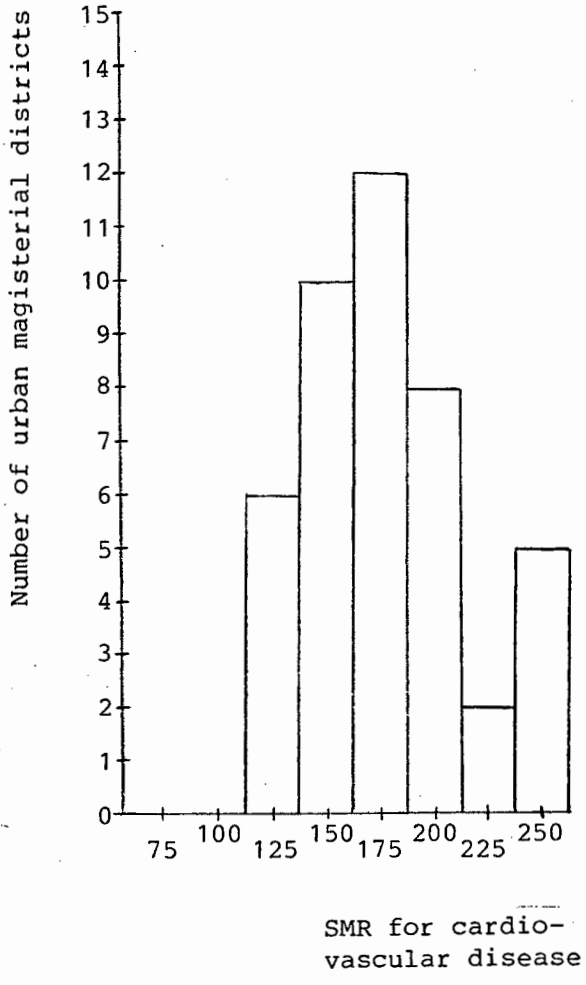

Figure 2: Distribution of SMRs for cardiovascular disease by urban magisterial district

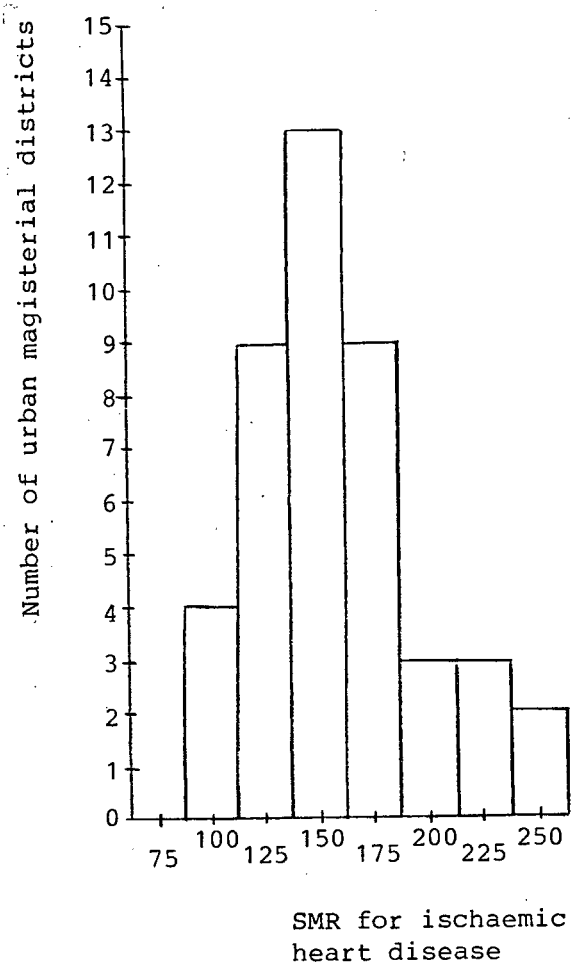

Figure 3 : Distribution of SMRs for ischaemic heart disease by urban magisterial district

3.2 WEIGHTING OF WATER QUALITY DATA

In many of the larger urban magisterial districts a number of sources or treatment works existed, each supplying water with a different set of chemical characteristics.

Where such waters were blended in a common reticulation system and results for standpipe samples were not available, it was necessary to produce data for an "average" water as received by the district as a whole, by weighting the individual quality factors on the basis of the volume of water from each supply source.

Sometimes doubt existed as to the exact volume of water from each source, which might change with the time of day or season of the year and in such cases "guesstimates" made by distribution engineers had to be relied on.

Where water was not blended but supplied from different treatment works to different sectors within each district, a population-weighted average for each water factor was calculated based on the number of residents receiving each water type within the overall magisterial district. In this way, a chemical quality profile for the water consumed by the "average resident" was obtained.

Detailed results for the weighted water data are not tabulated in this study but will in future form part of

Hydrolit, a national computerized data base, which is reachable through the South African Water Information Centre, Pretoria or through the Computing Centre for Water Research, Pietermaritzburg.

3.3 CORRELATION OF SMRs FOR CVD AND IHD WITH WATER QUALITY

3.3.1 TEST FOR CORRELATION, WITH POPULATION WEIGHTING

Because the total study population of each magisterial district was different, statistical weighting based on population number was applied within the correlation process.

The test used was Spearman's Rank Correlation (r_s), applicable to non-parametric data, with subsequent two-tailed testing for significance. Results are shown in Table 6.

Computations were carried out using the Biomedical Data Package (BMDP)(31), on a Sperry 1100 Series computer.

3.3.2 TEST FOR CORRELATION, WITHOUT POPULATION WEIGHTING

In order to facilitate comparison with studies where population weighting was not used, correlation of unweighted data was also carried out. Results are shown in Table 7.

TABLE 6: SPEARMAN'S RANK CORRELATION COEFFICIENTS (r_S) FOR 50 URBAN MAGISTERIAL DISTRICTS (WEIGHTED FOR POPULATION)

	SMR for CVD	SMR for IHD
SMR for CVD	+1,00xx	
SMR for IHD	+0,84xx	+1,00xx
Tot. hardness	-0,23xx	-0,21xx
Calcium	-0,34xx	-0,32xx
Magnesium	-0,24xx	-0,27xx
Iron	+0,21xx	+0,24xx
Aluminium	+0,25xx	+0,08NS
Silicon dioxide	+0,08NS	+0,08NS
Sodium	-0,21xx	-0,31xx
Potassium	-0,34xx	-0,41xx
Manganese	+0,17x	+0,41xx
Sulphates	-0,32xx	-0,36xx
Fluorides	-0,06NS	-0,26xx
Chlorides	-0,10o	-0,13x
Conductivity	-0,09NS	-0,06NS
pH	-0,17xx	-0,30xx
pHs	+0,17x	+0,04NS
LSI	+0,11o	+0,21xx
Alkalinity	-0,14x	-0,11o

Note:
Correlation coefficient is in each case followed by a code indicating the level of significance (two-tail test), viz:

xx indicates significant at α=0,01
x indicates significant at α=0,05
o indicates significant at α=0,10
NS indicates not significant at α=0,10

where α is the level of statistical significance

TABLE 7: SPEARMAN'S RANK CORRELATION COEFFICIENTS (r_S) FOR 50 URBAN MAGISTERIAL DISTRICTS (UNWEIGHTED FOR POPULATION)

	SMR for CVD	SMR for IHD
SMR for CVD	+1,00xx	
SMR for IHD	0,95xx	+1,00xx
Tot hardness	-0,26o	-0,13NS
Calcium	-0,24NS	-0,11NS
Magnesium	-0,24NS	-0,09NS
Iron	+0,20NS	+0,19NS
Aluminium	0,00NS	0,00NS
Silicon dioxide	-0,02NS	-0,02NS
Sodium	-0,16NS	-0,11NS
Potassium	-0,35x	-0,30o
Manganese	+0,31o	+0,32o
Sulphates	-0,32x	-0,21NS
Fluorides	-0,18NS	-0,07NS
Chlorides	-0,29o	-0,24NS
Conductivity	-0,22NS	-0,13NS
pH	-0,04NS	-0,03NS
pHs	+0,36x	+0,23NS
LSI	-0,19NS	-0,09NS
Alkalinity	-0,18NS	-0,01NS

Note:
Correlation coefficient is in each case followed by a code indicating the level of significance (two-tail test), viz:

xx indicates significant at α=0,01
x indicates significant at α=0,05
o indicates significant at α=0,10
NS indicates not significant at α=0,10

where α is the level of statistical significance

The desirability of population weighting in this study, because of the wide population range between South African urban magisterial districts, should however be kept in mind.

3.3.3 SCATTER PLOTS, WITH POPULATION WEIGHTING

These are shown in Appendix B, figures 27 to 60. Each plot is represented by a number equivalent to the weighting of the plot at that location, or the sum of weightings where two or more plots occupy the same location on the scattergram. Numbers not underlined consist of units only, whereas underlining of two numbers indicates tens and units. Thus "33" would indicate two proximate plots each with a weighting of 3, whereas "33" indicates a single plot (or the sum of superimposed plots) with a weighting of 33. In the latter case, the plot point on the graph appears midway between the two underlined digits.

3.3.4 DEMOGRAPHIC BASE MAPPING

Standard mapping techniques are often used in epidemiology to indicate, by means of suitably delineated and shaded areas, the pattern of mortality over a defined geographical region.

Whilst such techniques may effectively indicate the shape and extent of each area, they fail to indicate the population density and thus the size of the population at

risk in each area. Thus large but sparsely populated areas take prominence whilst small but densely populated areas are barely noticeable.

To overcome the problem, use was made in this study of demographic base mapping as described by Forster (32), which has the advantages of being able to represent the size of the populations at risk, the rates relevant to each population, and the approximate spatial relationships between populations, including the clustering of populations.

Demographic base maps are population-weighted devices whereas geographical regional maps are not.

The relevant maps in this study are best interpreted by first relating Figure 1, which is a geographical map of eligible magisterial districts, to Figure 4, which is a demographic base map of the selected urban magisterial districts showing district names and code numbers.

In the latter map, the key relates the size of plotted squares to the urban white population (male and female between the ages of 25 to 74 years) within each urban magisterial district.

Figure 4 then relates to Figures 5, 6 and 7, which map SMR for CVD, SMR for IHD and total hardness as CaCO3, on the same demographic base.

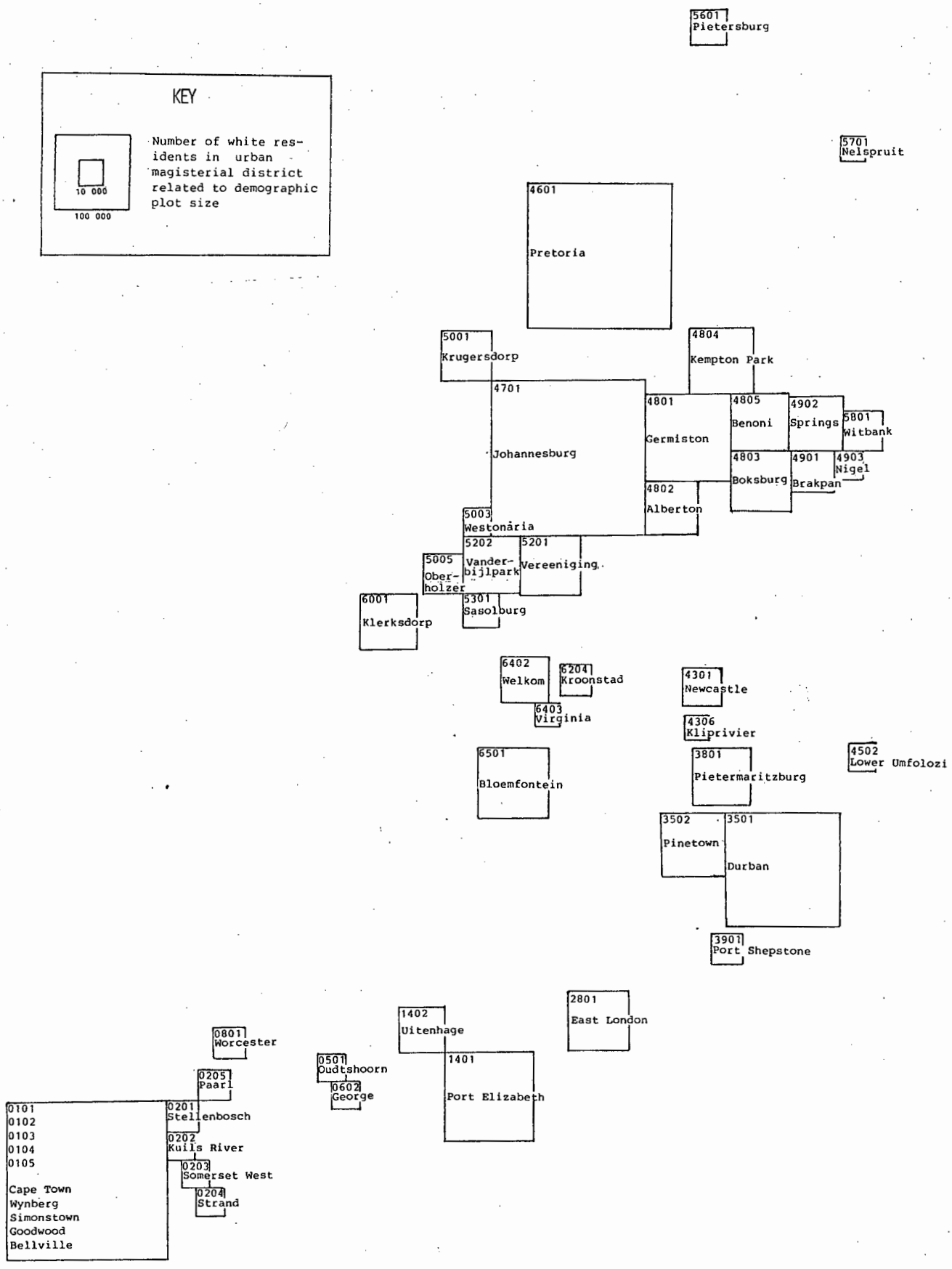

FIGURE 4: DEMOGRAPHIC BASE MAP OF URBAN MAGISTERIAL DISTRICTS SHOWING NAMES AND CODE NUMBERS

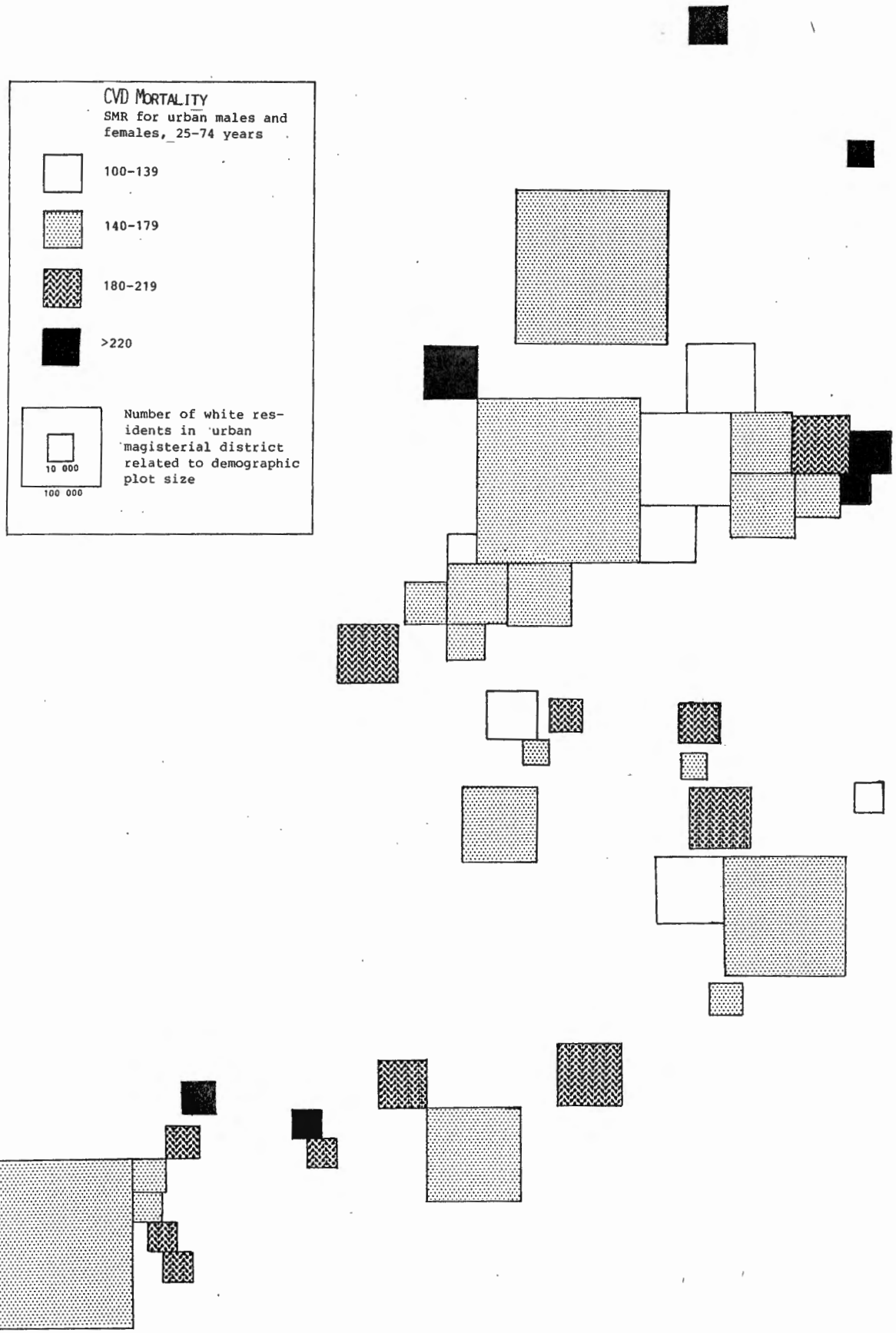

FIGURE 5: DEMOGRAPHIC BASE MAP SHOWING SMR FOR CVD BY URBAN MAGISTERIAL DISTRICT
(NAMES AND CODE NUMBERS OF URBAN MAGISTERIAL DISTRICTS ARE SHOWN IN FIGURE 4)

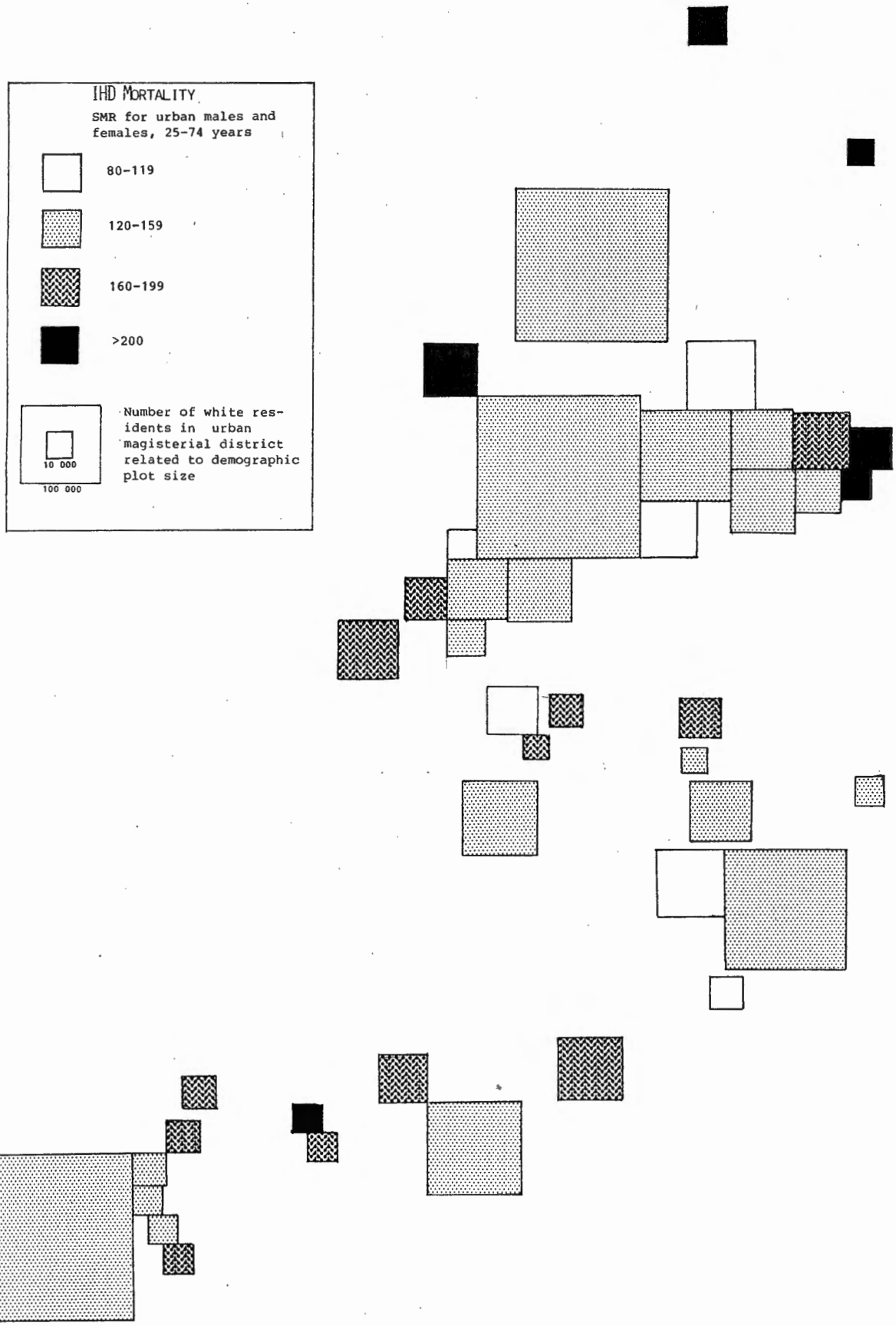

FIGURE 6: DEMOGRAPHIC BASE MAP SHOWING SMR FOR IHD BY URBAN MAGISTERIAL DISTRICT
(NAMES AND CODE NUMBERS OF URBAN MAGISTERIAL DISTRICTS ARE SHOWN IN FIGURE 4)

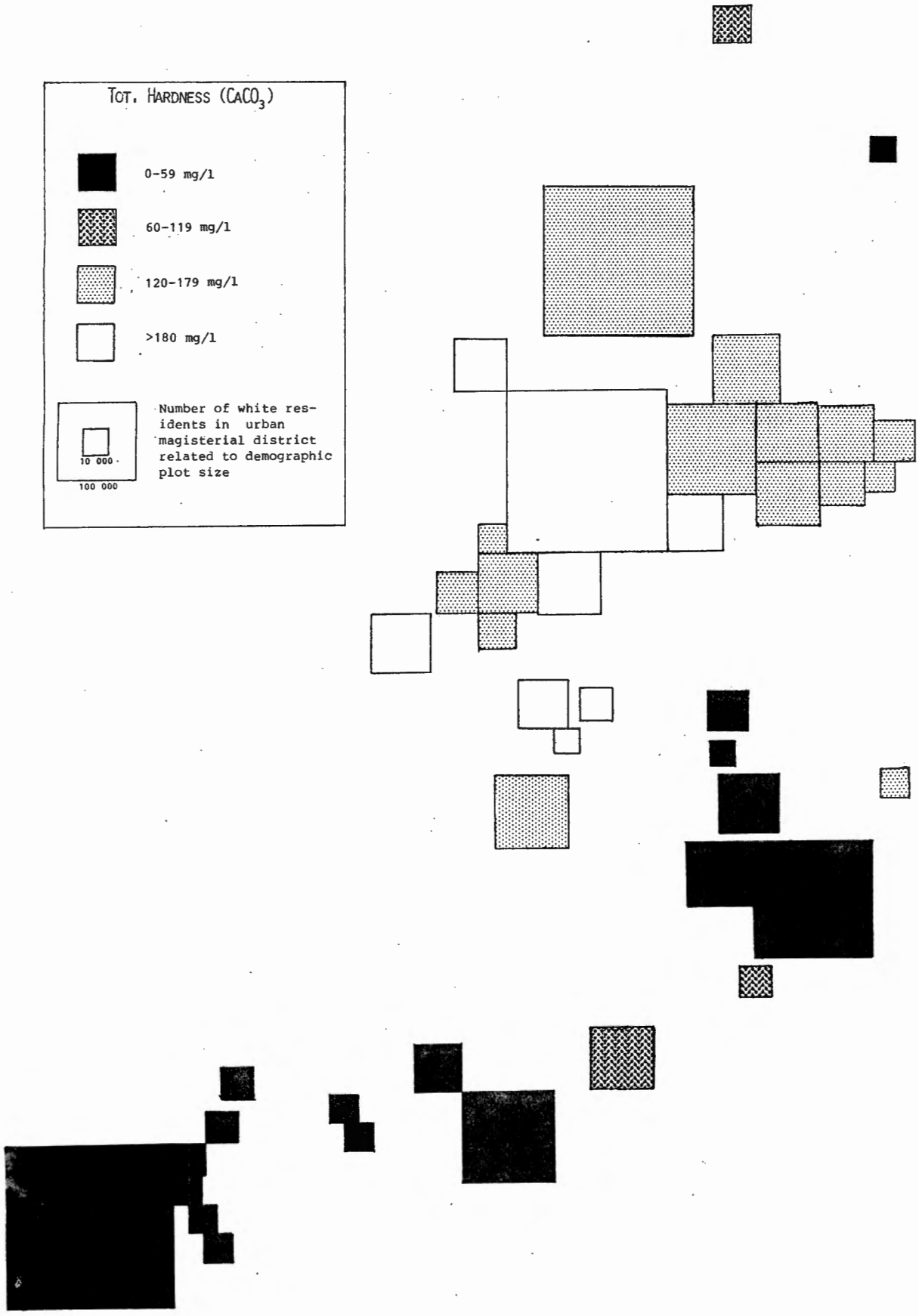

FIGURE 7: DEMOGRAPHIC BASE MAP SHOWING TOTAL WATER HARDNESS BY URBAN MAGISTERIAL DISTRICT (NAMES AND CODE NUMBERS OF URBAN MAGISTERIAL DISTRICTS ARE SHOWN IN FIGURE 4)

4. RESULTS

The need for population weighting in the correlation process has been discussed in section 3.3. For purposes of comparison with a number of existing studies relating to other countries; however, results have also been presented based on population-unweighted correlation.

4.1 TEST FOR CORRELATION, WITH POPULATION WEIGHTING (Table 6)

Both SMRs for CVD and IHD yielded highly significant negative correlations with total hardness, and with the major contributory factors, calcium and magnesium ($p < 0,01$; two-tailed test).

Highly significant positive correlations between CVD mortality and the minor contributory factors iron and aluminium and between IHD mortality and iron, related to the strongly negative correlations between these factors and hardness (see Table 4), a phenomenon which at this stage cannot be explained owing to the lack of research regarding potable water in South Africa.

Highly significant negative correlations were observed between CVD and IHD mortality and a number of factors indirectly associated with total hardness, ie. sodium, potassium, sulphates and pH. IHD mortality also showed correlation with fluoride at the same level of significance.

Significant negative correlations ($p < 0,05$; two-tailed test) were observed between CVD mortality and alkalinity, and IHD mortality and chlorides.

Highly significant positive correlations were observed between IHD mortality and indirect hardness-associated factors manganese and the Langelier saturation index (LSI), and significant positive correlations were noted between CVD mortality and manganese and pHs.

4.2 TEST FOR CORRELATION, WITHOUT POPULATION WEIGHTING (Table 7)

With the population weighting removed, negative correlations between both CVD and IHD mortality, and hardness and its major contributory factors, calcium and magnesium, were still obtained but not at the assumed level for statistical significance ($p < 0,05$; two-tailed test), although the correlation between CVD and hardness still showed slight statistical significance ($p < 0,10$).

For all factors, the only correlations found to be significant at the assumed level were those between CVD mortality and potassium and sulphates (negative correlations) and CVD mortality and pHs (positive correlation).

4.3 SCATTER PLOTS (Appendix B, Figures 25 to 60)

When unweighted scatter plots were compared with weighted scatter plots of SMRs for CVD and IHD against total hardness (figures 25 to 28), the effect of weighting on the statistical significance of the negative correlation became apparent.

The most heavily weighted districts (underlined values) tended to cluster in two groups, the 44, 35, 12 group representing the populous and contiguous Johannesburg-Pretoria-Germiston region, with its relatively hard water and low mortality ratios for CVD and IHD; and the 48, 24, 15 group representing the three main South African coastal urban settlements of Cape Town (composite area), Durban and Port Elizabeth, all receiving very soft potable water and showing as a group a relatively higher mean SMR for both CVD and IHD.

The strong negative correlation which was established between these two groups, created as a result of the weighting process, was likely to have resulted in the stronger negative correlation coefficient for all weighted data compared to that for unweighted data for CVD and IHD against water hardness.

Whilst weighting in the correlation process was desirable for reasons discussed in Chapter 3, weighting can be

criticized in that it simultaneously produced geographically distinct clusters of urban districts, as discussed above, and this inevitably increased the role of potentially confounding variables associated with each of the clusters.

Confounding variables which would have to be corrected for in a more extensive South African study are discussed in Chapter 5.

Weighted plots of SMRs for CVD and IHD against all other contributory and indirectly associated water factors are shown in Appendix B, figures 27 to 60, and these should be read in conjunction with Tables 6 and 7 showing weighted and unweighted correlation indices.

4.4 DEMOGRAPHIC BASE MAPS (Figures 5 to 7)

The base map for total hardness (Figure 7) showed a distinct soft water coastal belt in comparison to the moderately hard water region in the Pretoria-Witwatersrand-Vereeniging (PWV) area. The term "moderately hard" is used here because most of the PWV area received water with a total hardness of between 150 - 200 mg/l equivalent calcium carbonate, whereas a few districts in the study received water with a hardness in excess of 300mg/l.

The map for CVD mortality (Figure 5), showed that SMRs in the PWV area (including the Johannesburg, Kempton Park,

Germiston and Alberton districts) were in a lower range (100 to 179) than those SMRs applicable to coastally contiguous districts (140 to 219).

A similar trend was apparent for IHD mortality (Figure 6) where the SMR ranges for the PWV area and the coastally contiguous districts were 80 to 159 and 120 to 199 respectively, the exception being Port Shepstone, which had an SMR of 113, which was just below the coastal range.

Districts just inland of the coastally contiguous districts, but generally considered to be towns within the coastal belt, had SMRs for CVD and IHD in even higher ranges (140 to 245 and 140 to 216 respectively), the exception being Pinetown with uncharacteristically low SMRs for CVD and IHD for the coastal region (117 and 89 respectively). The possibility of a coding error between Pinetown and contiguous Durban, however, could have given rise to the lower SMRs and warrants further investigation.

Within the Transvaal, SMRs for both CVD and IHD were seen to progressively increase with increase in distance from the PWV region, the highest rates being seen at both extremities of the East-West belt of contiguous Southern Transvaal urban magisterial districts, and in the distant Northern and Eastern Transvaal districts of Pietersburg and Nelspruit.

This interesting phenomenon was not related to water hardness and would warrant investigation in terms of other variables in future studies.

5. DISCUSSION

The study supports the hypothesis that negative statistical relationships exist between SMR for CVD and IHD, and the hardness of potable water, whether measured as total hardness or in terms of its two major contributory cations, calcium and magnesium.

These negative correlations are highly statistically significant ($p < 0.01$) for the abovementioned factors when population weighting is applied within the correlation process, but when weighting is removed the negative correlations are not significant at $p < 0,05$, although the correlation between CVD and hardness is slightly significant at $p < 0,10$.

5.1 POPULATION-WEIGHTED CORRELATION

Population weighting is desirable when a wide population range exists for correlated districts, although weighting applied in this study had a simultaneous effect of producing geographically distinct clusters of urban districts, which would inevitably emphasise the role of potentially confounding variables associated with each of these clusters.

In order to assess the influence of such confounding variables, extensive data collection would be required with

regard to a number of factors, including geographic factors, such as latitude, longitude and altitude; climatic factors such as rainfall, relative humidity and seasonal temperature range (a winter high and summer low for CVD having already been observed in South Africa (33)); socio-economic factors, such as the number of manual workers per district, qualification distribution, percentage employment of economically active males, social class score, cars per household, dwelling size, percentage owner-occupied households, number of children, type of transportation used, etc.; physiological and behavioural factors already shown to influence the cardiovascular health of South Africans, including the "major reversible risk factors" of hypercholesterolaemia, hypertension and smoking; the "minor reversible risk factors" of inactivity, obesity, hyperuricaemia and coronary-prone behaviour, and the "irreversible risk factors" of chest-pain history, ischaemic changes on the electrocardiogram and family history of IHD (34).

Where direct measurement proved impractical because of complexity or cost, a number of indirect measurements could be substituted; for example, data which is relatively simple to obtain regarding the ratio of English to Afrikaans speaking residents in a magisterial district could be used as an indirect indicator of type IIa familial hyperlipidaemia which has been shown to be particularly

prevalent in White, Afrikaans-speaking South Africans in comparison with other language groups, and which presents an important IHD risk factor (35).

An optional study model which would limit the total volume of data to be collected for confounding variables would involve the treatment of the main clusters of urban areas which resulted from population weighting (i.e.: the most populous areas) as sample areas, and then apply inferential statistical techniques to the analysis.

In this way, variation in population number would be controlled, and the quality of the data could be improved by studying only larger urban districts for which certain well-tested data sets with regard to a number of potentially confounding variables already existed.

5.2 CORRELATION, UNWEIGHTED FOR POPULATION

The discussion regarding the need to correct for confounding variables also applies to the unweighted study, although here the more random geographical scatter of the study plots would have the effect of reducing the relative influence of such variables.

In Table 7, which relates to unweighted data, statistically significant negative correlations ($p < 0,05$) are shown for SMR for CVD and both sulphates and potassium, whilst

significant positive correlation was obtained for SMR for CVD and pHs.

Whilst negative correlations were obtained between SMR for CVD and IHD, and hardness and its two major contributory factors, calcium and magnesium, these correlations were not statistically significant at $p < 0,05$, although the correlation between CVD and total hardness was slightly significant ($p < 0,10$).

The significant result for CVD and sulphates suggests that the elusive "water factor" may be a function of permanent hardness, i.e.: hardness not effected by the boiling of water.

With regard to the significant negative correlation between SMR for CVD and potassium, clinical and epidemiological studies in other countries have shown significant inverse relationships between potassium intake and hypertension which, as already discussed, is a major risk factor in CVD.

Clinical studies by Kaplan (36) have shown that in hypertensive patients, potassium supplements effectively lower blood pressure, in addition to improving diuretic-induced hypokalaemia, and in epidemiological studies carried out in Japan where the traditional diet is high in salt, one research group showed that blood pressure correlated

inversely with potassium excretion and directly with the urinary Na+/K+ ratio (37) whilst another group found inverse correlations between blood pressure and dietary potassium intake in Northern Japanese villages (38).

Studies of mixed groups of normotensive and hypertensive Americans have also revealed negative correlations between blood pressure and potassium excretion (39).

Although small quantities of potassium are found in water relative to total dietary needs (40), it is possible that water levels act as an indicator of regional mineralisation which can determine the levels found in crops and thus, to some extent, the levels found in the diet.

The significant positive relationship between SMR for CVD and pHs cannot be explained at this stage, save to note that very strong negative correlations were obtained between pH and both sulphates and potassium (Table 4), and thus the strong correlation observed between CVD and pHs may have been the result of statistical synergy.

6. CONCLUSIONS

The fact that different correlation methodologies yielded appreciably different results in this study gives some insight as to why conflicting results have been obtained in previous studies of this complex epidemiological phenomenon.

It is possible that no "correct" methodology exists but that the problem should rather be approached using a number of suitable correlation models, and that results should then be compared and interpreted, as has been attempted here.

Improvement of the quality of data, however, can only improve results and here emphasis must be placed on the need for a National data bank for potable water in South Africa.

The collection of direct or indirect data for a number of confounding variables is also required if a meaningful, multiple-regression study is to be executed.

Although such data collection was found to be outside the scope of the present study, it must be emphasised that such data, when banked, would serve as a valuable basis for future studies in environmental epidemiology.

Whilst the present study supported the "water story" hypothesis, hard and fast conclusions, on which policy

decisions regarding the treatment and supply of potable water might be based, should be deferred until the augmentation and refinement of data enables a more accurate and advanced study to be carried out.

REFERENCES

1. WYNDHAM, C. H. A Comparison of the Mortality Rates of White South Africans with those of the Population of England and Wales. South African Medical Journal (1980) 57, 729-741.

2. WYNDHAM, C. H. Ischaemic Heart Disease Mortality Rates in White South Africans compared with Other Populations. South African Medical Journal (1978) 53, 595-601.

3. CRAWFORD, M. D., GARDNER, M. J. and MORRIS, J. N. Mortality and Hardness of Local Water Supplies. Lancet (1968) 1, 827-831.

4. MORRIS, J. N., CRAWFORD, M. D. and HEADY, J. A. Hardness of Local Water Supplies and Mortality from Cardiovascular Disease. Lancet (1961) 1, 860-862.

5. LACEY, R. F. Changes in Water Hardness and Cardiovascular Death Rates. Technical Report TR 171, Water Research Centre, Medmenham (1981) p33.

6. ANDERSON, T. W., NERI, L. C. and SCHREIBER, G., et al. Ischaemic Heart Disease, Water Hardness and Myocardial Magnesium. Canadian Medical Association Journal (1975) 113, 199-203.

7. NERI, L. C., MANDEL, J. S., and HEWITT, D. Relation between Mortality and Water Hardness in Canada. Lancet (1972) 1, 931-934.

8. DUDLEY, E. F., BELDRIN, R. A. and JOHNSON, B. C. Climate, Water Hardness and Coronary Heart Disease. Journal of Chronic Diseases (1969). 22, 25-28.

9. POWELL, P., PACKHAM, R. F., and LACEY, R. F. et al. Water Quality and Cardiovascular Disease in British Towns. Technical Report TR 178, Water Research Centre, Medmenham (1982) 35pp.

10. LEARY, W. P., REYES, A. J. and LOCKETT, C. J. et al. Magnesium and Deaths ascribed to Ischaemic Heart Disease in South Africa. South African Medical Journal (1983) 64, 775-776.

11. COMSTOCK, G. W. Water Hardness and Cardiovascular Diseases. American Journal of Epidemiology (1979) 110, 378.

12. DEPARTMENT OF WATER AFFAIRS, PRETORIA. Program Version 5.4. Hydro Bank (1984).

13. LINDEMANN, R. D. and ASSENZO, J. R. Correlations between Water Hardness and Cardiovascular Deaths in Oklahoma. American Journal of Public Health (1964) 54, 1071-1077.

14. MACKINNON, A. V. and TAYLOR, S. H. Relationship between "Sudden" Coronary Deaths and Drinking Water Hardness in Five Yorkshire Cities and Towns. International Journal of Epidemiology (1980) 9, 247.

15. BIERENBAUM, M. L., FLEISCHMAN, A. I. and DUNN, J., et al. Possible Toxic Water Factor in Coronary Heart Disease. Lancet (1975) 1, 1008-1010.

16. ALLWRIGHT, S. P. A., SHANE, P. A. and COULSON, A., et al. Mortality and Water Hardness. Lancet (1974) 2, 860-864.

17. HEYDEN, S. The Hard Facts behind the Hard Water Theory and Ischaemic Heart Disease. Journal of Chronic Disease (1976) 29, 259-266.

18. PUNSAR, S. Cardiovascular Mortality and Quality of Drinking Water. An Evaluation of the Literature from an Epidemiological Point of View. Work-Environ-Health (1973) 10, 107-125.

19. MINISTERIE VAN VOLKSGEZONDHEID EN MILIEUHYGIENE, NEDERLAND. Gezondheidsaspecten van Centrale Ontharding van Leidingwater (1982) Rapport van der werkgroup, p12.

20. DEPARTMENT OF CENTRAL STATISTICAL SERVICES, PRETORIA. Social Characteristics. Population Census 80, South Africa. (1980) 02-80-02, pp vii-xv.

21. MIDLANE, L. J. and RIP, M. R. A Spatial Analysis of Mortality in the Western Cape. South African Geographical Bulletin (1987), in press.

22. DEPARTMENT OF STATISTICS, PRETORIA. Statistical Classification of Diseases, Injuries and Causes of Death (1979) 07-03-15, pp. 23-27.

23. GILLIES, M. E. and PAULIN, H. V. Estimations of Daily Mineral Intakes from Drinking Water. Human Nutrition : Applied Nutrition (1982) 36A, 290.

24. ROBERTSON, J. S. Minerals and Mortality. Community Health (1977) 8, 227.

25. DERRY, C. W. The Importance of Appropriate Chemical Quality Data in Research on Health Aspects of Water (poster and extended abstract). First Symposium on Water Quality Assessment, 26-27 May 1986, Pretoria.

26. AMERICAN PUBLIC HEALTH ASSOCIATION. Standard Methods for the Examination of Water and Waste Water (1985) Washington. A.P.H.A. Press, p209.

27. AUSTIN, R. G. Aids to Inorganic Chemistry (1960) London. Bailliere, Tindall and Cox, pp. 69-70.

28. BOTHA, J. L. and BRADSHAW, D. African Vital Statistics - a Black Hole. South African Medical Journal (1985) 67, 977-982.

29. DERRY, C. W., BOURNE, D. E. and SAYED, A. R., et al. Mortality (1978-1982) of Coloured, White and Asian Population Groups in South Africa. II: Ischaemic Heart Disease. South African Medical Journal (1987), in press.

30. FLEISS, J. C. Statistical Methods for Rates and Proportions (1981) New York. Wiley, p240.

31 STATISTICAL SOFTWARE INC., CALIFORNIA. Biomedical Data Package (BMDP), Program Version for Sperry 1100 Series Computers. University of California (1983).

32. FORSTER, F. Use of a Demographic Base Map for the Presentation of Areal Data in Epidemiology. British Journal of Preventive and Social Medicine (1966) 20, 156-171.

33. WYNDHAM, C. H. and FELLINGHAM, S. A. Climate and Disease. South African Medical Journal (1978) 53, 1051.

34. ROSSOUW, J. E., DU PLESSIS, J. P. and BENADE, A. J. S., et al. Coronary Risk Factor Screening in Three Rural Communities. South African Medical Journal (1983) 64, 430-436.

35. STEIN, E. A. The Lipid Disorders Centre at the Transvaal Memorial Hospital for Children. South African Medical Journal (1977) 52, 1023-1030.

36. KAPLAN, N. M., CARNEGIE, A. and RASKIN, P., et al. Potassium Supplementation in Hypertensive Patients with Diuretic-Induced Hypokalaemia. New England Journal of Medicine (1985) 312, 746-749.

37. YAMORI, Y., KIHARA, M. and NARA, Y., et al. Hypertension and Diet : Multiple Regression Analysis in a Japanese Farming Community. Lancet (1981) 1, 1204-1206.

38. SASAKI, N. High Blood Pressure and the Salt Intake of the Japanese. Japanese Heart Journal (1962) 3, 313-316.

39. LANGFORD, H. G. Dietary Potassium and Hypertension: Epidemiologic Data. Annals of Internal Medicine (1983) 98, 770-772.

40. GILLIES, M. E. and PAULIN, H. V. Estimations of Daily Mineral Intakes from Drinking Water. Human Nutrition : Applied Nutrition (1982) 36A, 289.

APPENDIX A

DISTRIBUTIONS OF TOTAL WATER HARDNESS AND ITS CONTRIBUTORY AND ASSOCIATED FACTORS IN POTABLE WATER BY NUMBER OF URBAN MAGISTERIAL DISTRICTS

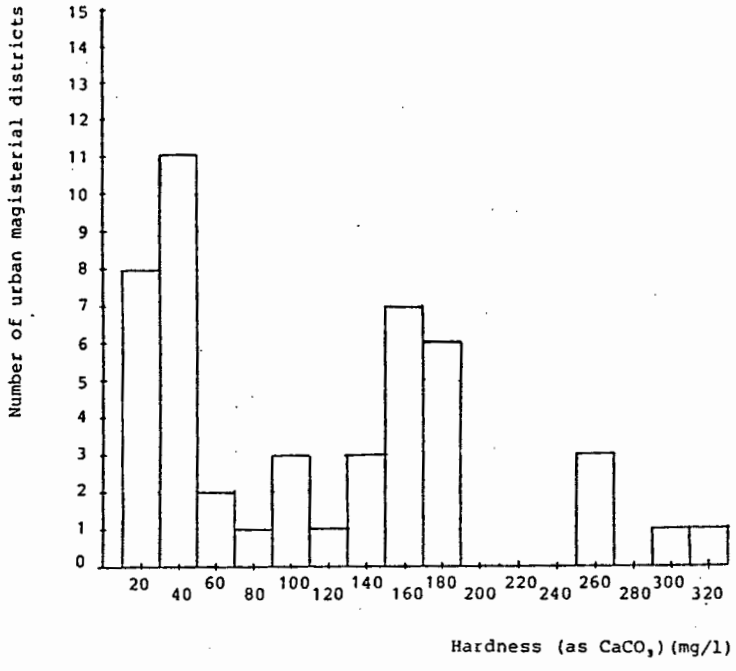

Figure 8:
Distribution of hardness (as $CaCO_3$) levels in potable water for 47 urban magisterial districts.

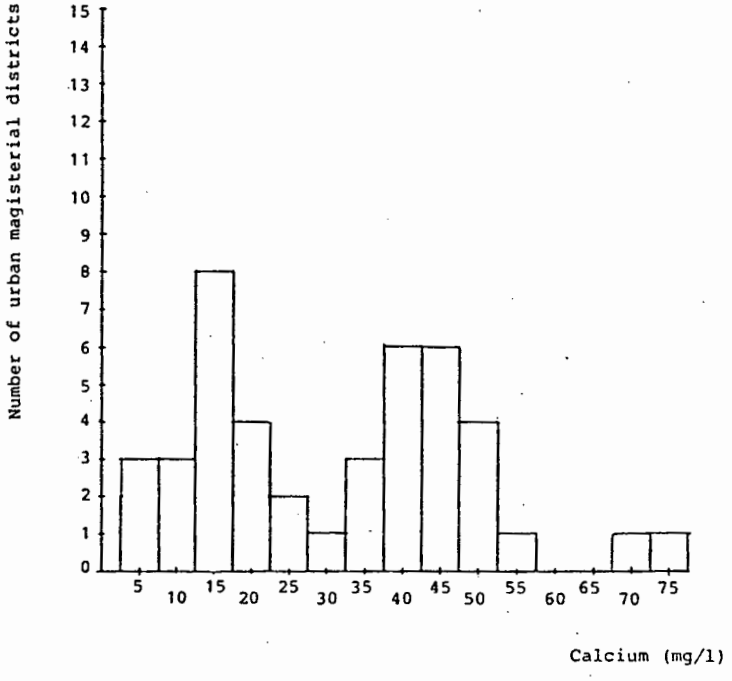

Figure 9:
Distribution of calcium levels in potable water for 43 urban magisterial districts.

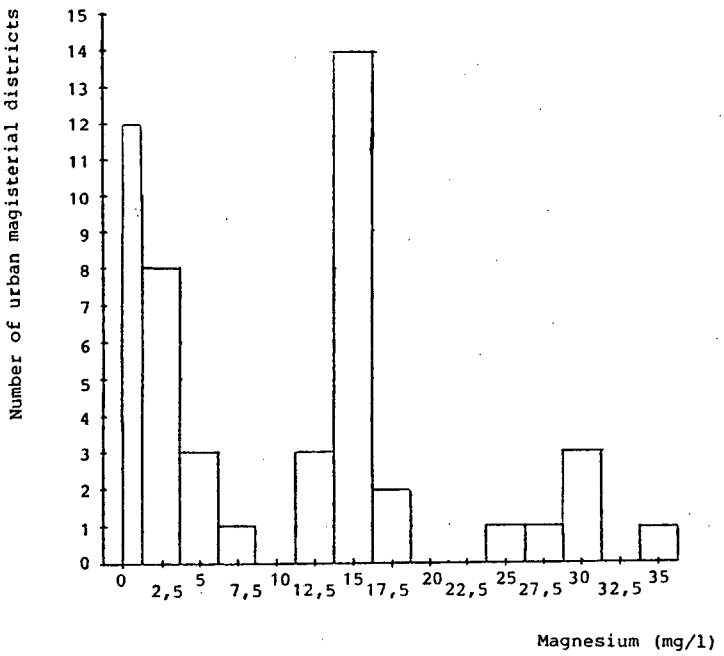

Figure 10:
Distribution of magnesium levels in potable water for 43 urban magisterial districts.

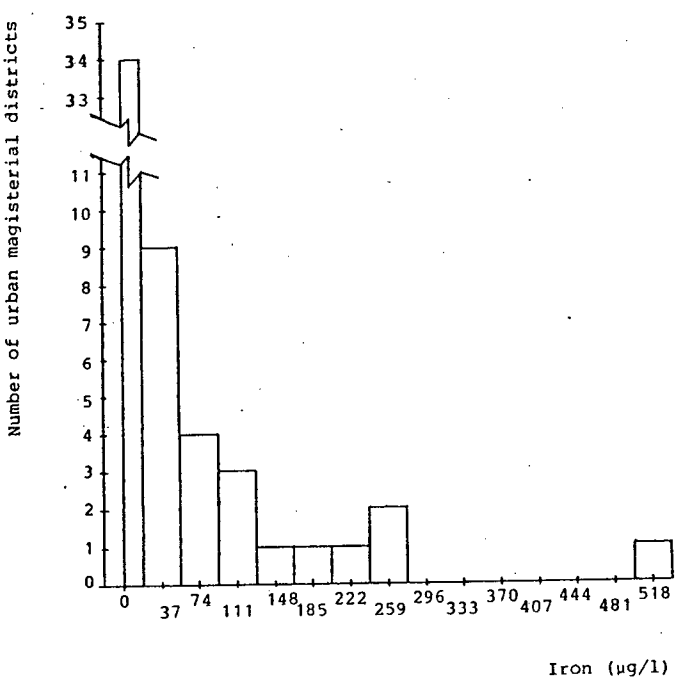

Figure 11:
Distribution of iron levels in potable water for 39 urban magisterial districts.

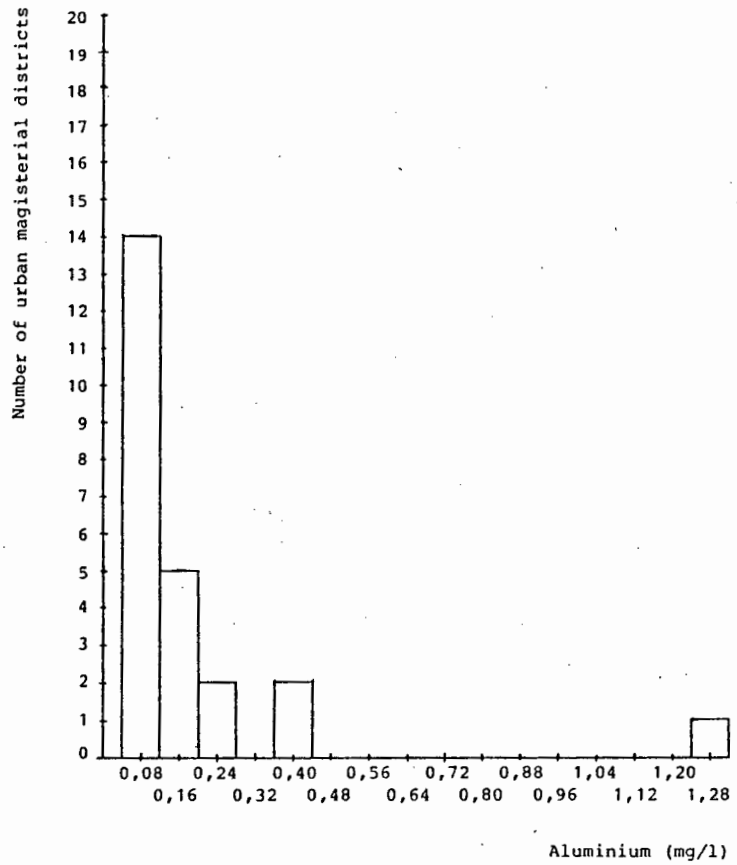

Figure 12:
Distribution of aluminium levels in potable water for 24 urban magisterial districts.

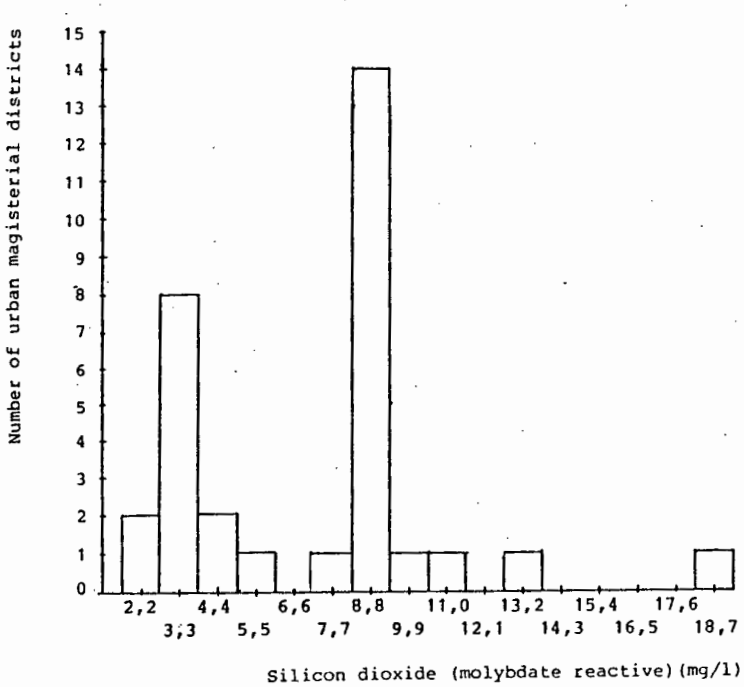

Figure 13:
Distribution of silicon dioxide (molybdate reactive) levels in potable water for 32 urban magisterial districts.

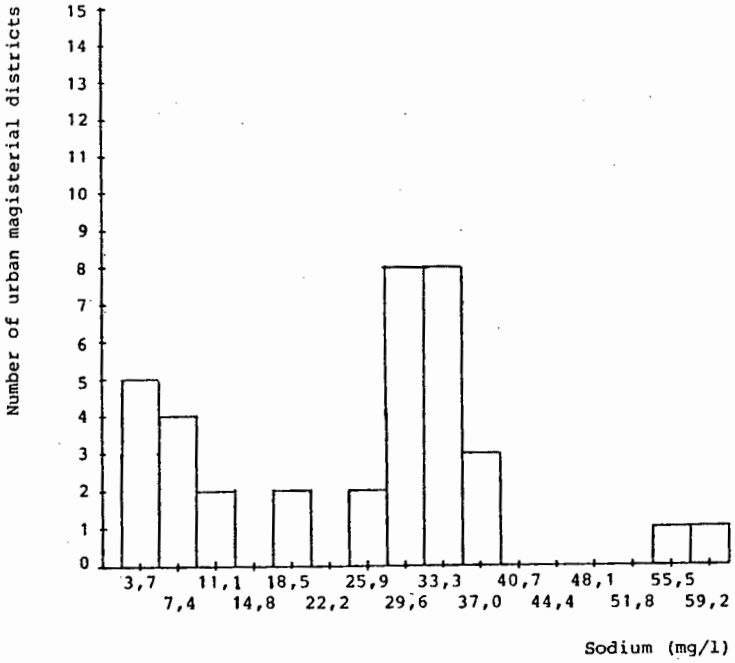

Figure 14:
Distribution of sodium levels in potable water for 36 urban magisterial districts.

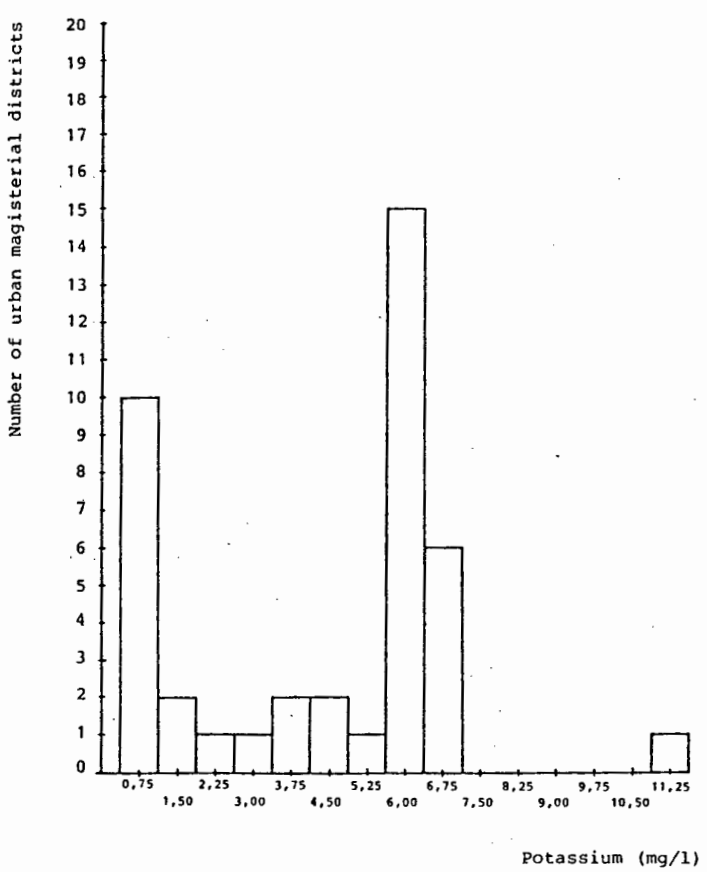

Figure 15:
Distribution of potassium levels in potable water for 41 urban magisterial districts.

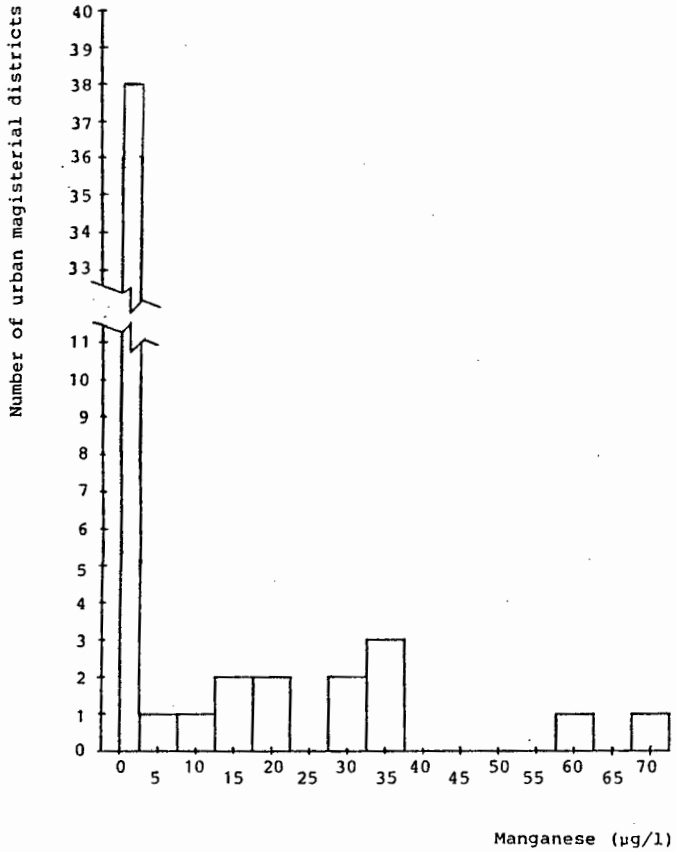

Figure 16:
Distribution of manganese levels in potable water for 32 urban magisterial districts.

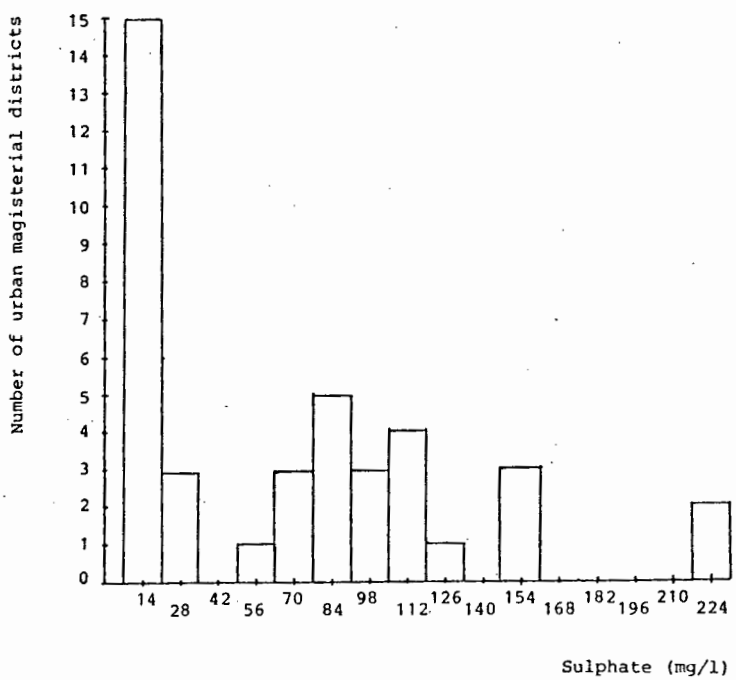

Figure 17:
Distribution of sulphate levels in potable water for 40 urban magisterial districts.

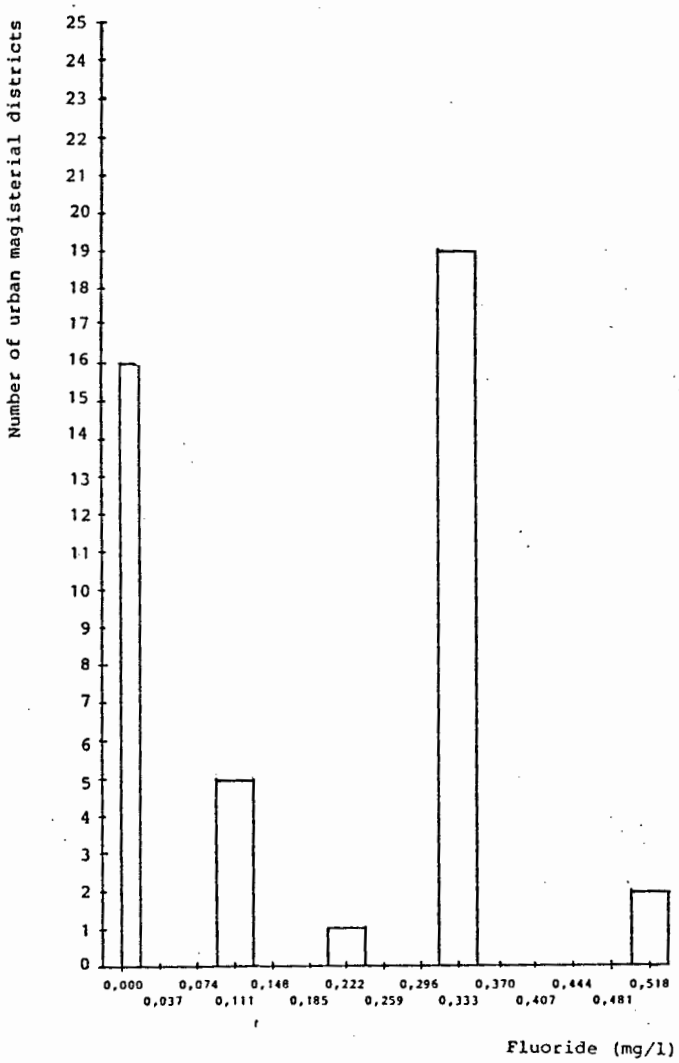

Figure 18:
Distribution of fluoride levels in potable water for 35 urban magisterial districts.

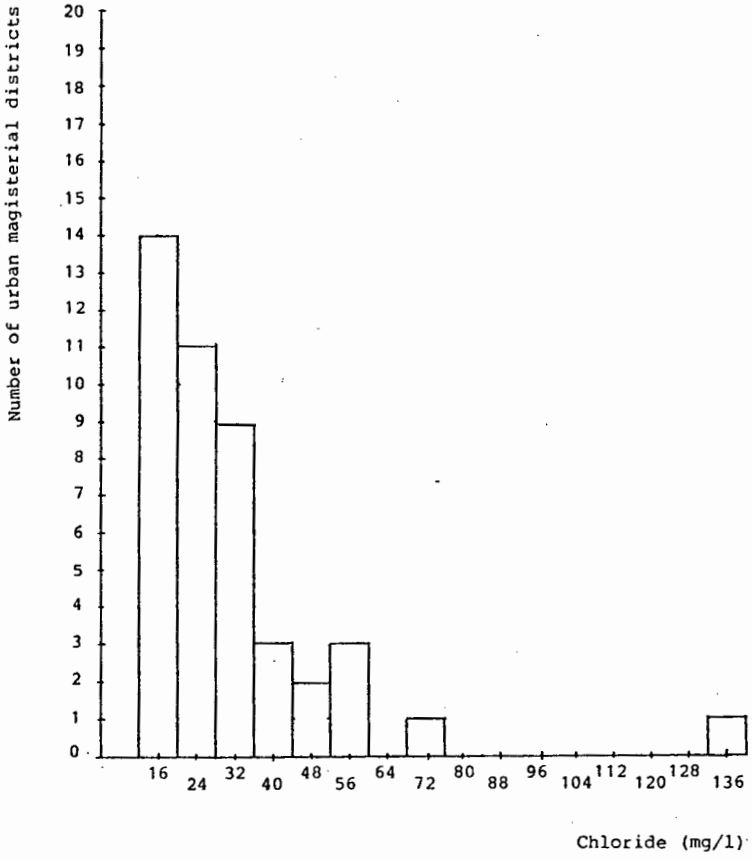

Figure 19:
Distribution of chloride levels in potable water for 44 urban magisterial districts.

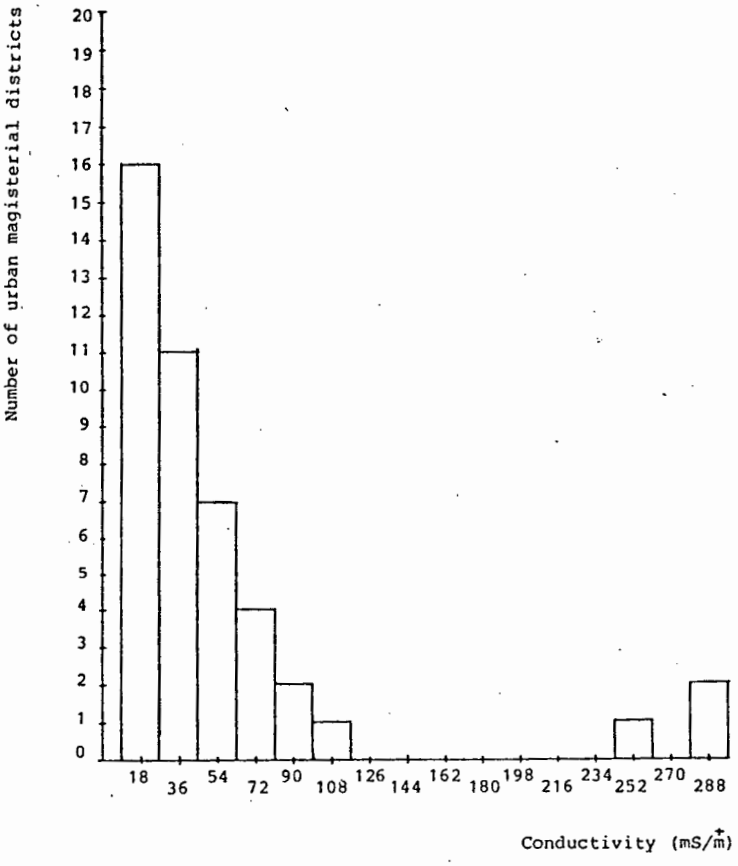

Figure 20:
Distribution of conductivity levels in potable water for 44 urban magisterial districts.

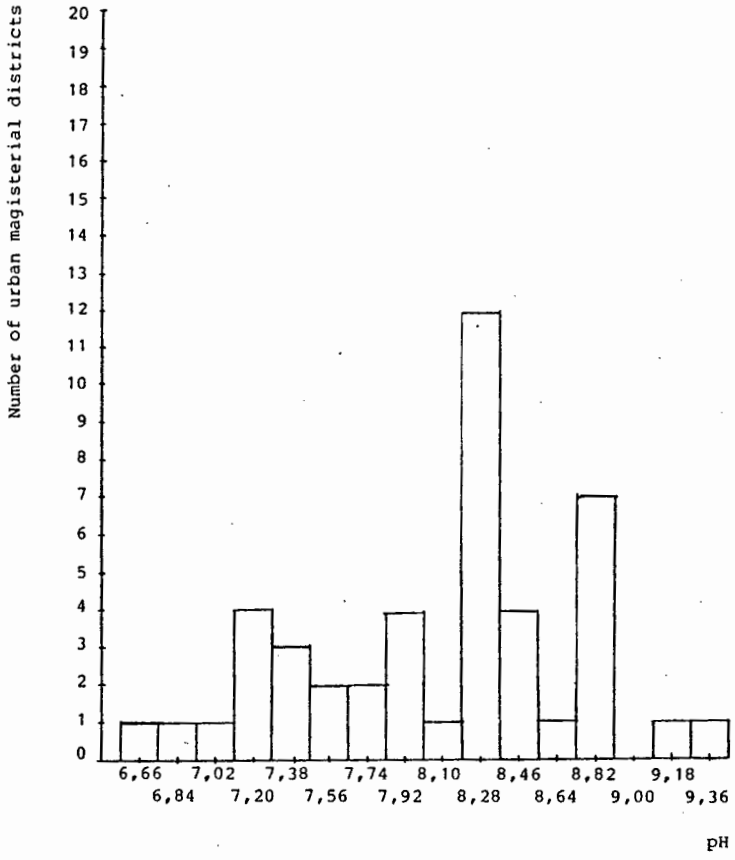

Figure 21:
Distribution of pH levels in potable water for 45 urban magisterial districts.

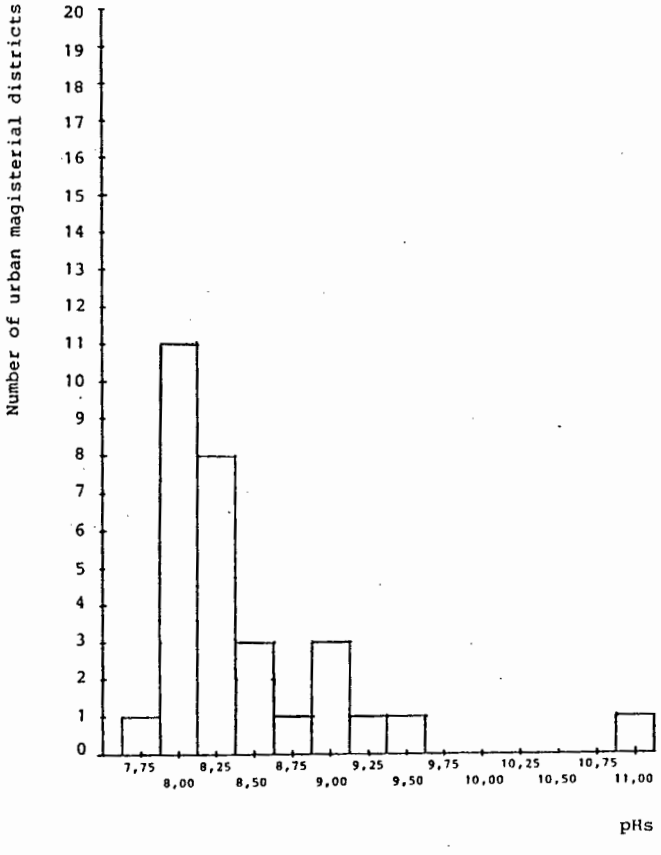

Figure 22:
Distribution of pHs levels in potable water for 30 urban magisterial districts.

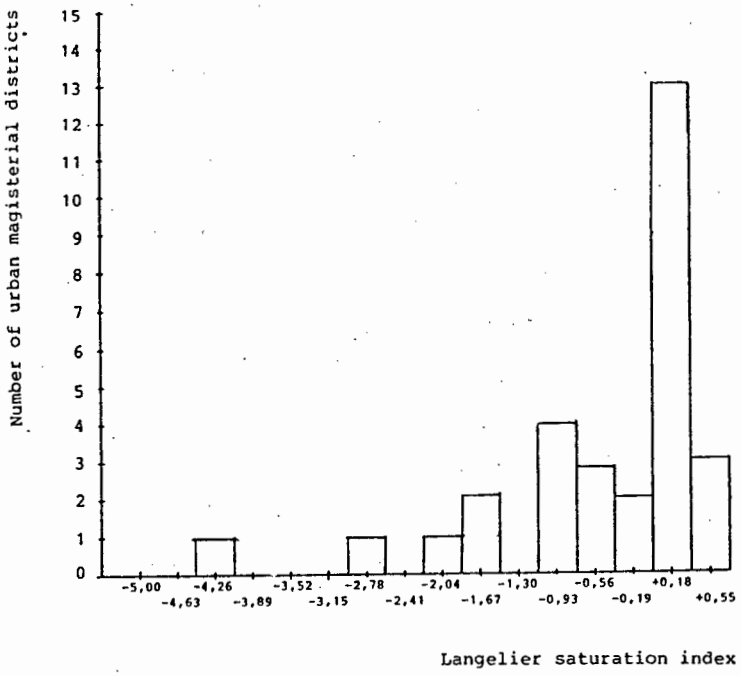

Figure 23:
Distribution of Langelier saturation indices for potable water for 30 urban magisterial districts.

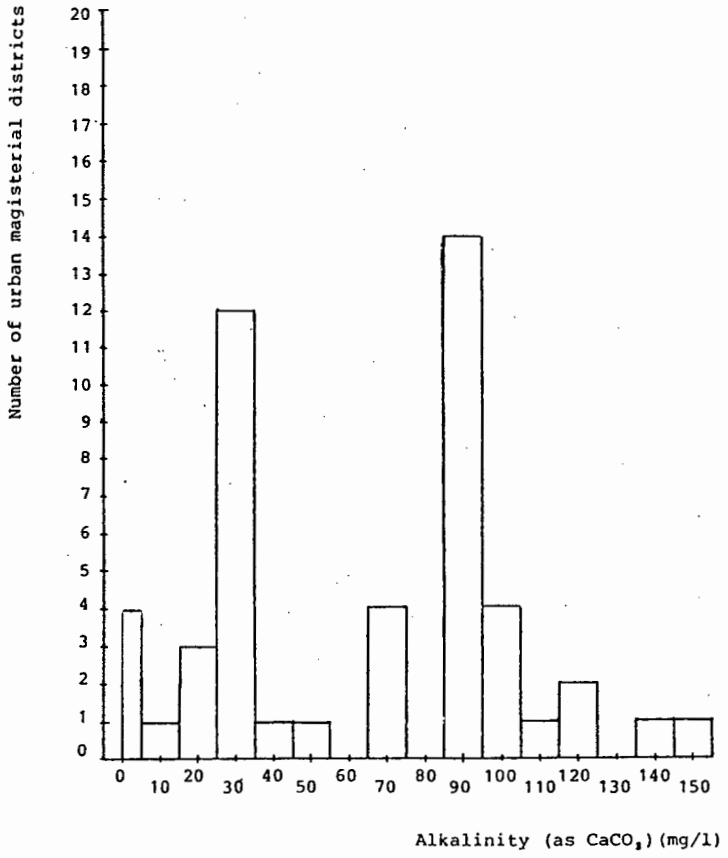

Figure 24:
Distribution of alkalinity (as $CaCO_3$) levels in potable water for 47 urban magisterial districts.

APPENDIX B

SCATTER PLOTS OF STANDARDIZED MORTALITY RATIOS FOR CARDIOVASCULAR AND ISCHAEMIC HEART DISEASE (MALE AND FEMALE) PLOTTED AGAINST TOTAL WATER HARDNESS AND ITS CONTRIBUTORY AND ASSOCIATED FACTORS IN POTABLE WATER

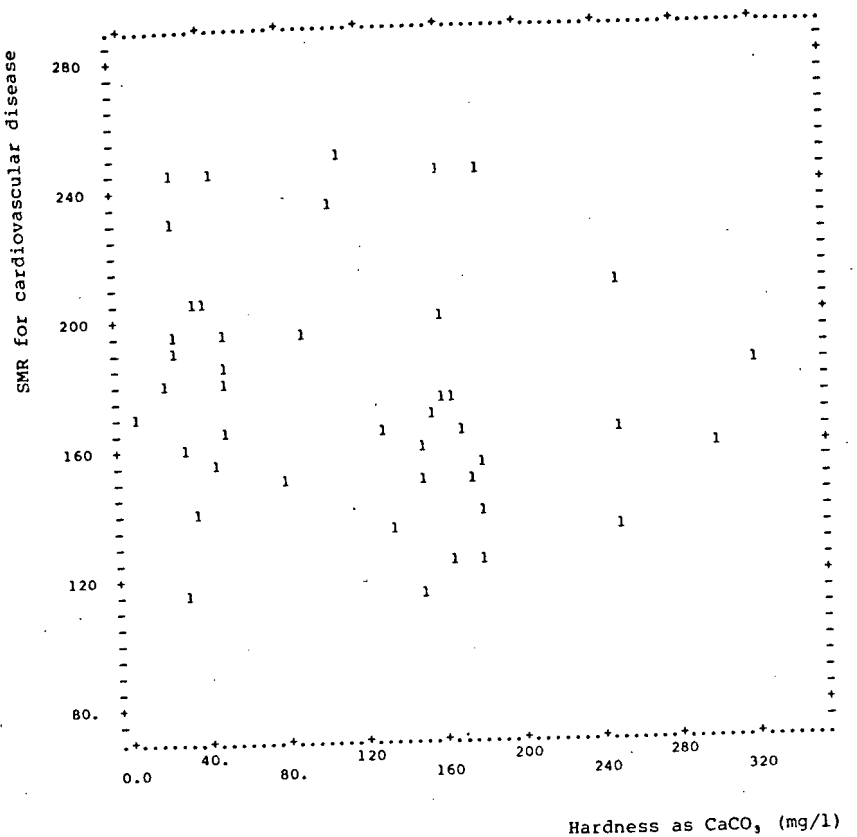

Figure 25:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the hardness of potable water as $CaCO_3$ (n=43, unweighted)

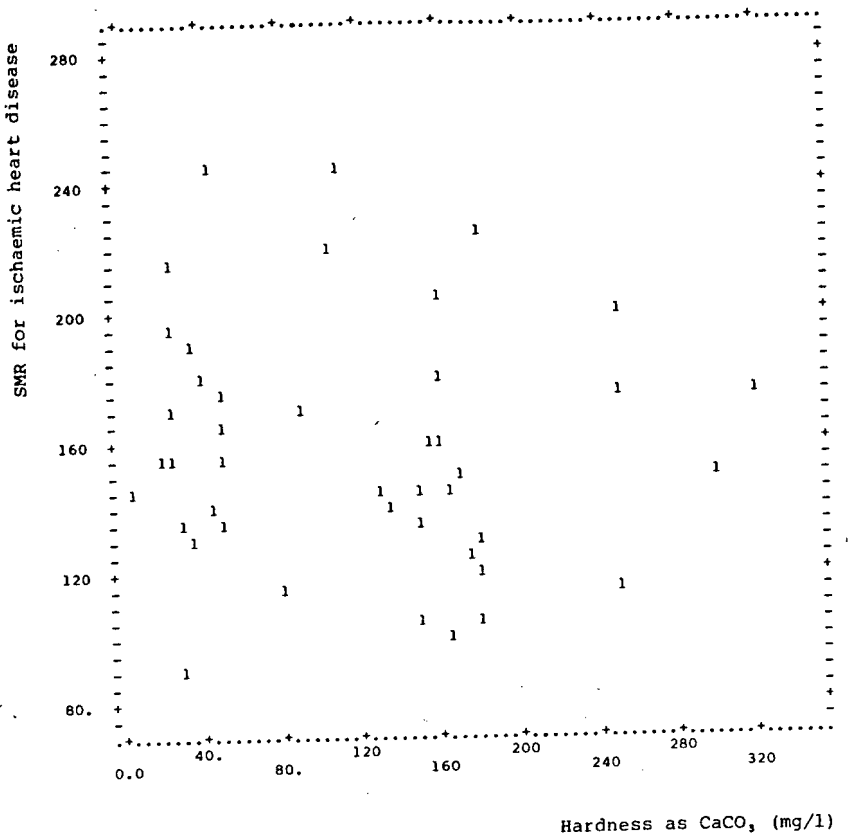

Figure 26:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the hardness of potable water as $CaCO_3$ (n=43, unweighted)

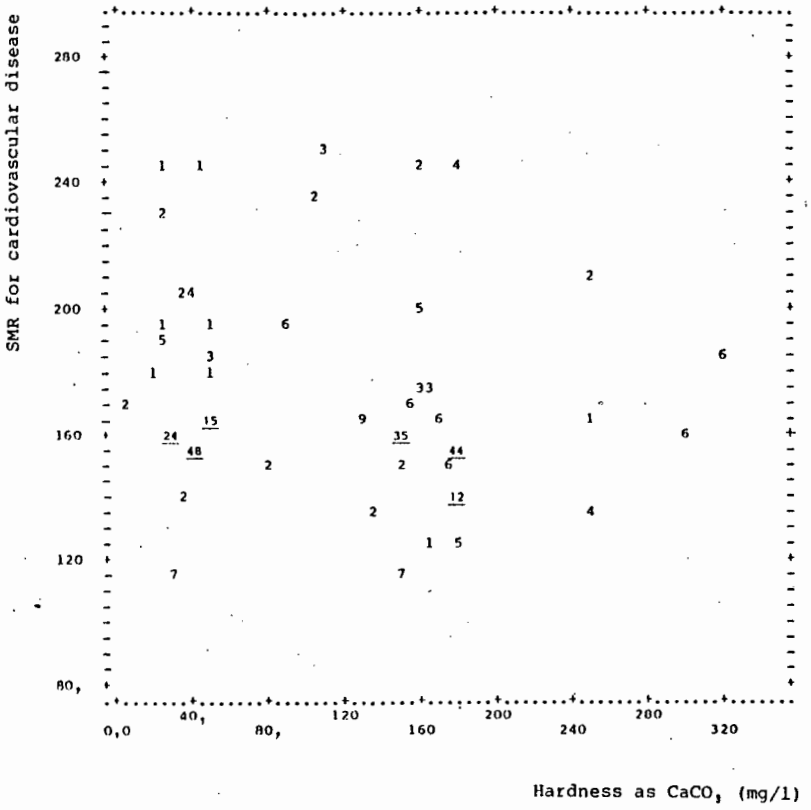

Figure 27:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the hardness of potable water as $CaCO_3$ (n=43, weighted)

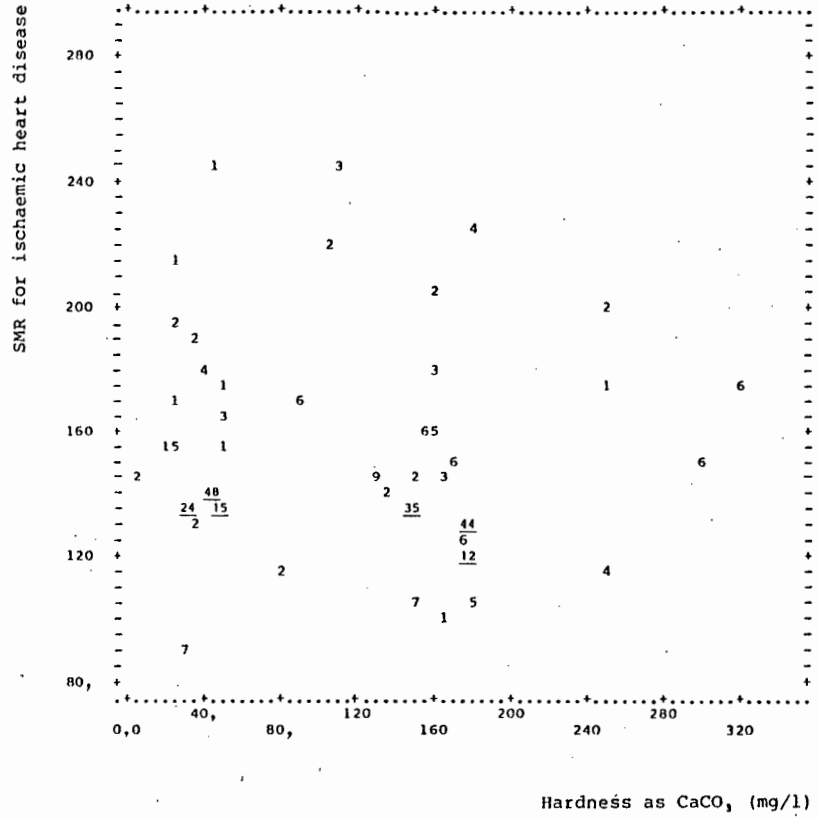

Figure 28:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the hardness of potable water as $CaCO_3$ (n=43, weighted)

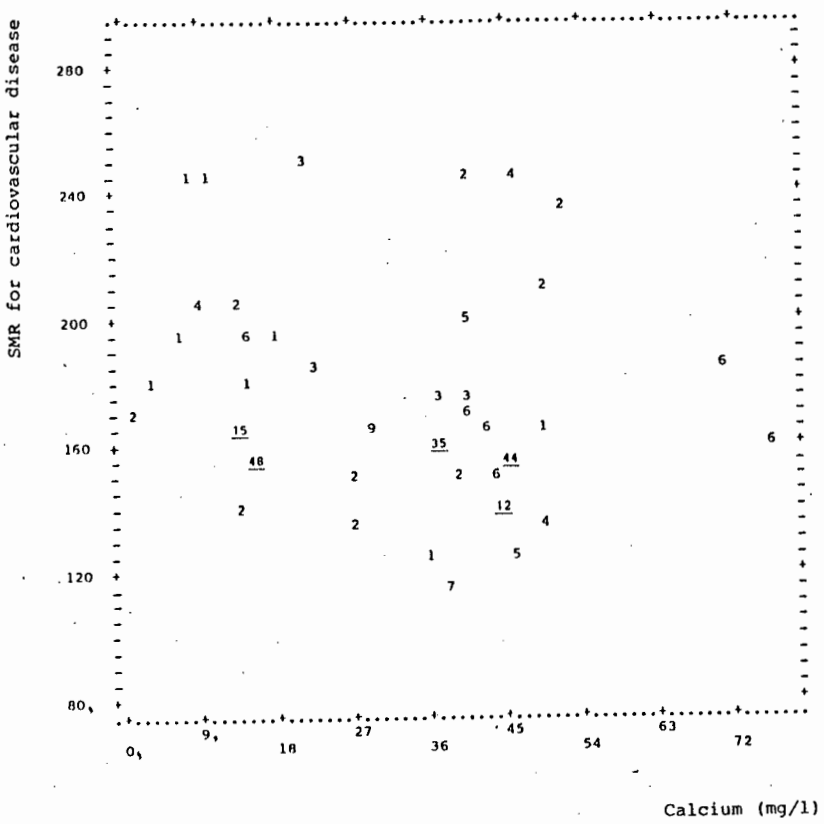

Figure 29:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against calcium in potable water.
(n=39, weighted)

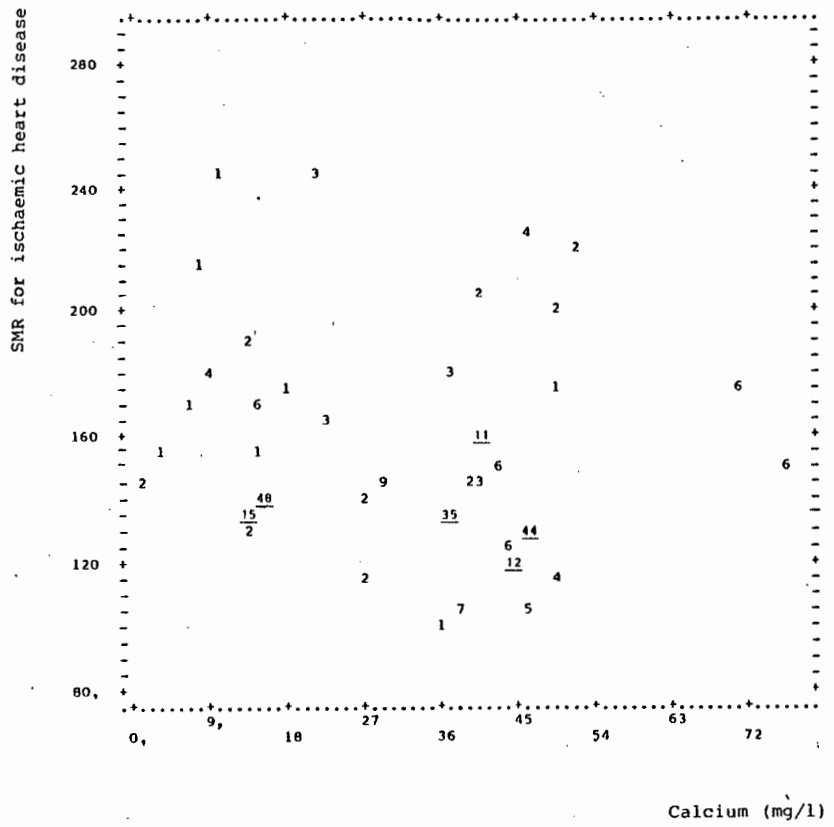

Figure 30:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against calcium in potable water.
(n=39, weighted)

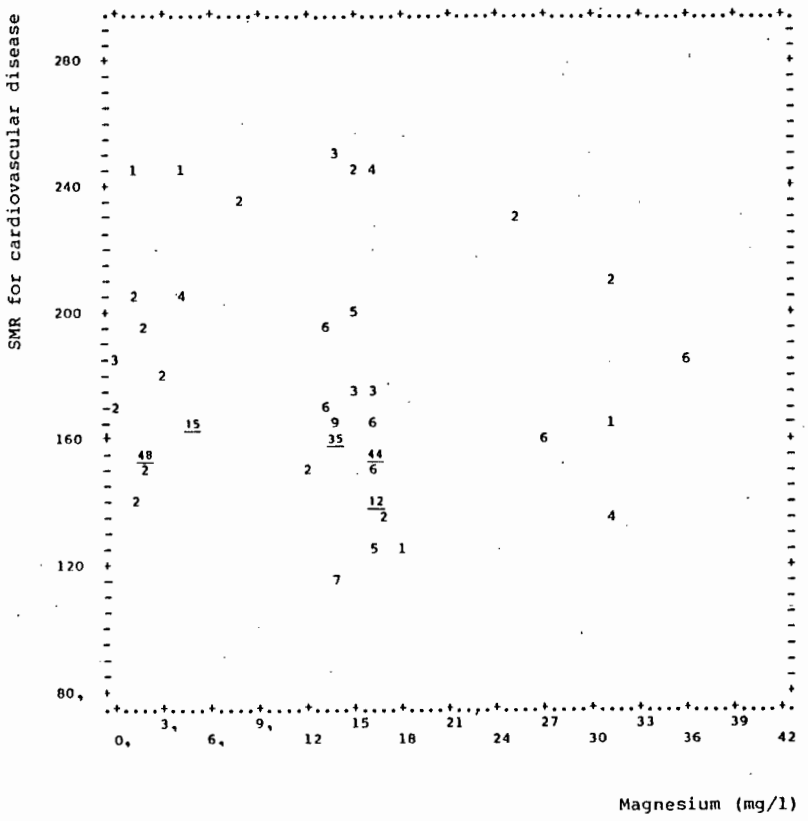

Figure 31:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against magnesium in potable water.
(n=40, weighted)

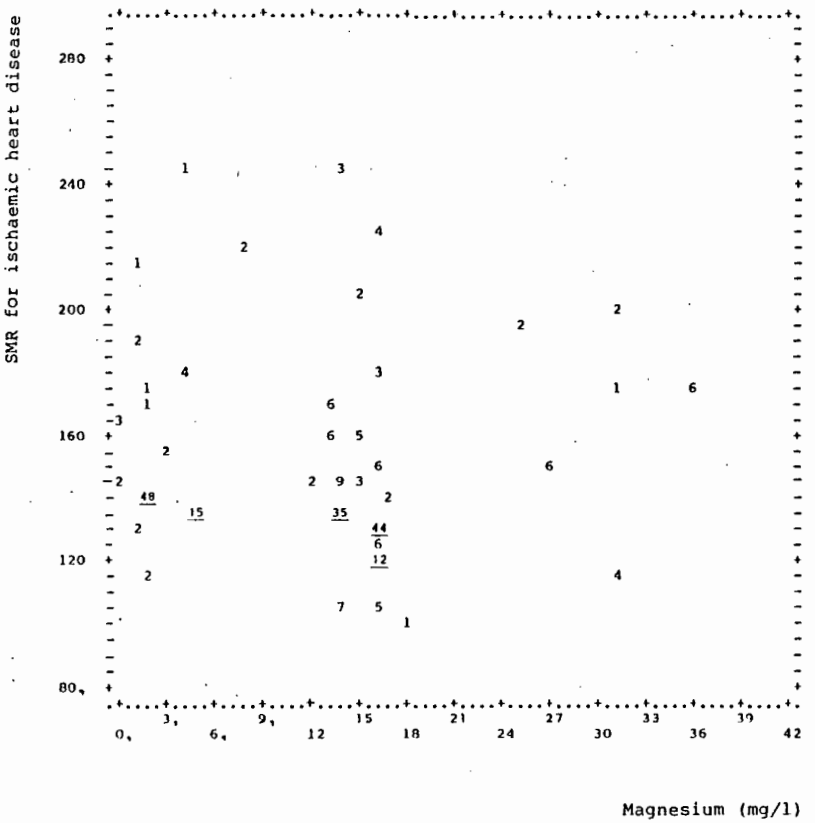

Figure 32:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against magnesium in potable water.
(n=40, weighted)

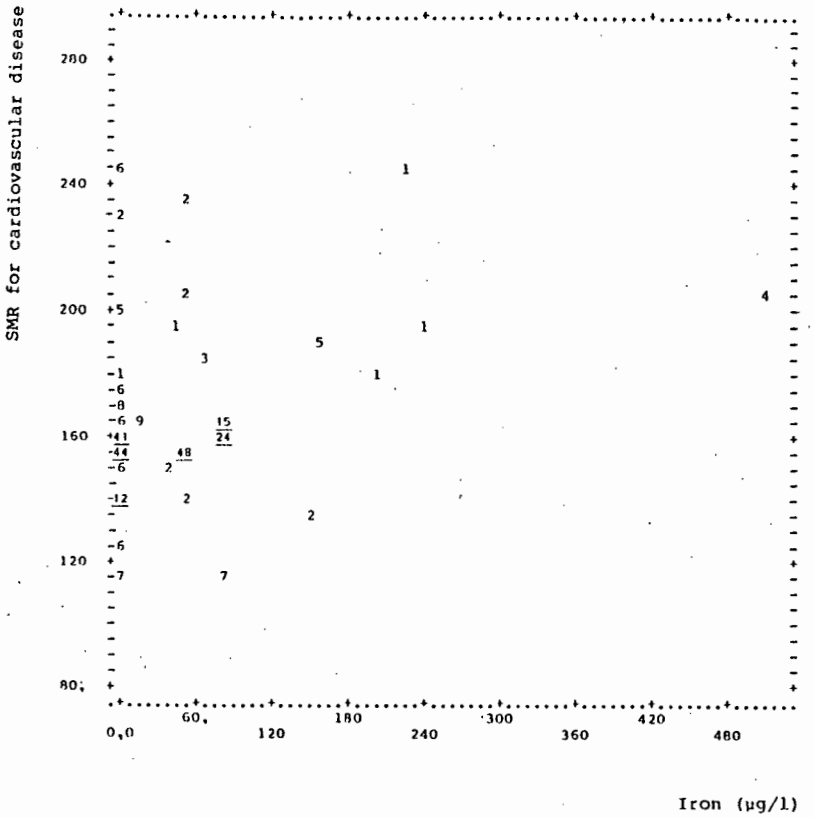

Figure 33:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against iron in potable water.
(n=35, weighted)

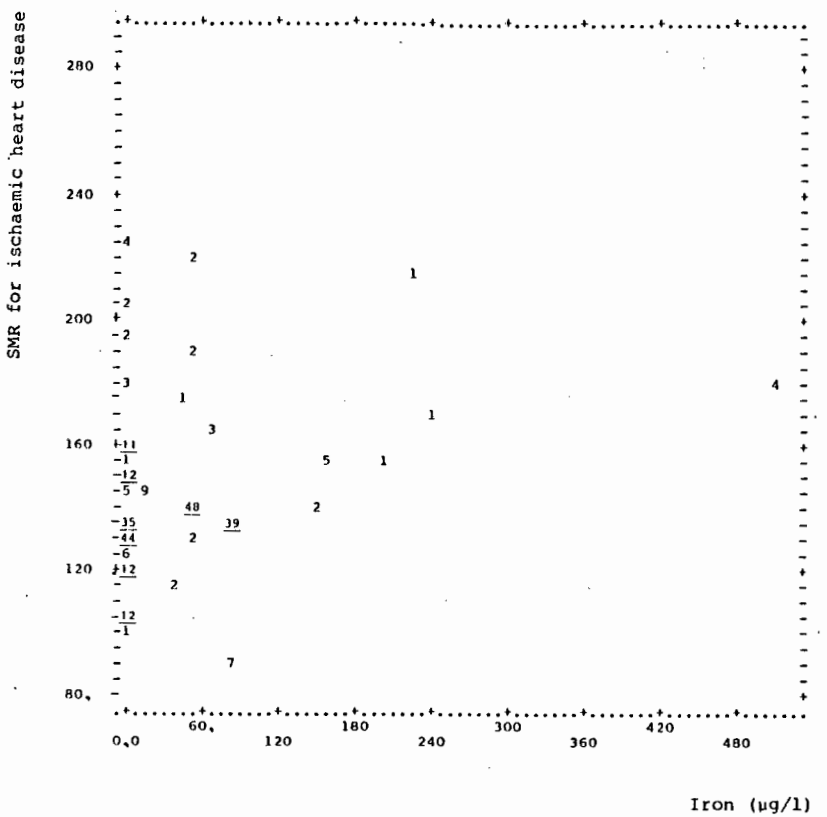

Figure 34:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against iron in potable water.
(n=35, weighted)

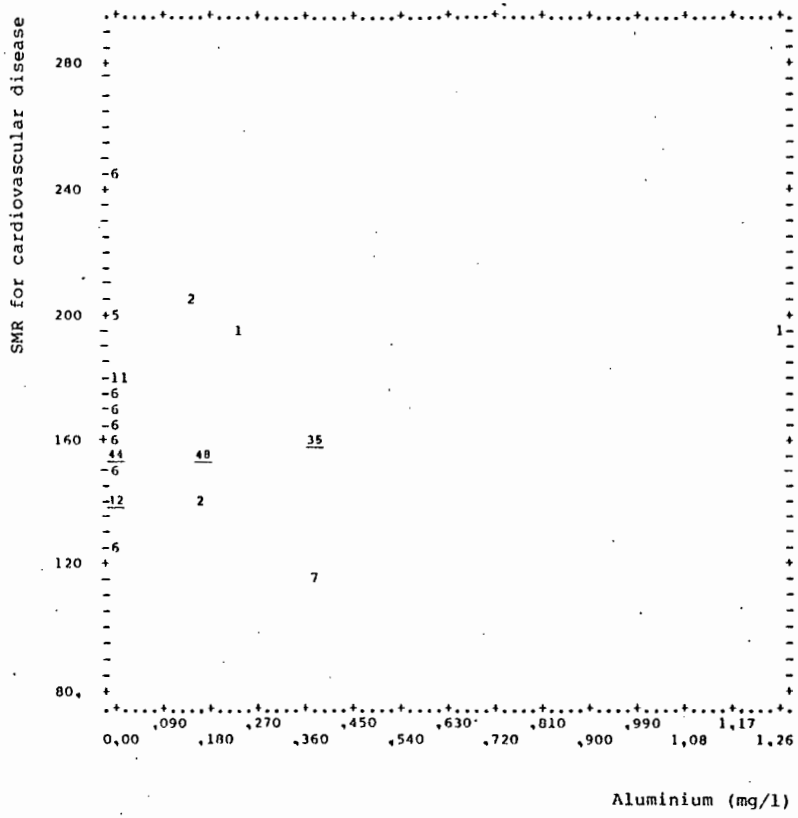

Figure 35:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against aluminium in potable water.
(n=22, weighted)

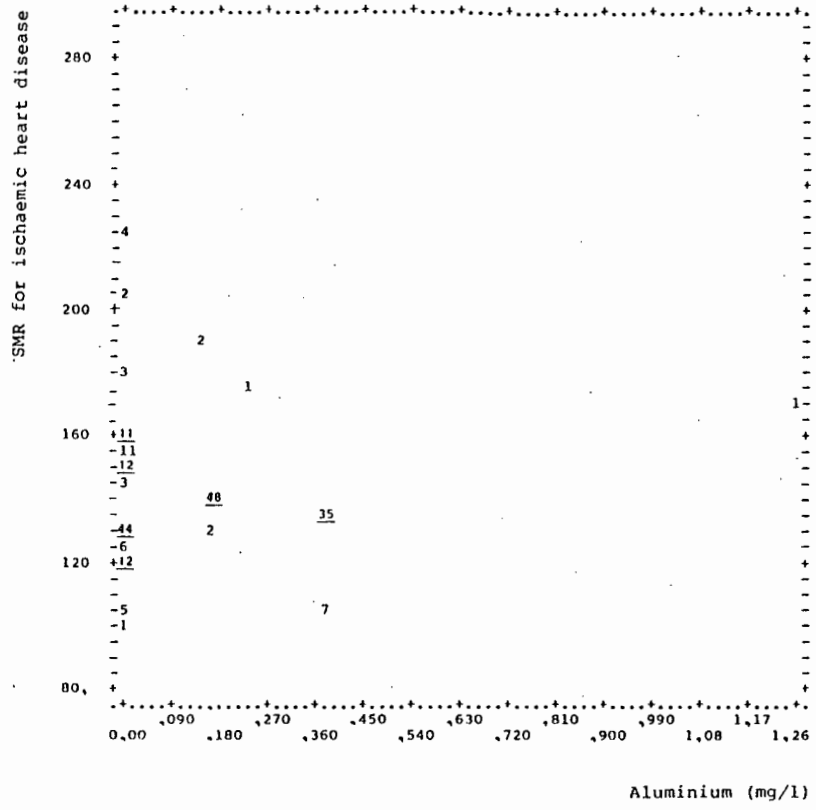

Figure 36:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against aluminium in potable water.
(n=22, weighted)

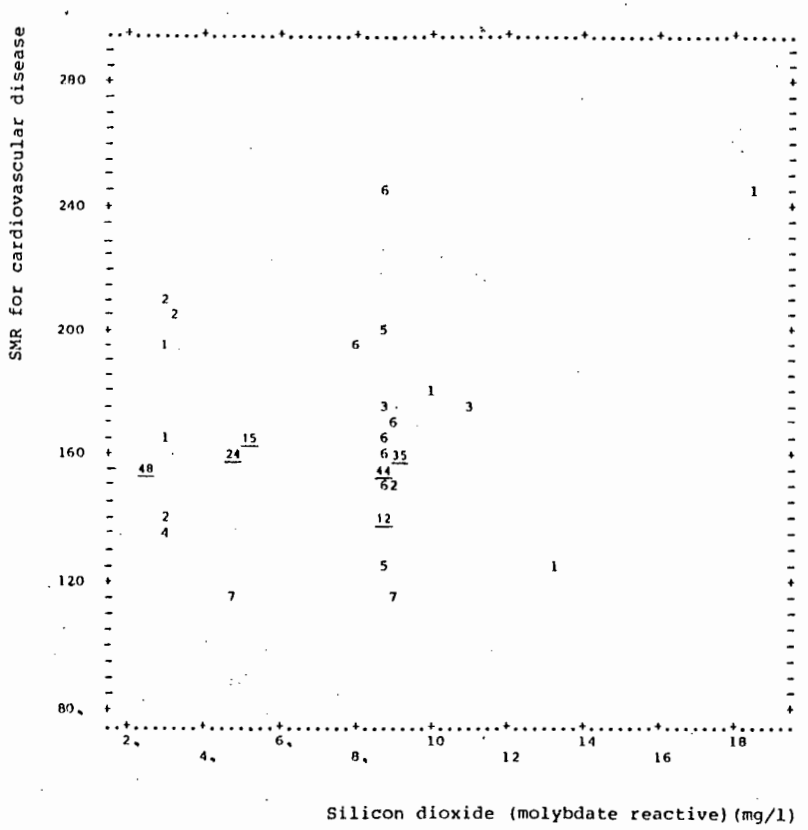

Figure 37:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against silicon dioxide (molybdate reactive) in potable water. (n=29, weighted)

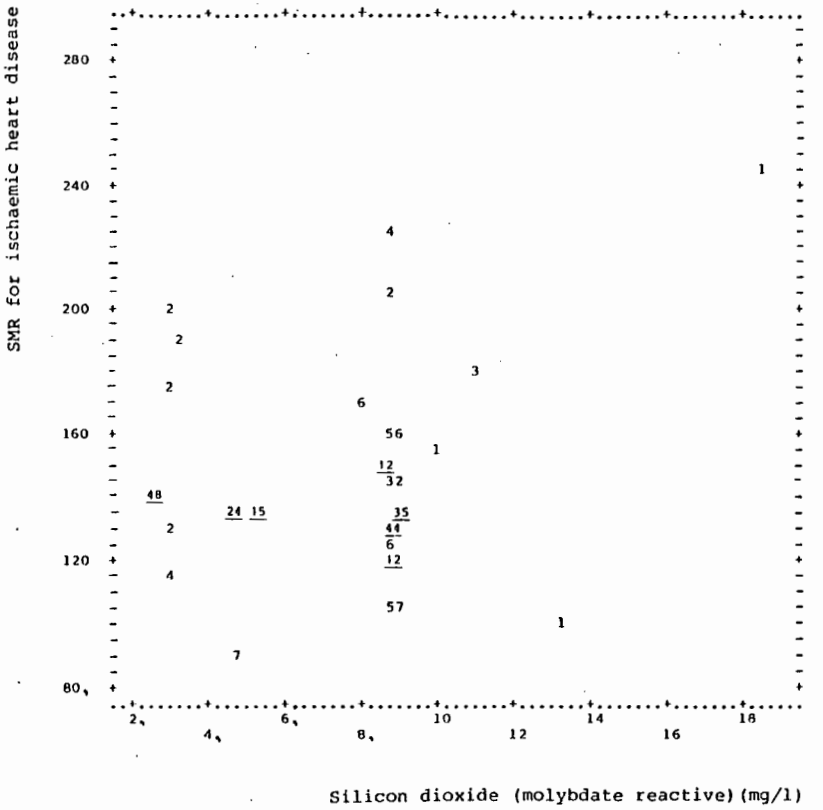

Figure 38:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against silicon dioxide (molybdate reactive) in potable water. (n=29, weighted)

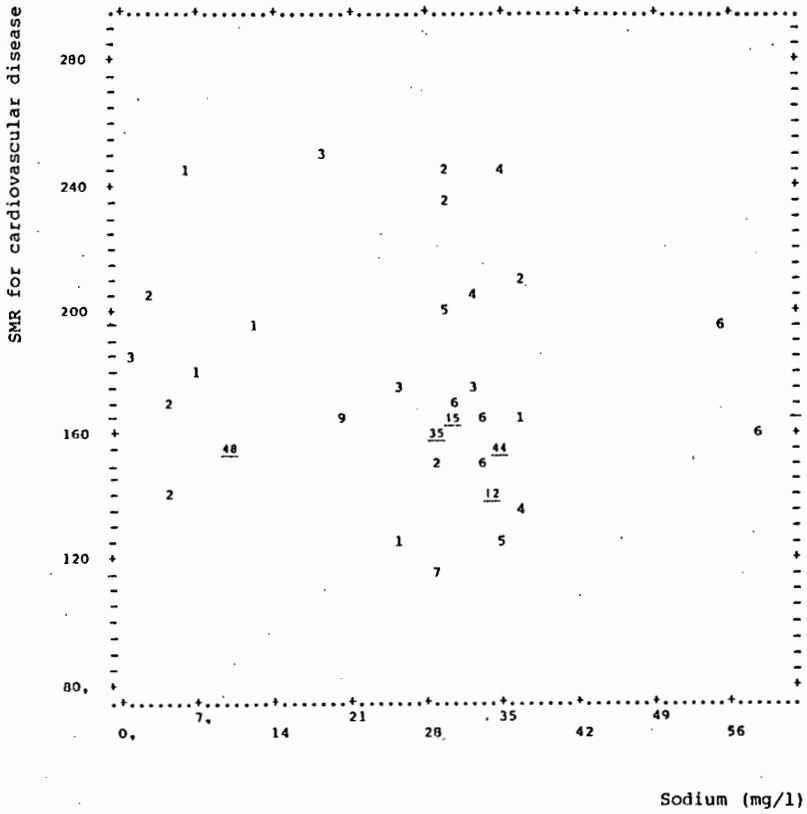

Figure 39:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against sodium in potable water.
(n=33, weighted)

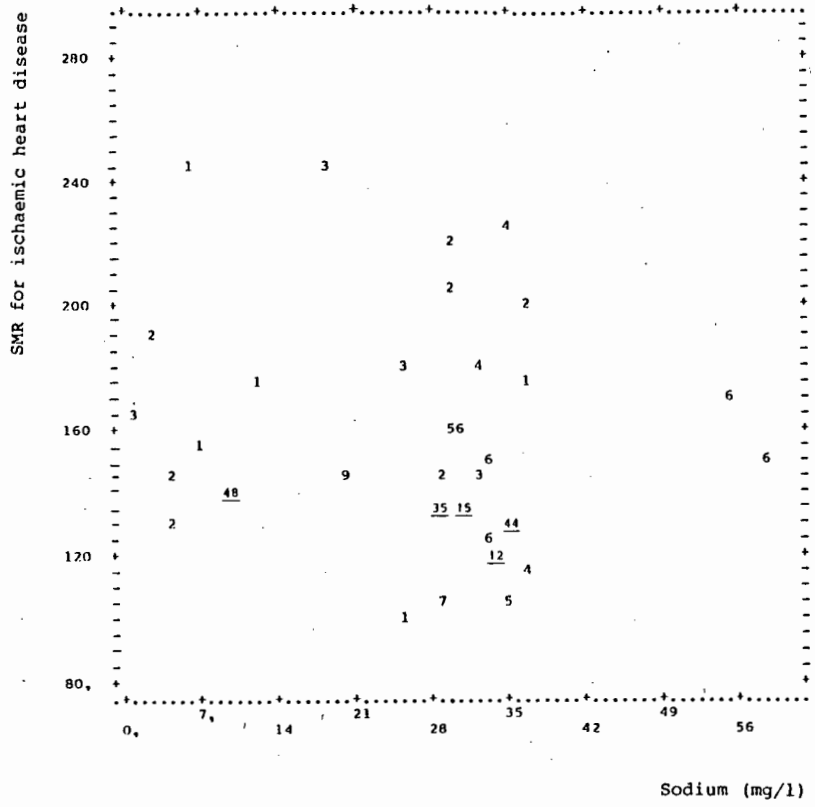

Figure 40:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against sodium in potable water.
(n=33, weighted)

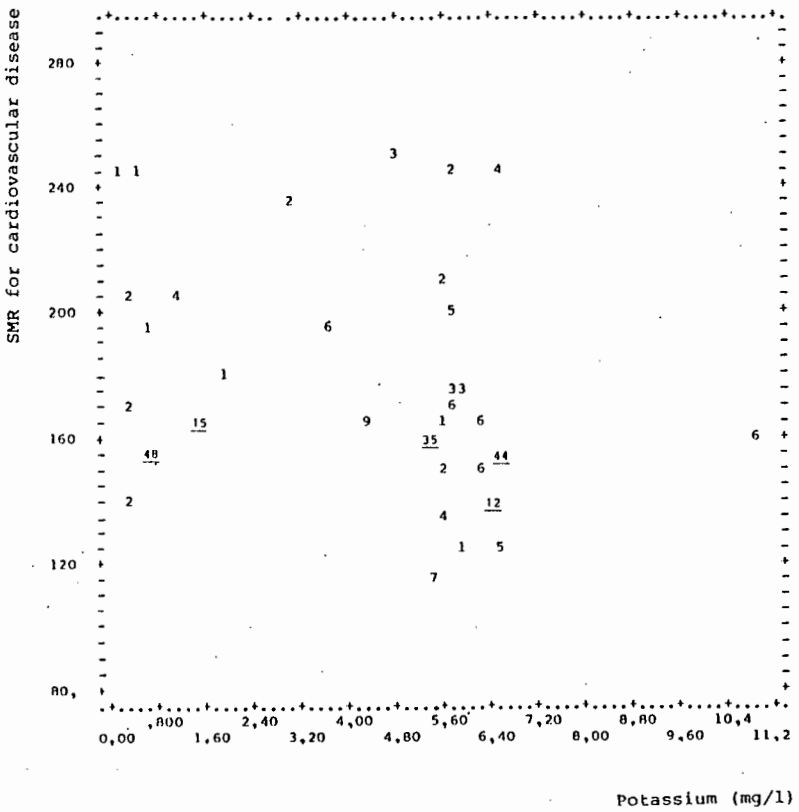

Figure 41:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against potassium in potable water.
(n=33, weighted)

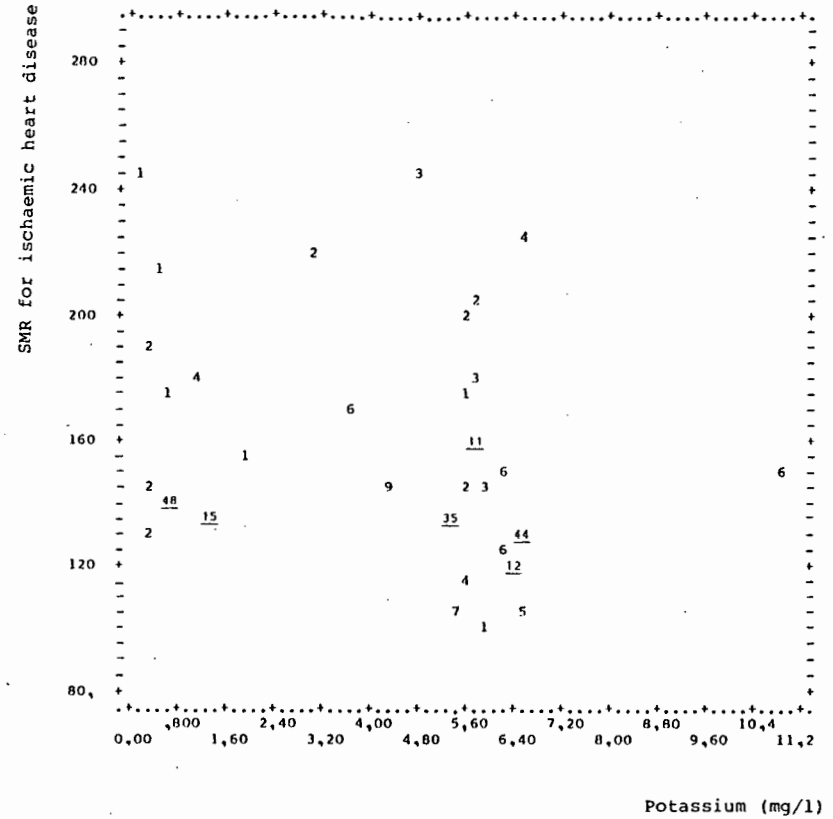

Figure 42:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against potassium in potable water.
(n=33, weighted)

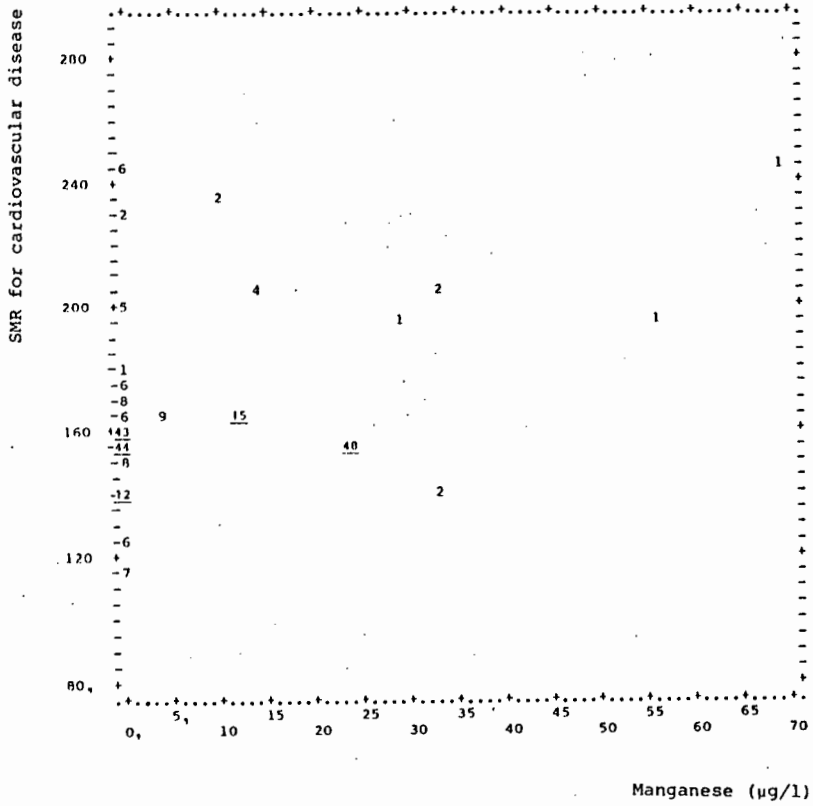

Figure 43:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against manganese in potable water.
(n=29, weighted)

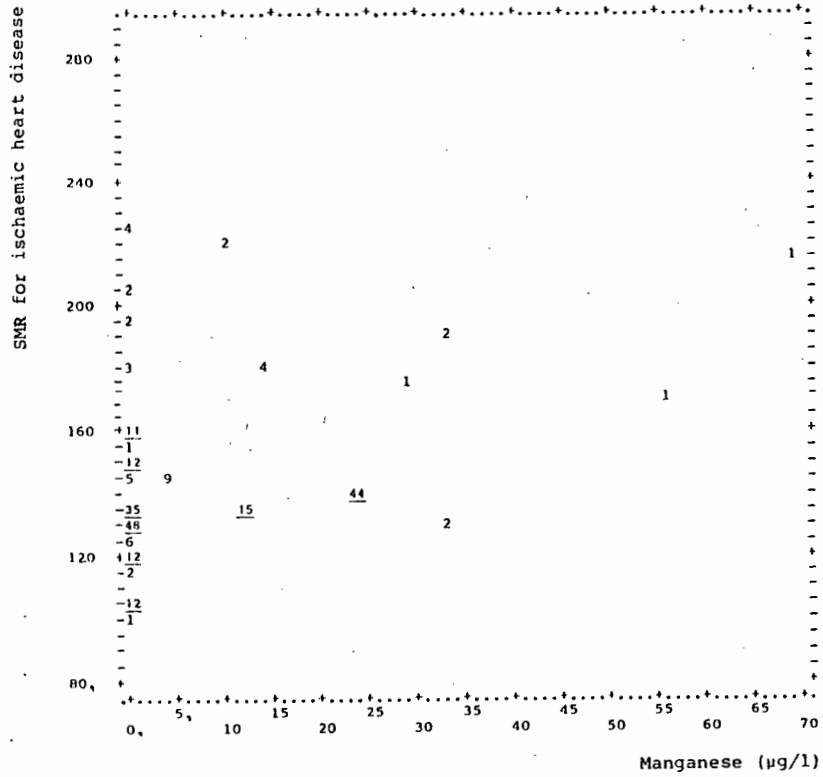

Figure 44:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against manganese in potable water.
(n=29, weighted)

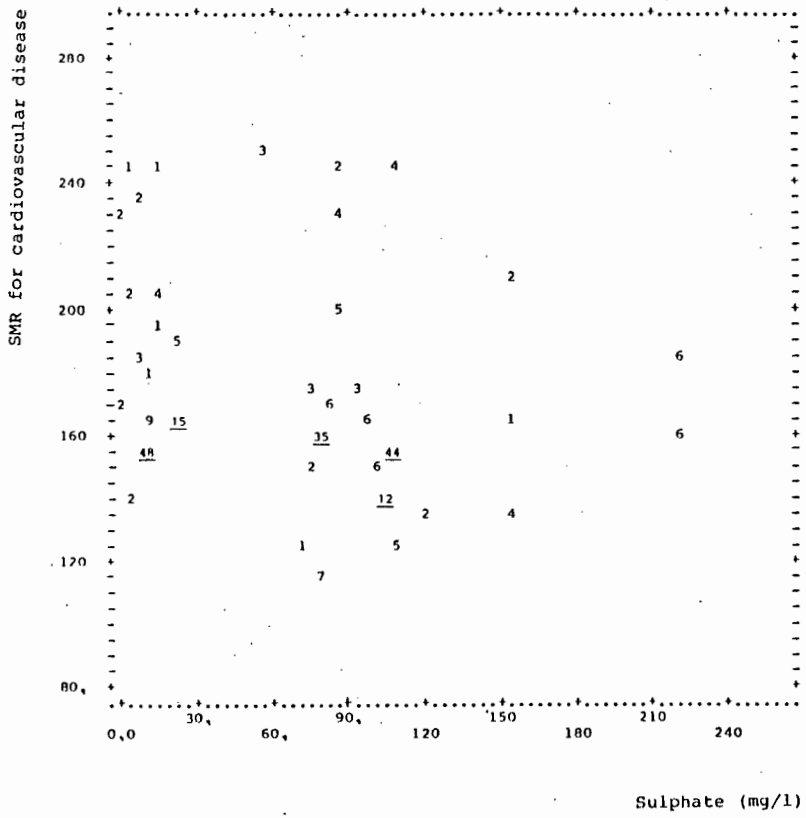

Figure 45:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against sulphate in potable water.
(n=37, weighted)

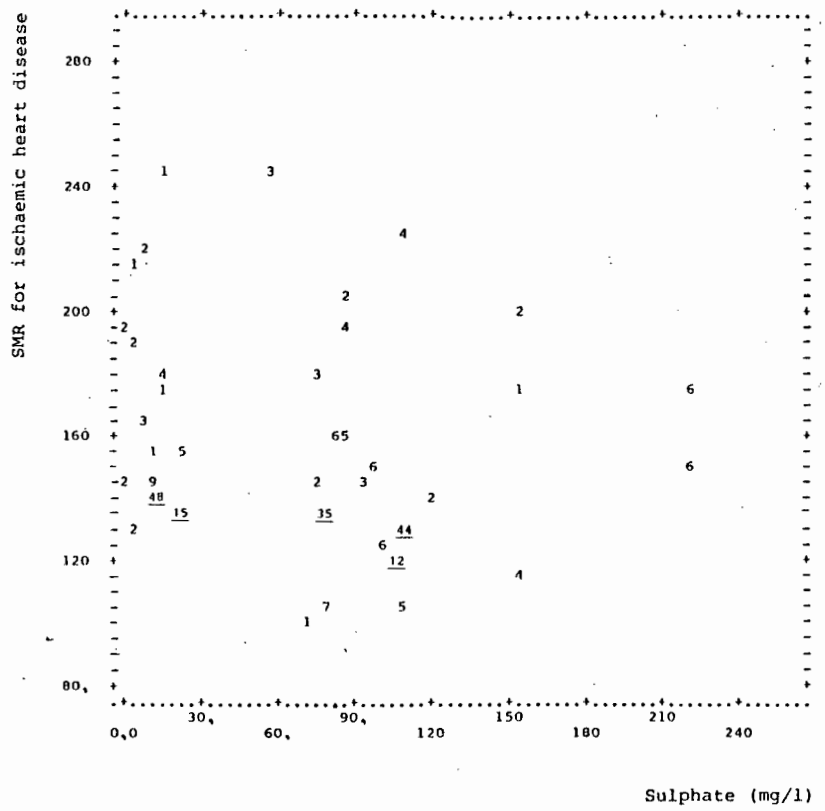

Figure 46:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against sulphate in potable water.
(n=37, weighted)

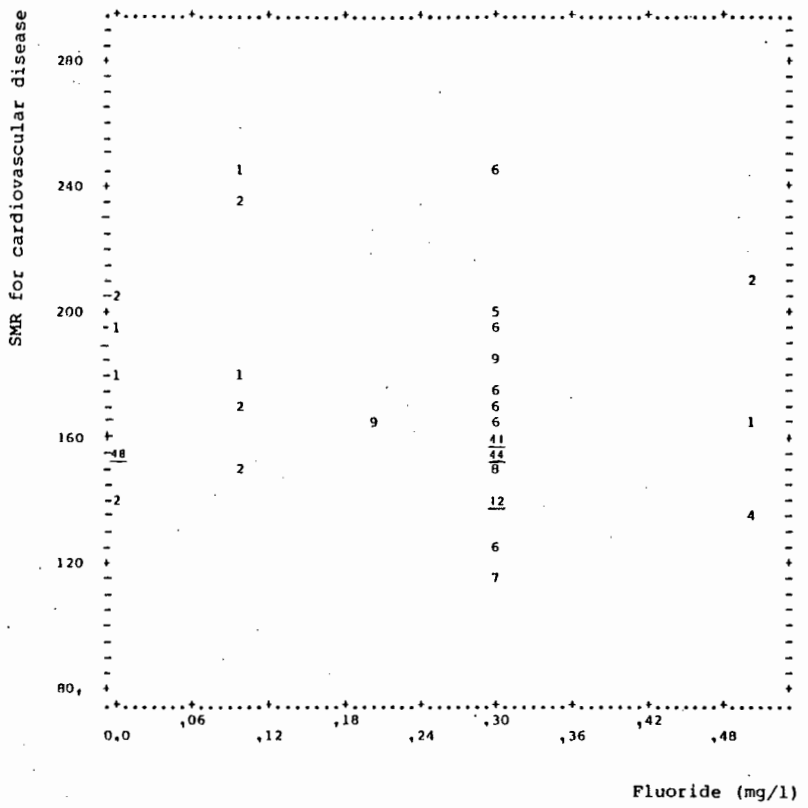

Figure 47:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against fluoride in potable water.
(n=33, weighted)

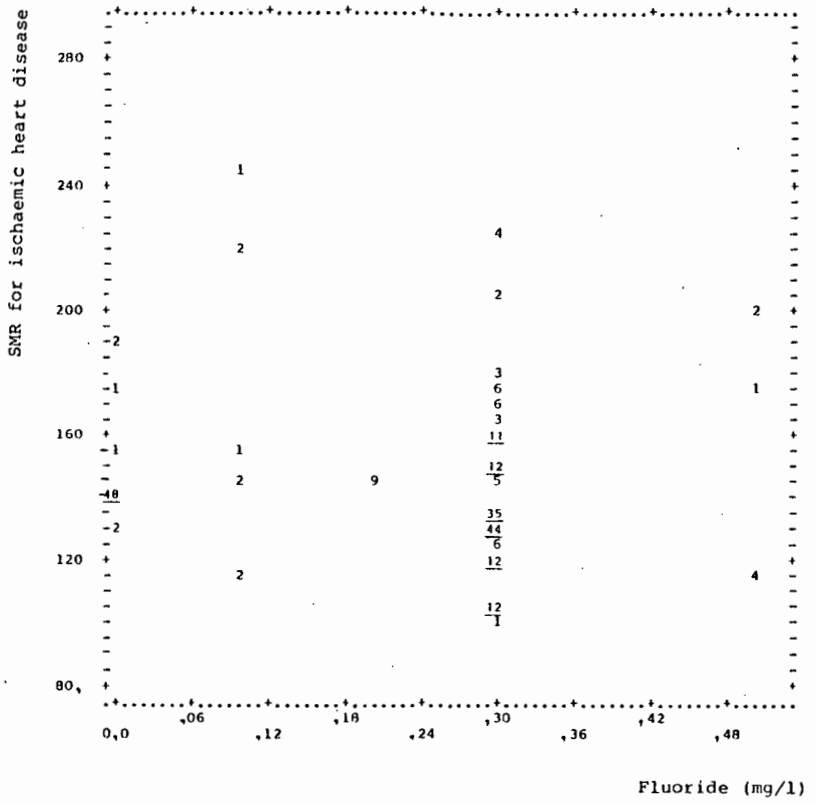

Figure 48:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against fluoride in potable water.
(n=33, weighted)

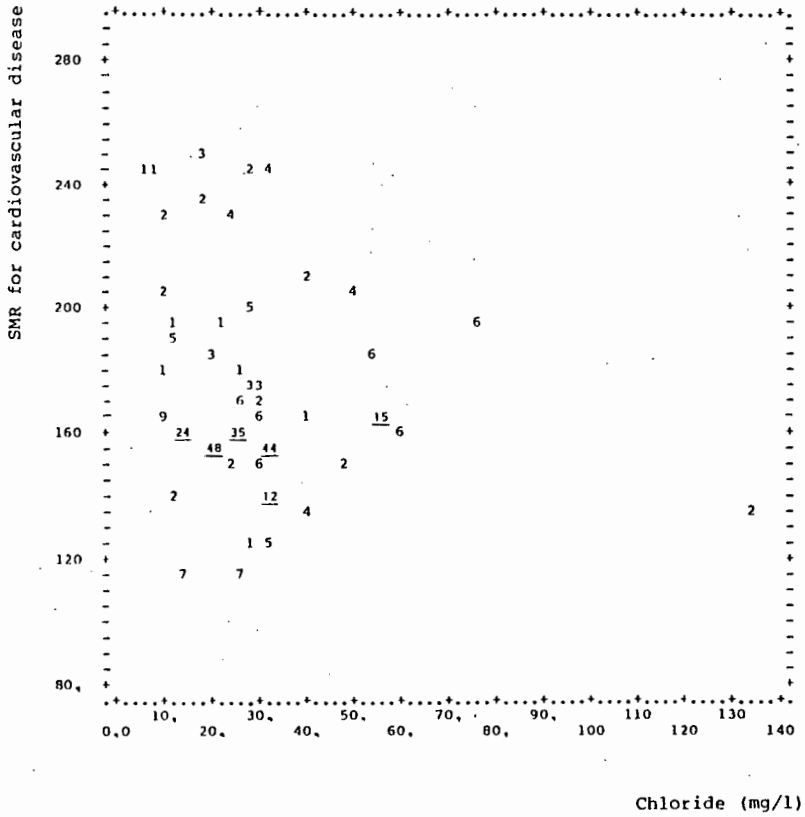

Figure 49:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against chloride in potable water.
(n=43, weighted)

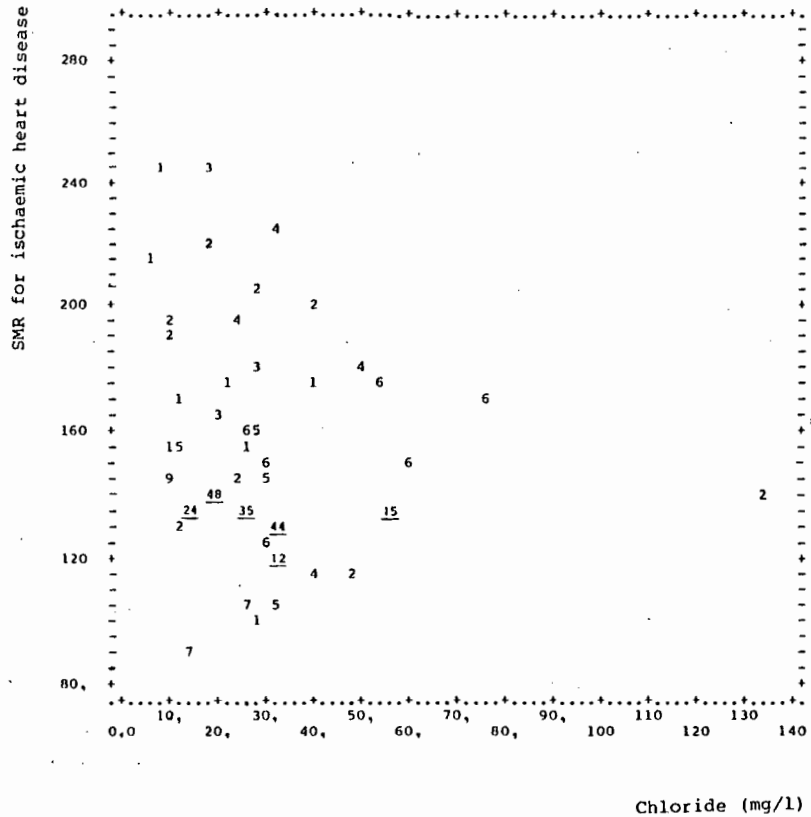

Figure 50:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against chloride in potable water.
(n=43, weighted)

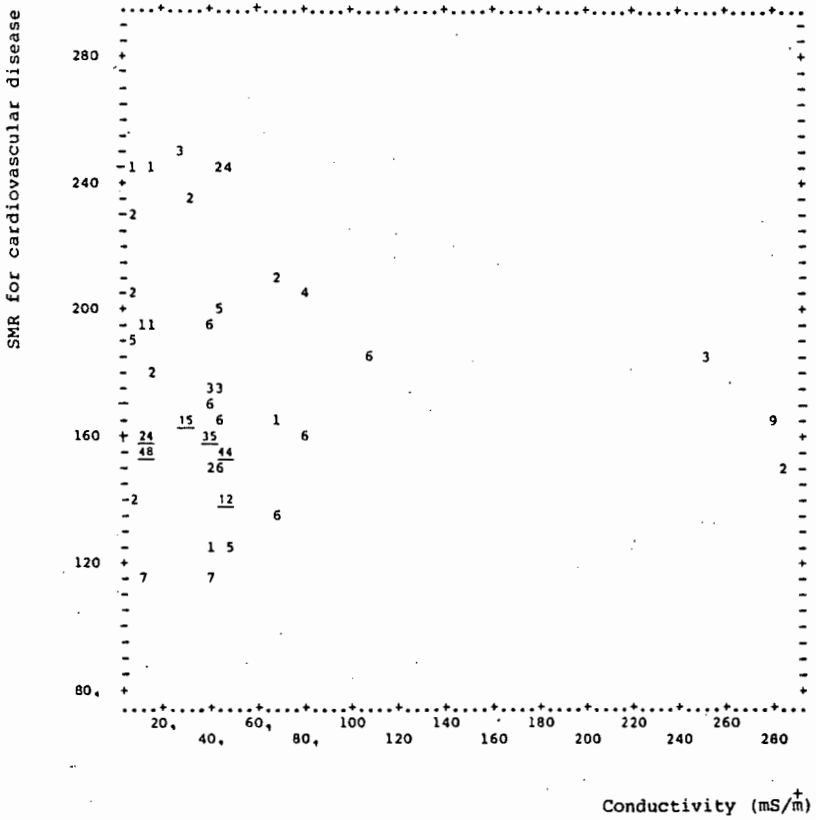

Figure 51:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the conductivity of potable water. (n=42, weighted)

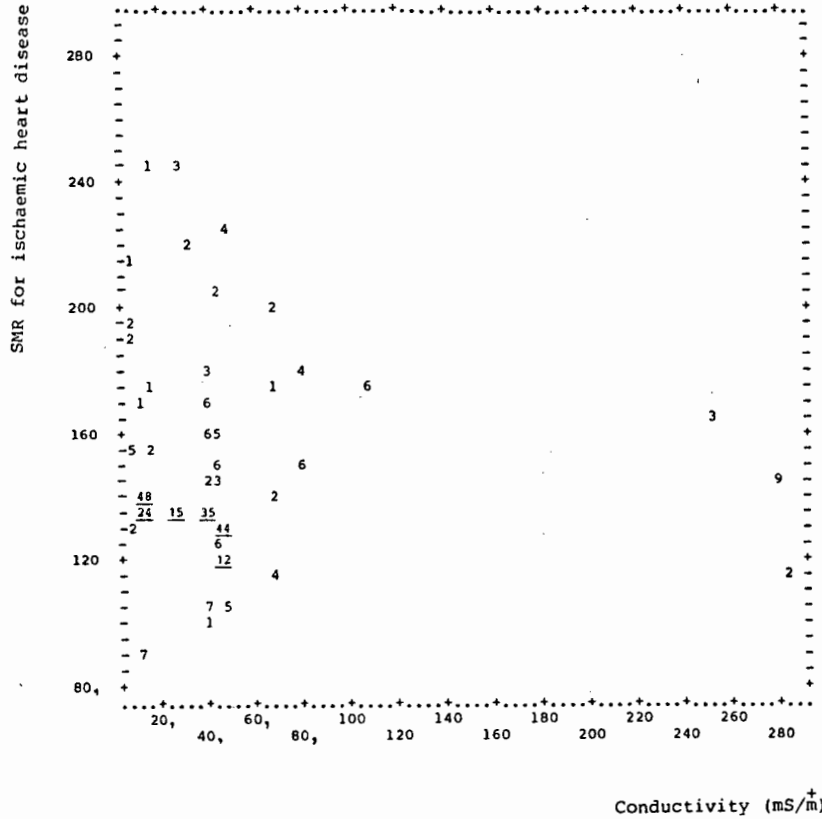

Figure 52:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the conductivity of potable water. (n=42, weighted)

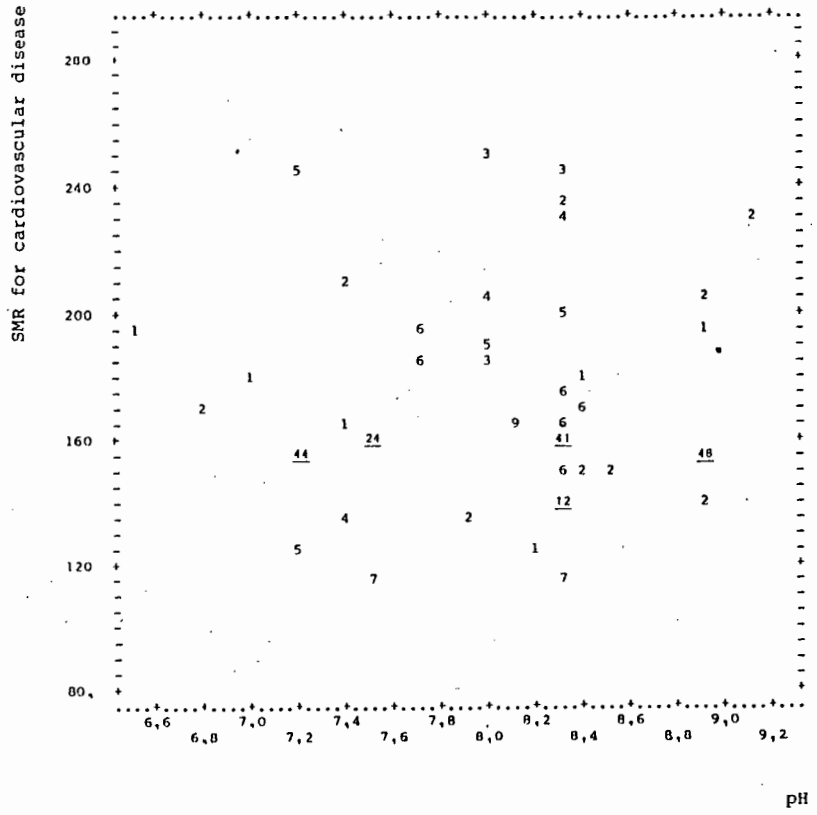

Figure 53:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the pH of potable water.
(n=43, weighted)

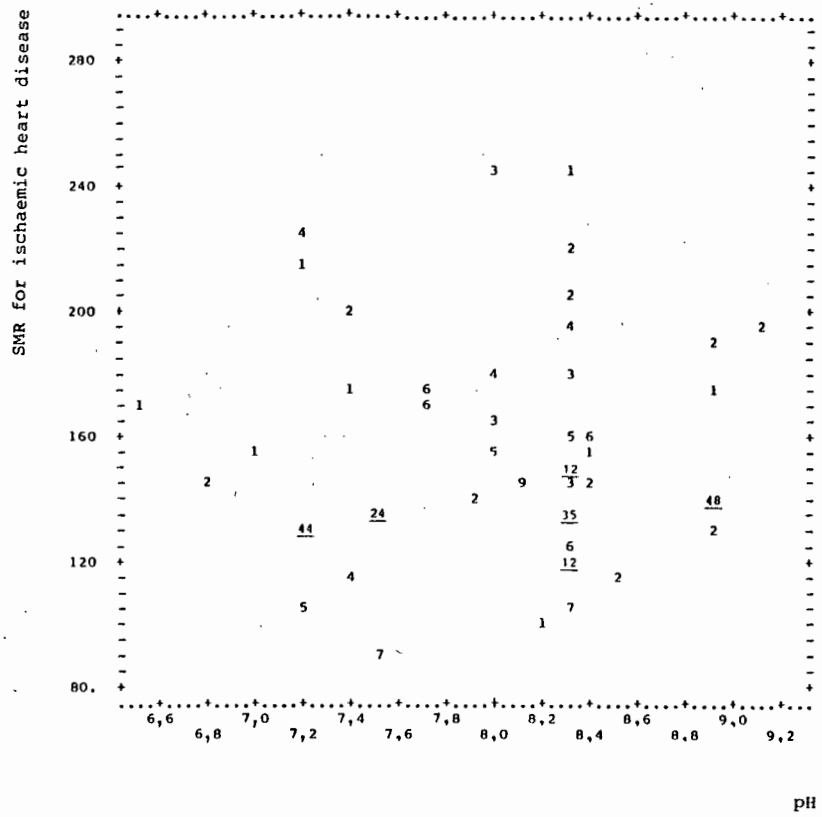

Figure 54:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the pH of potable water.
(n=43, weighted)

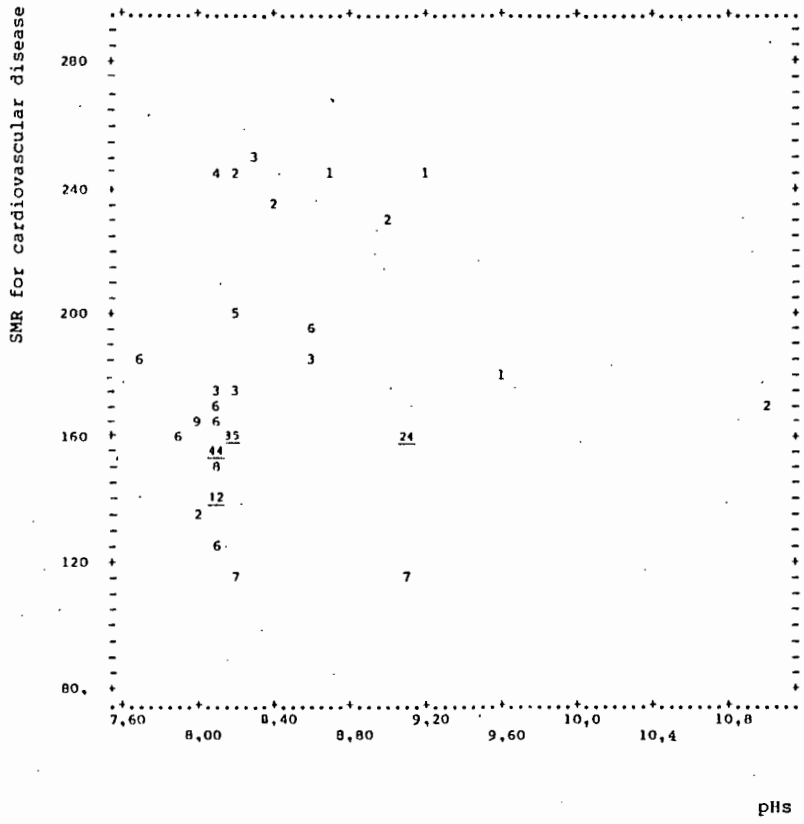

Figure 55:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the saturation pH (pHs) of potable water. (n=30, weighted)

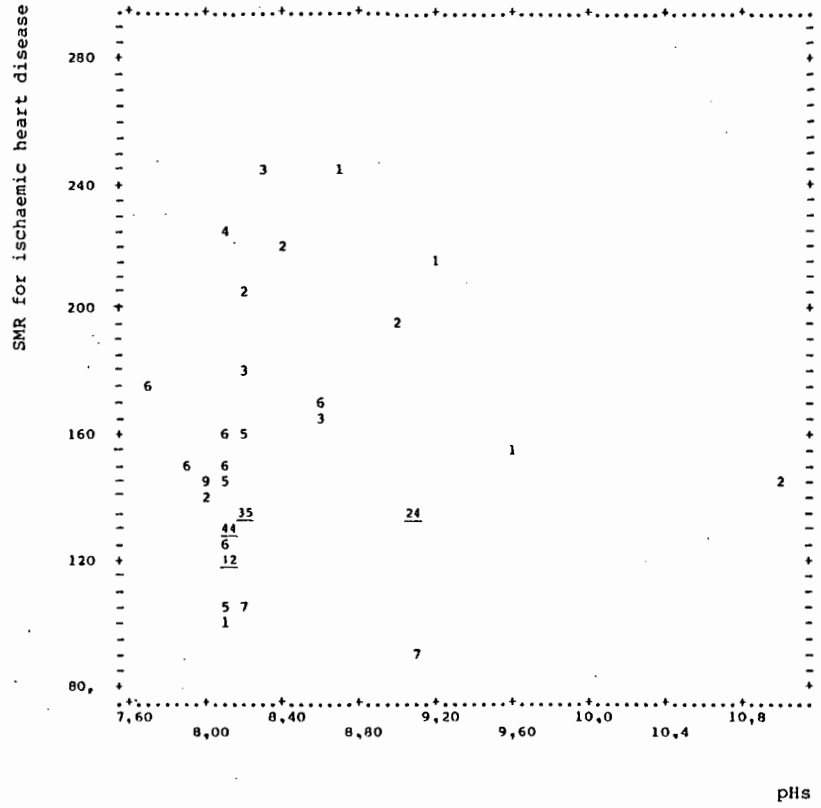

Figure 56:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the saturation pH (pHs) of potable water. (n=30, weighted)

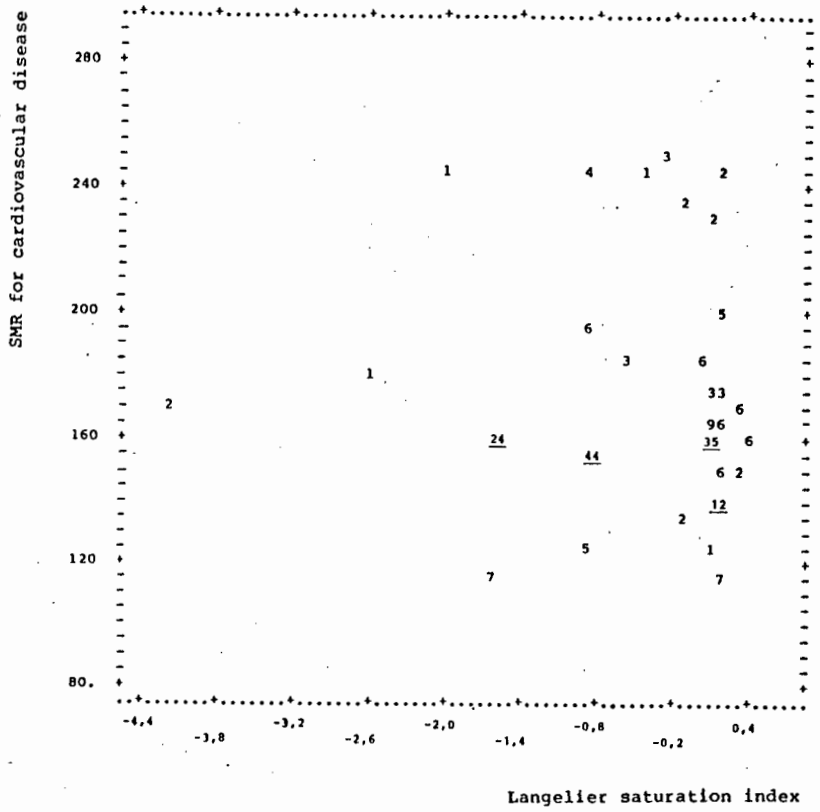

Figure 57:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the Langelier saturation index for potable water. (n=30, weighted)

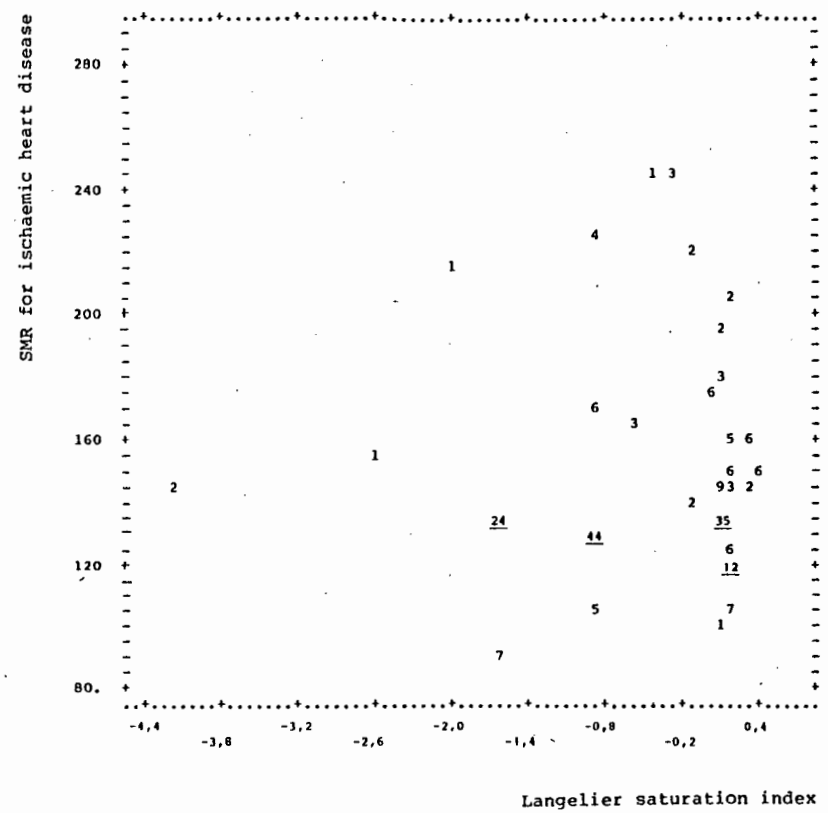

Figure 58:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the Langelier saturation index for potable water. (n=30, weighted)

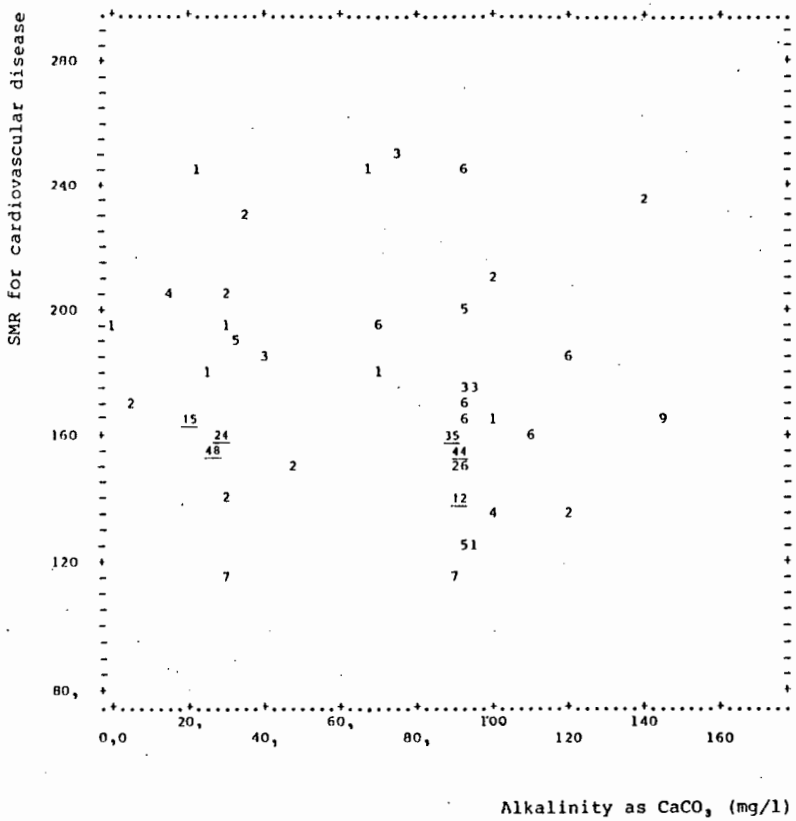

Figure 59:
Standardized mortality ratio for cardiovascular disease (male and female) plotted against the alkalinity of potable water as $CaCO_3$ (n=43, weighted)

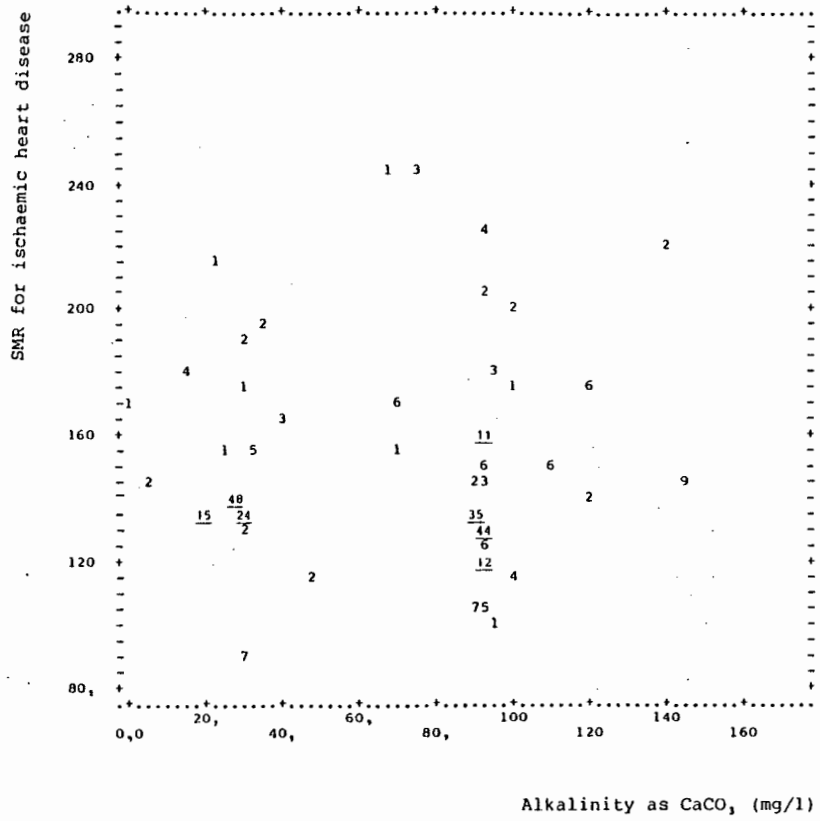

Figure 60:
Standardized mortality ratio for ischaemic heart disease (male and female) plotted against the alkalinity of potable water as $CaCO_3$ (n=43, weighted)