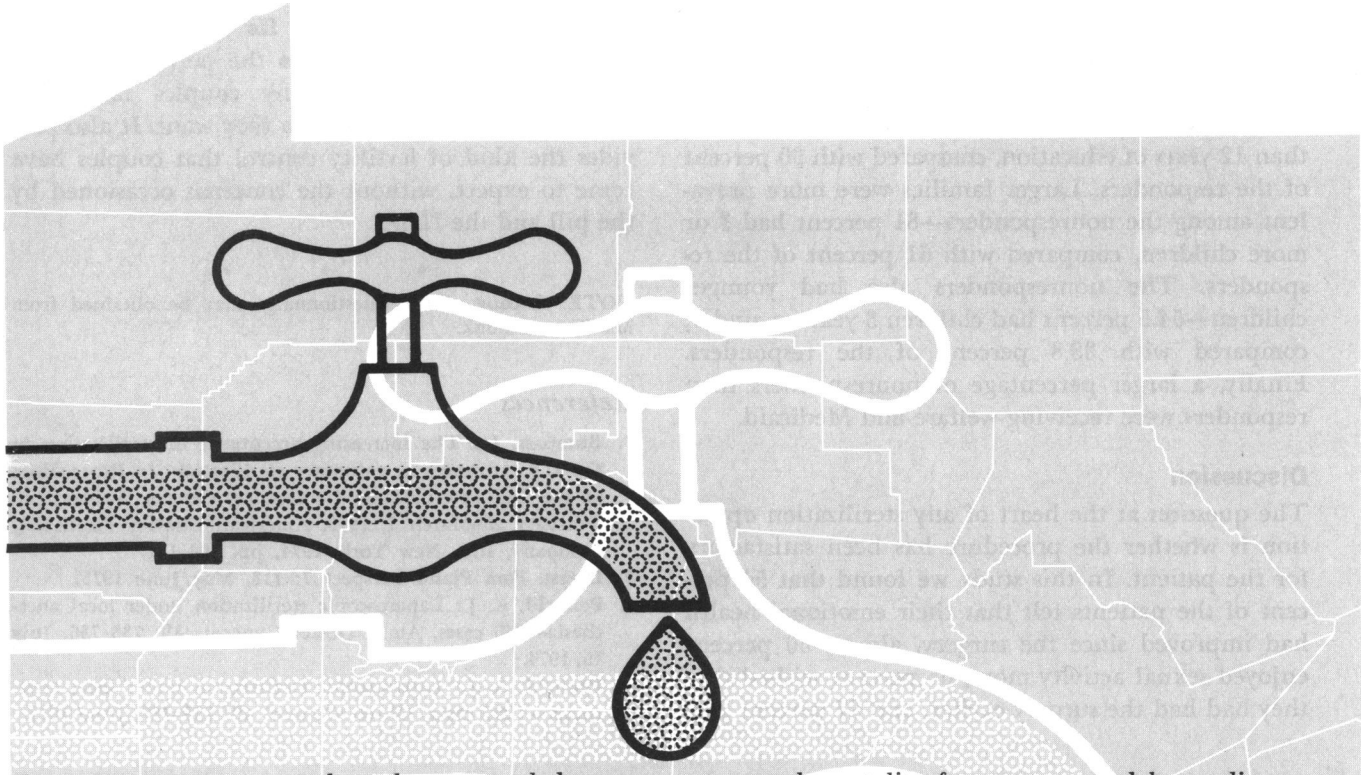
Fluoridation and Mortality— an Epidemiologic Study of Pennsylvania Communities



FEWER HEALTH MEASURES have been accorded more clinical and laboratory research, epidemiologic study, massive clinical trials of total community populations, and public attention (both favorable and adverse) than the fluoridation of public water supplies. As a result, knowledge of the dental and nondental physiological effects of fluoridation has increased significantly since Grand Rapids, Mich., was first experimentally fluoridated in 1945. There is now considerable evidence that fluoridation of community water supplies is both effective and safe. In 1975, the Council on Foods and Nutrition of the American Medical Association updated its earlier statement confirming the efficacy and safety of fluoridation under controlled administration (*1*). Jackson (*2*) provides a list of 22 statements and findings supportive of fluoridation from lay, legal, and professional groups throughout the world.

However, controversy (maintained by a segment of the general public and professional community) continues regarding possible adverse effects of fluoridation on human health. The National Cancer Institute (*3*) and the National Heart and Lung Institute (*4*) have recently issued statements that refute claims suggesting a relationship between fluoridated water and mortality from cancer and heart disease.

A review of the existing literature on fluoridation and mortality indicates that (*a*) the number of epidemiologic studies of the relationship between fluoridation of public water supplies and mortality in the human population is rather limited, (*b*) most of these studies were conducted during the 1940s and 1950s, (*c*) many of the results were based on unadjusted or crude mortality rates, and (*d*) the study designs were often less than adequate or were unclear. Because of the gravity of the controversy, more up-to-date and carefully designed epidemiologic studies based on large human populations are needed. The study described in this paper represents one such effort. Before discussing this study, a brief summary of each of the mortality studies already reported is in order.

Earlier Studies

In 1951, Taylor (*5*) refuted the claim that the incidence of human breast cancer rises according to the fluoride content of the water supply. In the New England States, where public water supplies generally contain practically no fluoride, Taylor found that the average rate of breast cancer was 17 per 100,000 population, whereas in Texas, where the

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water frequently contains up to 5 ppm fluoride, the average rate was only 7 per 100,000. These rates, however, were apparently not adjusted for race, sex, or age.

The Illinois State Department of Health released a special report in 1952 regarding standardized mortality differentials for fluoride and nonfluoride areas (*6*). Mortality statistics for 1940 were analyzed for 22 Illinois cities, 18 having no fluoride in their water supplies and 4 having more than 0.7 ppm. Real differences were found in mortality rates from city to city; however, "classification into fluoride and nonfluoride areas is not a criterion which makes the difference." Mortality from all causes, heart disease, cancer, diabetes, and nephritis was evaluated in the Illinois study. Nevertheless, details of the variations in mortality according to the level of fluoride are not clear.

Hagan and his associates (7) reported in 1954 a study of 32 pairs of cities in different parts of the United States having a population of more than 10,000; each pair consisted of one city with 0.70 or more ppm fluoride and the nearest fluoride city with 0.25 or less ppm fluoride. The populations under study included 892,625 persons in the fluoride cities and 1,297,000 in the nonfluoride cities. The 1950 mortality for these populations from all causes, from heart diseases, from cancer, and from nephritis showed no discernible difference relative to the presence or absence of fluoride in water supplies. Death rates were presumably unadjusted for age, sex, or race.

Knutson *(8)* reported in 1954 on a mortality study of 28 "fluoride cities" (0.7 or higher ppm) and 60 "nonfluoride cities." Age-sex-race-adjusted mortality rates for heart disease, cancer, nephritis, and intracranial lesions for the two groups of cities were com-

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This article is based on a paper presented on October 21, 1976, at the annual meeting of the American Public Health Association (Epidemiology Section), which was held in Miami Beach, Fla. Tearsheet requests to Dr. George K. Tokuhata, Bureau of Health Research, Pennsylvania Department of Health, P.O. Box 90, Rm. 725, Harrisburg, Pa. 17120.

pared. Although heart disease, cancer, and nephritis death rates were slightly lower in the fluoride cities, the death rate for intracranial lesions (cerebrosvascular diseases) was higher. The definition of "nonfluoride" was not given.

The Ontario Department of Health (*9*) reported in 1961 on a study of mortality and fluoride. The mortality rates for all causes, stillbirths, and infant mortality in three Ontario communities where the water supplies contained 1.0 or more ppm fluoride naturally were compared with the rates for 18 cities using "fluoride-free" water. No differences were observed. Whether the mortality rates were adjusted for demographic factors or whether the two areas were otherwise comparable is not known. The actual level of fluoride in the 18 "fluoride-free" cities is also not known.

Objective of Present Study

The major objective of the study we undertook was to determine if there was sufficient epidemiologic evidence to indicate that fluoridation of the public water supplies in Pennsylvania was significantly associated with an increased risk of mortality in general and also with an increased risk of mortality from certain selected chronic diseases. Although a study of the relationship between fluoridation and morbidity would have been more desirable, data on morbidity were not available. Data on mortality can, however, provide indirect evidence of the effects of fluoridation on health, particularly on severe, life-threatening conditions.

Study Method

Four steps were followed in our investigation: (*a*) selection of the communities (fluoridated and unfluoridated) for study, (*b*) determination of the level of fluoride in the drinking water in each of the selected communities, (*c*) computation of appropriate mortality measures for each of the selected communities, and (*d*) establishment of possible relationships between fluoridation (and the level of fluoride) and increased mortality rates.

Selection of communities. Twelve communities with fluoridated water supplies and 12 communities without fluoridation were selected. Except for Philadelphia and Pittsburgh, both fluoridated, an effort was made to have the population sizes of the fluoridated and unfluoridated communities comparable. The names of the communities selected, along with their 1970 census population and the date fluoridation was instituted, are presented in table 1. Easton (fluoridated since 1952) and Pittsburgh (fluoridated since 1952) had the longest history of fluoridation; Harrisburg (fluoridated since 1972) and Lebanon (fluoridated since 1972) had the shortest.

Table 1. Pennsylvania cities and boroughs (fluoridated and unfluoridated) in study

City	County	1970 population	Date fluoridation started
Fluoridated:			
Easton	Northampton	30,256	May 12, 1952
Pittsburgh	Allegheny	520,117	Dec. 19, 1952
Philadelphia	Philadelphia	1,948,609	Sept. 22, 1954
New Kensington	Westmoreland	20,312	Jan. 1, 1958
Lancaster	Lancaster	57,690	Oct. 19, 1960
Williamsport	Lycoming	37,918	Dec. 9, 1966
Chester	Delaware	56,331	Oct. 12, 1968
Reading	Berks	87,643	Oct. 15, 1968
Sharon	Mercer	22,653	Oct. 20, 1970
Bethlehem	Northampton	72,686	June 25, 1971
Harrisburg	Dauphin	68,061	Sept. 15, 1972
Lebanon	Lebanon	28,572	Nov. 27, 1972
Unfluoridated:			
Aliquippa	Beaver	22,277	.....
Allentown	Lehigh	109,527	.....
Altoona	Blair	62,900	.....
Butler	Butler	18,691	.....
Erie	Erie	129,231	.....
Hazleton	Luzerne	30,426	.....
Johnstown	Cambria	42,476	.....
Norristown	Montgomery	38,169	.....
Pottsville	Schuylkill	19,715	.....
Scranton	Lackawanna	103,564	.....
Wilkes-Barre	Luzerne	58,856	.....
York	York	50,335	.....

Determination of fluoride levels. Data on the level of fluoride in public water supplies were provided by the Bureau of Water Quality Management of the Pennsylvania Department of Environmental Resources. While the data for fluoridated communities covered, in general, the period from 1966 through 1975, only the measures for 1966–71 were used because of the limitations of the demographic data for the 1972–75 period.

The level of natural fluoride in the water supply of unfluoridated communities is not measured routinely, but some 1974 data were available for each of the 12 unfluoridated communities in our study. These data were used on the assumption that the level of natural fluoride tends to be relatively stable from year to year.

Computation of mortality rates. Since mortality rates are highly sensitive to the age-sex-race composition of a population, these three factors were simul-

taneously taken into account in computing adjusted mortality rates. Using the age-sex-race distribution of Pennsylvania's population enumerated in 1960 as the standard population, we applied the direct method of adjustment. Three-year-average adjusted death rates for 1969–71 were used for group comparisons (fluoridated communities versus unfluoridated) as well as for correlational analyses. Adjusted death rates for diabetes mellitus and cerebrovascular diseases for the 1959–61 period were also calculated, so that decennial changes in mortality from these causes could be examined.

For analyses of secular mortality trends, the unadjusted or crude average mortality rates were calculated for each of the 12 fluoridated communities for a series of 5-year intervals beginning 5 years before the year of fluoridation and ending in 1974. Adjusted rates for this analysis could not be computed because the necessary demographic data were lacking.

Establishment of relationships between fluoridation and mortality. We used three different methods to establish possible relationships between fluoridation (and the level of fluoride) and mortality rates: group analyses, correlational analyses, and analyses of secular mortality trends.

1. Group analyses. The average mortality rates of fluoridated and unfluoridated communities were compared. Comparisons were also made of communities grouped according to the level of fluoride in their water supplies. The statistical methods used in the tests of significance are based on the work of Bailar and Ederer (*10*), who calculated significance factors for the ratio of an observed value of a Poisson variable to its expectation. The basic assumption underlying this method is that deaths follow a Poisson distribution.

2. Correlational analyses. Each community was considered an independent unit of observation regardless of the level of fluoride or of the mortality.

3. Analyses of secular mortality trends. Each community "served as its own control;" that is, the level of mortality from selected causes was examined in a historical context to see if the rate changed in a consistent and significant pattern. The pattern of changes observed among fluoridated communities was then compared with that observed among unfluoridated communities during the same period.

Results

Flouridated communities versus unfluoridated. Age-sex-race-adjusted mortality rates were computed for all causes, cerebrovascular diseases, and diabetes mellitus. Eight fluoridated and 12 unfluoridated communities were included in this analysis. Four communities now fluoridated were not included either because they had not been fluoridated during the 1969–71 period or because fluoridation had been initiated during that period.

A comparison of the 1969–71 mortality rates for the fluoridated and unflouridated municipalities is presented in table 2. The mortality for all causes for the fluoridated group was 1,093.9 per 100,000 population as compared with 1,136.4 for the unfluoridated group. This difference was statistically significant ($P<0.01$). The cerebrovascular disease mortality rates of 152.8 for the fluoridated group and 156.5 for the unfluoridated were not significantly different. The diabetes mortality rate was lower (37.0) for the fluoridated areas than for the unfluoridated (45.2); this difference was statistically significant ($P<0.01$). Thus, the adjusted mortality rates for all causes and for cerebrovascular diseases and diabetes mellitus all tended to be higher in the unfluoridated areas than in the fluoridated.

Communities with different levels of fluoride. Since fluoride levels varied within the fluoridated group as well as within the unfluoridated, further analysis was carried out in which the fluoride level was considered. The 20 communities studied, 8 fluoridated and 12 unfluoridated, were grouped into 4 classes according to 4 selected levels of fluoride in their public water supplies: less than 0.20 ppm, 0.20–0.49 ppm, 0.50–0.99 ppm, and 1.00 or more ppm.

Table 2. Average fluoride levels and selected age-sex-race-adjusted mortality rates for fluoridated and unfluoridated municipalities, 1969–71

Variables	All municipalities in study	Municipalities fluoridated before 1969	Unfluoridated municipalities
Number of municipalities ..	20	8	12
1970 population	3,445,043	2,758,876	686,167
Average fluoride level (ppm)	0.46	[1] 0.96	[2] 0.12
Adjusted death rates per 100,000 population, 1969–71: [3]			
All causes	1,119.4	1,093.9	1,136.4
Cerebrovascular diseases	155.0	152.8	156.5
Diabetes mellitus	41.9	37.0	45.2

[1] Based on all data available through 1971.
[2] Represents average fluoride level in 1974. Data for prior years not available, but levels assumed to be relatively constant.
[3] Computed by direct method, with age-sex-race distribution of 1960 Pennsylvania population as standard.

As shown in table 3, all 12 unfluoridated communities fell into the less than 0.20 ppm class. No communities were in the 0.20–0.49 ppm class. Five of the eight fluoridated communities fell into the 0.50–0.99 ppm class, whereas the remaining three fell into the 1.00 or more ppm class.

The age-sex-race-adjusted 3-year-average mortality rates for selected causes were computed for each of the three classes of communities with different fluoride levels. A general pattern was observed in the mortality for all causes and for diabetes; that is, that the lower the level of fluoride, the higher the level of mortality. Specifically, the mortality rates for all causes and for diabetes for communities with less than 0.20 ppm fluoride were significantly higher ($P<0.01$) than those for communities with 0.50–0.99 ppm or 1.00 or more ppm fluoride. In contrast, neither the mortality rates for all causes nor for diabetes were significantly different when the 0.50–0.99 ppm and 1.00 or more ppm clases were compared separately.

The cerebrovascular disease mortality presented a somewhat different pattern. The level of mortality was significantly higher in the two extreme fluoride classes, less than 0.20 ppm ($P<0.05$) and 1.00 or more ppm ($P<0.01$), than in the 0.50–0.99 class. The slight difference noted between the two extreme fluoride categories was not statistically significant.

The results of these mortality analyses, especially with respect to cerebrovascular disease mortality, are extremely difficult to interpret. There are no apparent reasons why the pattern of cerebrovascular mortality according to the level of fluoride should be different from that of mortality from all causes and for diabetes. Since these mortality rates were adjusted for sex, race, and age, the observed significant differences were not due to these demographic variables. However, the differences observed may be due to other factors that have not been taken into account, such as environmental hazards or the socioeconomic characteristics of the local communities.

Correlational analysis. The age-sex-race-adjusted mortality rates for all causes, cerebrovascular diseases, and diabetes mellitus were correlated with the average fluoride levels in the water supplies of all 20 communities in our study, of the 8 fluoridated communities, and of the 12 unfluoridated communities. The results of this analysis are summarized in table 4. The data indicate that the level of fluoride was inversely correlated with the level of mortality for all causes in all three groups. This inverse correlation was statistically significant only when the group of 12 unfluoridated communities was considered separately; that is, it was significant for the group of communities in which the natural fluoride levels were below 0.20 ppm and the fluctuations from one community to another were slight.

The overall correlation between the level of fluoride and the level of cerebrovascular disease mortality among the 20 communities was negative and negligible in degree. In contrast, there was a positive, but not statistically significant, correlation between fluoride levels and cerebrovascular mortality rates in the eight fluoridated communities.

Table 3. Average age-sex-race-adjusted mortality rates for selected fluoridated and unfluoridated Pennsylvania cities and boroughs, 1969–71, by fluoride level

Average fluoride level (ppm) [1]	*Cities and boroughs*	*Number of communities*	*1970 population*	*Adjusted rates per 100,000 population* [2]		
				All causes	*Cerebrovascular disease*	*Diabetes mellitus*
Less than 0.20	Aliquippa, Allentown, Altoona, Butler, Erie, Hazleton, Johnstown, Norristown, Pottsville, Scranton, Wilkes-Barre, and York	12	686,167	1,136.4	156.5	45.2
0.20–0.49						
0.50–0.99	Philadelphia, Lancaster, Williamsport, Chester and Reading	5	2,188,191	1,096.0	145.8	37.3
1.00 or more	Easton, Pittsburgh and New Kensington	3	570,685	1,090.4	164.4	36.4

[1] Average fluoride levels for fluoridated cities and boroughs based on all data available through 1971. Fluoride levels for unfluoridated municipalities represent average fluoride level in 1974. Data for prior years not routinely available.

[2] Computed by direct method, with age-sex-race distribution of 1960 Pennsylvania population as standard.

Table 4. Correlation between average fluoride levels and selected age-sex-race-adjusted mortality rates for fluoridated and unfluoridated municipalities, 1969–71

Study areas	Correlation coefficient		
	Fluoride levels and death rates for all causes	Fluoride levels and cerebrovascular disease death rates	Fluoride levels and diabetes mellitus death rates
All municipalities ($N=20$)	−0.233	−0.058	−0.329
Municipalities fluoridated before 1969 ($N=8$)	−0.374	+0.348	−0.250
Unfluoridated municipalities ($N=12$).	[1] −0.592	−0.030	−0.309

[1] Significant at 5 percent level.
NOTE: Cities and boroughs fluoridated in 1970, 1971, and 1972 not included in analysis because fluoride levels for 1971 and prior years could not be determined.

The diabetes mortality rates were inversely correlated with the levels of fluoride in both the fluoridated and the unfluoridated communities. These correlations were not statistically significant.

Analysis of secular trends. Secular trends in mortality for all 12 fluoridated communities were analyzed. The average 5-year death rates for all causes, heart disease, diabetes mellitus, and cardiovascular diseases were computed for each of the 12 communities for the 5-year period preceding fluoridation and for each of the consecutive 5-year periods following fluoridation, with each community serving as its own control. The results of this analysis are presented in table 5.

As can be seen from the data in table 5, the mortality rates for all causes fluctuated randomly, with no clear or consistent pattern of changes from prefluoridation periods to postfluoridation periods. When the prefluoridation 5-year periods were compared with the 5-year periods immediately following fluoridation, the mortality rate for all causes increased in five communities but decreased in the remaining seven. When the prefluoridation periods were compared with the most recent 5-year periods, the pattern remained the same.

The data on heart disease mortality, like the data on mortality for all causes, failed to indicate a clear or consistent pattern of changes from the prefluoridation to the postfluoridation periods. A comparison of the prefluoridation periods with the 5-year periods immediately following fluoridation showed increases in the heart disease death rate in seven communities and decreases in the remaining five. When the prefluoridation periods were compared with the most recent periods ,the heart disease death rate increased in five communities, but decreased in seven.

In only one community was the diabetes death rate higher for the 5-year period immediately following fluoridation than for the period preceding it. The rates for the other 11 communities decreased. When compared with the most recent periods, the diabetes mortality rate increased in three communities and decreased in nine.

As was the case with the other diseases considered, no clear or consistent pattern of changes in cerebrovascular disease mortality was observed. When the prefluoridation 5-year periods were compared with the 5-year periods immediately following fluoridation, the average cerebrovascular disease death rate increased in six communities and decreased in six. When the prefluoridation periods were compared with the most recent intervals, the rate was seen to increase in seven communities and decrease in five.

Since these 5-year-average mortality rates were not adjusted for age, sex, and race, the observed historical changes may have been influenced by these demographic factors or by other factors such as the socioeconomic characteristics (including medical care) of the local population.

When only prefluoridation periods were considered, certain cross-sectional variations were noted among the 12 communities. These differences may have been due to (*a*) possible differences in population characteristics, (*b*) known differences in the historical periods for which the rates were computed (since the years in which fluoridation was initiated were different), and (*c*) recognized bias in the death reporting system (for example, due to improper allocation of decedent's residence). These considerations are also equally applicable when postfluoridation periods are compared.

Decennial changes in death rates—fluoridated versus unfluoridated communities. Thus far we have reported only our analyses of secular mortality trends for the fluoridated communities. Decennial changes in the death rates for diabetes mellitus and cerebrovascular diseases, however, were also analyzed and the results for fluoridated communities and unfluoridated communities compared (table 6).

To compute age-sex-race-adjusted death rates for individual communities more accurately, it was necessary to use the 1960 and 1970 census populations as denominators while employing the 1959–61 and 1969–71 3-year-average deaths as numerators. Because of this time constraint, four communities in

Table 5. Average annual death rates per 100,000 population for all causes, heart diseases, diabetes mellitus, and cerebrovascular diseases for selected Pennsylvania cities and boroughs, before and after fluoridation

City or borough, with year fluoridated	5 years before fluoridation	Year of fluoridation	5 years after fluoridation	6 to 10 years after fluoridation	11 to 15 years after fluoridation	16 to 20 years after fluoridation
			Death rates for all causes			
Easton, 1952	1,388.7	1,475.8	1,506.2	1,400.5	1,444.1	1,428.2
Pittsburgh, 1952	1,196.3	1,278.7	1,273.4	1,328.3	1,375.4	1,389.2
Philadelphia, 1954	1,175.3	1,151.8	1,210.8	1,253.3	1,273.8	1,230.1
New Kensington, 1958	1,084.9	1,079.1	1,109.9	1,218.6	1,286.3	.....
Lancaster, 1960	1,398.7	1,454.4	1,360.6	1,323.1	[1] 1,284.5	.....
Williamsport, 1966	1,379.1	1,464.4	1,333.0	[2] 1,240.5	.....	.....
Chester, 1968	1,338.1	1,394.6	1,299.3	[3]	.....	.....
Reading, 1968	1,542.1	1,702.5	1,516.5	[3]	.....	.....
Sharon, 1970	1,360.2	1,275.8	[1] 1,285.6	.....	.....	.....
Bethlehem, 1971	1,033.7	1,041.5	[2] 1,079.5	.....	.....	.....
Harrisburg, 1972	1,585.1	1,525.2	[4] 1,435.7	.....	.....	.....
Lebanon, 1972	1,574.7	1,150.9	[4] 1,071.8	.....	.....	.....
			Heart disease death rates			
Easton, 1952	557.9	679.1	715.5	685.3	670.6	609.6
Pittsburgh, 1952	445.3	517.6	494.1	537.6	575.0	604.1
Philadelphia, 1954	476.6	467.6	511.3	511.8	465.2	434.4
New Kensington, 1958	418.7	415.7	492.2	544.0	478.9	.....
Lancaster, 1960	572.8	637.1	600.9	541.8	[1] 536.6	.....
Williamsport, 1966	612.0	657.6	597.1	[2] 557.1	.....	.....
Chester, 1968	564.7	638.5	541.7	[3]	.....	.....
Reading, 1968	695.3	717.5	673.8	[3]	.....	.....
Sharon, 1970	543.6	569.5	[1] 562.4	.....	.....	.....
Bethlehem, 1971	465.4	470.3	[2] 503.7	.....	.....	.....
Harrisburg, 1972	663.4	603.9	[4] 573.2	.....	.....	.....
Lebanon, 1972	511.2	536.8	[4] 499.1	.....	.....	.....
			Diabetes death rates			
Easton, 1952	39.1	14.3	26.0	28.1	32.8	29.7
Pittsburgh, 1952	24.9	23.6	24.1	29.0	29.5	27.0
Philadelphia, 1954	25.5	26.6	23.0	27.5	28.5	24.9
New Kensington, 1958	30.4	33.6	18.2	17.6	31.9	.....
Lancaster, 1960	36.2	45.9	35.3	33.2	[1] 29.7	.....
Williamsport, 1966	26.0	25.3	23.0	[2] 13.4	.....	.....
Chester, 1968	33.3	36.3	32.7	[3]	.....	.....
Reading, 1968	32.1	33.4	36.4	[3]	.....	.....
Sharon, 1970	49.5	39.7	[1] 47.7	.....	.....	.....
Bethlehem, 1971	22.9	30.4	[2] 21.5	.....	.....	.....
Harrisburg, 1972	34.4	28.2	[4] 33.1	.....	.....	.....
Lebanon, 1972	32.0	38.6	[4] 27.4	.....	.....	.....
			Cerebrovascular disease death rates			
Easton, 1952	139.3	131.8	154.5	152.8	153.0	141.7
Pittsburgh, 1952	117.9	133.5	141.1	145.6	136.4	132.6
Philadelphia, 1954	100.7	106.7	112.0	104.4	102.5	102.2
New Kensington, 1958	129.1	117.6	118.5	125.1	141.3	.....
Lancaster, 1960	183.6	155.6	147.9	150.4	[1] 145.7	.....
Williamsport, 1966	127.1	126.5	137.8	[2] 129.9	.....	.....
Chester, 1968	91.7	67.5	94.6	[3]	.....	.....
Reading, 1968	176.7	200.6	163.2	[3]	.....	.....
Sharon, 1970	133.1	97.1	[1] 126.5	.....	.....	.....
Bethlehem, 1971	93.7	96.8	[2] 109.7	.....	.....	.....
Harrisburg, 1972	149.1	133.5	[4] 145.5	.....	.....	.....
Lebanon, 1972	105.0	112.3	[4] 102.6	.....	.....	.....

[1] Data available only for 4-year interval.
[2] Data available only for 3-year interval.
[3] Data available only for single year, so not included.
[4] Data available only for 2-year interval.

NOTE: Leaders (...) indicate not applicable.

which fluoridation began after 1970 were not included in this particular analysis.

The diabetes mellitus death rate increased in three of the eight fluoridated communities, but decreased in the remaining five. For the eight fluoridated communities considered together, the average diabetes death rate changed from 37.8 per 100,000 in 1960 to 37.0 per 100,000 in 1970, a decline of 0.8 per 100,000 during the decade. In the 12 unfluoridated communities, the diabetes death rate increased in 5 communities, but decreased in the remaining 7. For the 12 unfluoridated communities considered together, the average diabetes death rate changed from 44.9 per 100,000 in 1960 to 45.2 per 100,000 in 1970, a gain of 0.3 per 100,000 during the decade. These observations indicate that there were no significant differences between the fluoridated and unfluoridated communities in the secular trend in diabetes mortality.

The cerebrovascular disease mortality declined in all eight fluoridated communities during the 1960–70 decade. For the fluoridated communities as a whole, the rate changed from 189.6 per 100,000 to 152.8 per 100,000, a decline of 36.8 per 100,000 during the decade. Within the group of 12 unfluoridated communities, the cerebrovascular disease mortality increased in 1 community but declined in the remaining 11 during the 1960–70 decade. For the entire group of unfluoridated communities under study, the rate changed from 196.5 per 100,000 to 156.5 per 100,000, a decline of 40.0 per 100,000 during the decade. Again, these results indicated that there were no significant differences between fluoridated and unfluoridated communities in the secular trend in cerebrovascular disease mortality.

Conclusions and Comment

Based on the data and analyses we have presented, the following general conclusions may be drawn:

1. There is no clear evidence that fluoridation under controlled administration adversely affects overall mortality or diabetes mortality. Fluoridation

Table 6. Age-sex-race-adjusted diabetes and cerebrovascular disease death rates per 100,000 population for selected fluoridated and unfluoridated cities and boroughs in Pennsylvania, 1959–61 and 1969–71

City or borough	Diabetes			Cerebrovascular disease		
	1959–61	1969–71	Difference	1959–61	1969–71	Difference
Fluoridated, with year started: [1]						
Easton, 1952	32.7	39.4	+ 6.7	176.7	166.1	− 10.6
Pittsburgh, 1952	39.0	30.0	− 9.0	198.0	147.3	− 50.7
Philadelphia, 1954	33.8	31.7	− 2.1	156.8	122.5	− 34.3
New Kensington, 1958	24.9	39.9	+15.0	189.5	179.7	− 9.8
Lancaster, 1960	49.4	42.8	− 6.6	229.9	176.7	− 53.2
Williamsport, 1966	37.7	25.3	−12.4	187.5	145.2	− 42.3
Chester, 1968	51.6	48.4	− 3.2	161.4	127.2	− 34.2
Reading, 1968	33.1	38.2	+ 5.1	217.3	157.5	− 59.8
Area average	37.8	37.0	− 0.8	189.6	152.8	− 36.8
Unfluoridated:						
Aliquippa	27.0	41.4	+14.4	262.8	161.9	−100.9
Allentown	40.4	30.4	−10.0	145.1	143.8	− 1.3
Altoona	60.4	39.0	−21.4	198.1	167.9	− 30.2
Butler	32.0	36.8	+ 4.8	198.6	143.2	− 55.4
Erie	35.3	27.3	− 8.0	222.8	162.9	− 59.9
Hazleton	53.1	50.0	− 3.1	175.4	123.9	− 51.5
Johnstown	63.1	55.5	− 7.6	243.8	177.7	− 66.1
Norristown	27.2	59.7	+32.5	165.6	193.0	+ 27.4
Pottsville	49.0	84.5	+35.5	208.6	162.6	− 46.0
Scranton	36.8	43.5	+ 6.7	191.2	160.8	− 30.4
Wilkes-Barre	77.0	42.4	−34.6	150.6	144.0	− 6.6
York	36.9	31.4	− 5.5	194.9	136.8	− 58.1
Area average	44.9	45.2	+ 0.3	196.5	156.5	− 40.0

[1] 4 communities fluoridated since 1970 not included in analysis: Sharon, Bethlehem, Harrisburg, and Lebanon.

NOTE: Age-sex-race distribution of 1960 Pennsylvania population used as standard for adjusted rates.

under controlled administration may have no significant effect on mortality for these causes.

2. The inconsistent relationships found between high levels of fluoride and cerebrovascular disease mortality should be investigated further.

In interpreting the results and conclusions in this report, one needs to be aware that only mortality data, not data on morbidity, were made available and, also, that not all of the factors that might influence mortality could be taken into account. Most of the mortality rates used were adjusted for age, sex, and race. It would be premature, however, to draw direct conclusions about causes and effects from these results alone.

Certain questions remain unanswered, for example, the threshold dosage of fluoride for disease-specific pathogenesis (if any) or for protective impact (if any) and the importance and delineation of exposure time for possible harmful as well as protective influences.

These and other important questions must be answered in future studies. The results and conclusions presented here also need to be substantiated, because they have significant public health implications both for the general public and the scientific community.

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SYNOPSIS

TOKUHATA, GEORGE K. (Pennsylvania Department of Health), DIGON, EDWARD, and RAMASWAMY, KRISHNAN: *Fluoridation and mortality—an epidemiologic study of Pennsylvania communities. Public Health Reports, Vol. 93, January-February 1978, pp. 60–68.*

To evaluate the possible health impact of the fluoridation of public water supplies, a large epidemiologic study based on mortality data was conducted in Pennsylvania. The study covered 24 major communities (12 with fluoridated water supplies and 12 without fluoridation), having a total population of approximately 3.5 million. Cross-sectional and historical patterns, as well as group and individual community differences, were analyzed.

When the fluoridated and unfluoridated populations were compared as two groups, irrespective of the actual level of fluoride, mortality for all causes and for diabetes was significantly higher in the unfluoridated population than in the fluoridated. Cerebrovascular disease mortality for the two populations did not differ. When the actual level of flouride was considered, some additional, but inconsistent and difficult-to-explain, relationships emerged between fluoride and mortality. Specifically, mortality for all causes and for diabetes was significantly higher for those communities with less than 0.20 ppm fluoride than for communities with 0.50-0.99 ppm or with more than 1.00 ppm. In contrast, cerebrovascular disease mortality was significantly higher in the two extreme fluoride classes than in the 0.50–0.99 ppm class.

When individual communities were considered as separate units, there was a general inverse correlation ($r = -0.233$) between fluoride levels and mortality rates. This general relationship was statistically significant only for mortality for all causes considered together in those unfluoridated communities whose fluoride levels were less than 0.20 ppm ($r = -0.592$). An exception to this general relationship was the positive correlation ($r = +0.348$) that was found between cerebrovascular disease mortality rates and fluoride levels in the fluoridated communities.

The pattern of secular changes in mortality from prefluoridation periods to postfluoridation periods was similar for both fluoridated and unfluoridated populations.

No clear evidence was found to suggest that fluoridation under controlled administration adversely affects overall mortality or diabetes mortality. Under controlled administration, fluoridation may have no significant effect on either mortality rate. The inconsistent relationships found between high levels of fluoride and cerebrovascular disease mortality require further investigation.