# Effects of Prenatal Exposure to Fluoridation on Dental Caries

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THE CARIES-INHIBITING effect produced in children who drink water with optimal levels of fluoride has been well documented by numerous clinical investigations (*1–3*). Moreover, there is general agreement that waterborne fluorides afford greatest protection against decay when exposure begins during the period of tooth development and calcification. There is some disagreement, however, as to when during the period of tooth formation exposure must begin in order to produce maximum benefits.

Some data suggest that ultimate inhibition of caries occurs only when teeth receive fluoride during the entire calcification period, and that generally a steady decline in protection is related to the time when the developing tooth is initially exposed to fluoride (*3–5*). Other findings indicate that maximum benefits can be attained if the consumption of fluoride is started during the last stage of tooth calcification (pre-eruptive maturation) (*6–8*). Backer Dirks and co-authors (*9*) have taken an intermediate position concerning the critical time to begin exposure to fluorides. Their data indicate that almost full protection is provided the smooth surfaces of teeth when exposure to systemic fluorides is started 2 to 3 years before eruption, but for maximum benefits to pit and fissure surfaces, fluorides must be introduced during the earliest stages of tooth calcification.

Because of these discrepant findings, it is difficult to formulate a sound scientific opinion on when fluoride ingestion should be started to produce the greatest decay preventive benefits. For deciduous teeth, which calcify both prenatally and postnatally, formulating an opinion becomes even more difficult because the question concerning placental transfer of fluoride is superimposed on the conflicting data. It has not been shown unequivocally that fluoride at optimum levels passes the placental barrier in sufficient amounts to impart resistance to caries in the developing deciduous teeth (*10–12*).

Most calcification of deciduous teeth occurs after birth (*13*). Therefore, if exposure to fluoride during the last stage of tooth mineralization is sufficient to produce maximum inhibition of caries, whether or not fluoride crosses the placental barrier has only theoretical implications. On the other hand, if the mechanism for greatest protection necessitates exposure to fluoride before birth, the question of maternal transfer of fluoride is of more than academic interest. The answer would provide some basis for either recommending or discouraging the use of dietary supplements of fluoride by preg-

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nant women to protect the deciduous teeth of their offspring.

Clinical data on the efficacy of prenatal exposure to fluoridated water are limited. Some inferences can be made from data of surveys made before and after fluoridation. For example, data from Grand Rapids (*14*) showed that 6-year-old children who drank fluoridated water from birth had 42.0 percent fewer decayed, extraction indicated, and filled (def) teeth than 6-year-old children examined before fluoridation was initiated, whereas children aged 6 years with both prenatal and postnatal exposure to fluorides had 53.7 percent fewer def teeth than the same controls.

As part of the evaluation of fluoridation in Evanston, Ill. (*15*), selected age groups were examined specifically to determine the value of prenatal exposure to fluoride (*16*). Children who were 6 years old in 1955, who had not yet been conceived when fluoridation was initiated in 1947, demonstrated a 36 percent lower prevalence of caries in deciduous teeth than children the same age examined during the baseline survey. Children born in 1947, about half of whom had no prenatal exposure to fluoridated water, when examined at age 6 showed only about 10 percent fewer def teeth than the controls. The investigators, Blayney and Hill (*16*), concluded that prenatal exposure to fluoridated water at 1 ppm gave greater protection to deciduous teeth than that provided by only postnatal exposure.

Carlos and associates (*17*) compared the prevalence of caries in deciduous teeth of 6-year-old Newburgh children, born in the 3-month period immediately before fluoridation was introduced (no prenatal exposure), with the prevalence of caries in deciduous teeth of children age 6 born 1 and 2 years later. The children born 1 and 2 years later were exposed to fluorides during the entire gestation period. The authors reported little difference between the children who were exposed and those who were not exposed to fluorides prenatally in average number of decayed and filled teeth or percentage of children free of caries. However, the three study groups comprised a total of only 178 children.

Tank and Storvick (*18*) investigated the prevalence of caries in children 1 through 6 years of age, who were exposed to fluoridated water. They reported that youngsters who began consuming the fluoridated water from 1 to 11 months postnatally had a higher prevalence of dental caries than those who were exposed to fluoridated water both prenatally and postnatally. Although just 42 children comprised the group with postnatal exposure only, and the difference in caries experience between the two groups was not statistically significant, their results are similar to those reported in the Evanston study (*16*).

Our investigation was undertaken to help resolve the conflict in existing evidence on the benefits of maternal ingestion of fluoride. The specific purpose was to determine if additional dental benefits accrue to children exposed both prenatally and postnatally to optimally fluoridated water compared with children exposed only postnatally. Also, if benefits accrue, the study was designed to pinpoint the trimester of pregnancy during which it is best for a pregnant woman to begin consuming fluoridated water in order to reduce significantly her child's dental caries.

## Methods

Minneapolis, Minn., was selected as the site for the project. The city began to fluoridate its water supply August 6, 1957 (*19*), and has maintained a fairly uniform fluoride content, ranging from 1.0 to 1.2 ppm, considered optimal for a community in its geographic location (*20*, *21*). In September 1964, when the project was started, the community had been fluoridating its water for approximately 7 years. The city is large, and many children were available who were lifelong residents and whose primary teeth were at a developmental stage considered appropriate for the study purpose.

Based on the large size and stability of the school population, we requested administrative school personnel to prepare a roster of elementary and parochial schools with kindergarten classes. From this roster, we selected for participation in the study 39 elementary schools and 3 parochial schools that had students primarily from middle-class socioeconomic backgrounds. Residence history forms were distributed in May 1964 to approximately 8,100 children in the kindergarten and first grades of these schools. A total of 6,593 forms were

completed and returned by parents. From these forms, it was determined that 3,010 children were eligible to participate in the project, because they met the following criteria:

1. They were born May 6, 1957 through August 5, 1958, from 3 months preceding to 12 months following the date fluoridation was initiated.

2. They had always lived within the city limits of Minneapolis.

3. Their mothers had lived within the city limits of Minneapolis for at least a full year before the children were born.

According to date of birth, the child was classified in one of five groups designated by the letters A through E (table 1). The children in group A were born during the 3-month period preceding the addition of fluoride to the city's water supply, and thus they were not exposed prenatally to fluoridated water. The children in group B were born in the first, second, or third month following the start of fluoridation and therefore were first exposed to the fluoridated water during their mothers' last trimester of pregnancy. The children in group C were born 3 to 6 months after the initiation of fluoridation, and thus their mothers were in the second trimester of pregnancy when fluoridation began. Group D children were born 6 to 9 months after fluoridation was started, and therefore their mothers began drinking fluoridated water during the first trimester of pregnancy. All the children in group E were born 9 to 12 months after fluoridation was initiated, and thus they had been exposed to fluoridated water for the full term of their mothers' pregnancies. This schedule of prenatal exposure to fluoridated water is based on pregnancies of exactly 9 months. Administrative considerations prevented us from obtaining an exact history of duration of pregnancy from each mother.

Because caries prevalence is a function of age, and the ages of the children in the five cohorts would differ if all clinical examinations were made at the same time, a series of five examinations was scheduled at trimonthly intervals to obtain near perfect comparability in the mean age of each cohort. At the time of examination, the children were 7 years and 2 through 4 months old (table 1). By controlling age at the time of examination, postnatal exposure to fluorides was controlled, except for cohort A; and thus the sole study variable was the differential period of prenatal exposure to fluoride.

Each survey in the series was scheduled to begin on or as close as possible to the sixth day of the following months: September 1964, December 1964, March 1965, June 1965, and September 1965. These dates were selected within the framework of the school-teaching schedule and relate to the date fluoridation was started (August 6, 1957) in such a way as to provide children with appropriate differential patterns of exposure to prenatal fluorides for study. None of the actual starting dates for examinations deviated from the ideal time by more than 8 days.

**Table 1. Examination schedule based on varying patterns of prenatal exposure to fluoridated water**

| Group | Month of examination [1] | Date of birth | Period exposed to fluoride | | |
|---|---|---|---|---|---|
| | | | Prenatal (months) | Postnatal | |
| | | | | Years | Months |
| A | September 1964 | May 6–Aug. 5, 1957 | None | 7 | 1 |
| B | December 1964 | Aug. 6 [2]–Nov. 5, 1957 | Less than 3 | 7 | 2–4 |
| C | March 1965 | Nov. 6, 1957–Feb. 5, 1958 | 3–6 | 7 | 2–4 |
| D | June 1965 | Feb. 6–May 5, 1958 | 6–9 | 7 | 2–4 |
| E | September 1965 | May 6–Aug. 5, 1958 | Full term | 7 | 2–4 |

[1] All children were 7 years, 2 through 4 months of age as of the 6th day of the month of examination.
[2] Date fluoridation was initiated in Minneapolis.

The children, seated under good artificial light from a portable dental lamp, were examined with a plane mouth mirror and sharp explorer. Examinations were made by two experienced Public Health Service dentists. For each survey in the series, the examiners visited the same schools. Each dentist examined approximately equal numbers of children in each of the five cohorts. The criteria for carious teeth were the same as in other studies conducted by this research unit (*22, 23*).

Examiners often have difficulty, because of individual variation in the time that teeth are shed, in determining whether some deciduous teeth have been extracted or have exfoliated naturally, and thus they may have difficulty in accurately assessing caries experience in the primary dentition. Because of the age of the children in this study, therefore, only deciduous cuspids and molars were examined. Data obtained from studies of the Division of Dental Health and others (*24–26*) indicated that these teeth, if missing, could safely be considered as extracted. (The examiners found several children without mandibular deciduous cuspids; they gave no history of extractions and their other deciduous teeth were relatively free of caries. The missing cuspids, therefore, were not included in the score for decayed, missing, or filled teeth. It was the impression of the examiners that these teeth had exfoliated when the adjacent permanent lateral incisors had erupted.)

Spaces for 12 deciduous teeth were examined in each child. A count was made of the decayed, missing due to extraction, and filled (dmf) deciduous teeth and surfaces, identical to the DMF index used for permanent teeth. Eliminating deciduous incisors from the examination made the scoring of extracted teeth more reliable and concentrated the findings on the more functional areas of the mouth where maintenance of space is important.

Traditionally, it has been accepted that initial calcification of first permanent molars begins at birth or just before birth. More recent studies, however, suggest that calcification of these teeth may begin as early as 28 weeks in utero (*27, 28*). Even if development of first permanent molars begins prenatally, these teeth would receive little benefit, if any, from a few weeks of prenatal exposure to fluorides.

It has been hypothesized, but not yet demonstrated however, that the developing bones of the fetus may serve as a repository for fluoride, which may subsequently be mobilized and enter tooth enamel that develops postnatally (*29*). If this phenomenon occurs, first permanent molars could receive protection from fluoride that was incorporated in the developing bones before birth. To measure this possible effect, first permanent molars were included in the clinical examination.

### Findings

A total of 2,509 children were examined, or approximately 83 percent of those who met the requirements for participation. Absence of the child on the day of examination, movement of the family out of the school district, or transfer of the child to a public or parochial school not included in the study constituted the major reasons for the loss of study subjects. The participants were well distributed among the five

**Table 2. Distribution of children in each group according to sex**

| Sex | Group | | | | | | | | | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | A | | B | | C | | D | | E | | | |
| | Number | Percent | Number | Percent | Number | Percent | Number | Percent | Number | Percent | Number | Percent |
| Male | 262 | 51. 5 | 217 | 50. 9 | 235 | 49. 7 | 296 | 52. 1 | 264 | 49. 5 | 1, 274 | 50. 8 |
| Female | 247 | 48. 5 | 209 | 49. 1 | 238 | 50. 3 | 272 | 47. 9 | 269 | 50. 5 | 1, 235 | 49. 2 |
| Total | 509 | ------ | 426 | ------ | 473 | ------ | 568 | ------ | 533 | ------ | 2, 509 | ------ |

**Table 3. Average caries prevalence of deciduous teeth according to group**

| Group | Number of children | Prenatal fluoride exposure (months) | Average caries rates [1] | |
|---|---|---|---|---|
| | | | dmf teeth per child | dmf surfaces per child |
| A | 509 | None | 3. 43 | 6. 35 |
| B | 426 | Less than 3 | 3. 47 | 6. 70 |
| C | 473 | 3–6 | 3. 32 | 6. 20 |
| D | 568 | 6–9 | 3. 09 | 5. 72 |
| E | 533 | Full term | 3. 23 | 6. 02 |

[1] Spaces for 12 deciduous teeth were examined in each child.

cohorts according to sex and total numbers (table 2). Each cohort contained more than 425 children.

The average prevalence of caries per child for 12 deciduous tooth spaces, according to cohort, is shown in table 3. Despite some fluctuation in the mean rates, the tabular data indicate that the children in the five cohorts experienced only slight differences in caries experience. Mean rates of dmf teeth in the various groups ranged between 3 and 3½ "attacked" teeth. Differences in the rates of dmf surface caries did not exceed one per child. Although the prevalence rates are similar, a very slight downward trend in dental caries is suggested as the length of prenatal exposure to fluoride increases.

A breakdown of the average rates of dmf teeth, according to type of tooth for each group, is shown in table 4. For all cohorts, second deciduous molars exhibited the greatest decay, averaging more than 1½ teeth per child. Decay rates for cuspids were low—less than 0.25 teeth per child in each group.

A comparison of tooth-specific rates (table 4) reveals essentially the same pattern of results as found on all deciduous teeth. Only small differences existed between the groups in the average number of affected teeth. Again, there may be a slight downward trend in prevalence rates of caries as prenatal exposure to fluoride increases.

From a visual inspection of the findings on dmf teeth and surfaces, it appears that the relation, if any, between prenatal exposure to fluorides and resistance of deciduous teeth to decay is minimal. To assess the relation statistically, however, correlation coefficients ($r$) were calculated between individual dmf tooth and surface scores and length of prenatal exposure to fluoride. The rationale for using this method of analysis was based on the assumption that the relations were essentially linear. For the correlation analysis, the mean length of prenatal exposure to fluoride, by group, was measured according to number of half trimesters of pregnancy, as follows: A=0, B=1, C=3, D=5, and E=6. For these data the calculated correlation coefficients were −0.04 for both deciduous teeth and surfaces, indicating extremely low inverse relations between caries experience in deciduous teeth and prenatal exposure to fluoridated water. More than 99 percent of the variance in experience in both dmf teeth and surfaces is left "unexplained" by prenatal exposure to fluorides.

Data on prevalence of caries in first perma-

**Table 4. Average tooth-specific dmf rates according to group**

| Group | Type of deciduous tooth | | | |
|---|---|---|---|---|
| | Cuspid | 1st molar | 2d molar | Total |
| A | 0. 24 | 1. 39 | 1. 80 | 3. 43 |
| B | . 23 | 1. 36 | 1. 88 | 3. 47 |
| C | . 22 | 1. 35 | 1. 75 | 3. 32 |
| D | . 20 | 1. 23 | 1. 66 | 3. 09 |
| E | . 20 | 1. 29 | 1. 74 | 3. 23 |

**Table 5. Average caries prevalence of first permanent molars according to group**

| Group | Number of children | Prenatal fluoride exposure (months) | Average caries rates | |
|---|---|---|---|---|
| | | | DMF teeth per child | DMF surfaces per child |
| A | 509 | None | 0. 66 | 0. 81 |
| B | 426 | Less than 3 | . 63 | . 79 |
| C | 473 | 3–6 | . 78 | . 98 |
| D | 568 | 6–9 | . 52 | . 64 |
| E | 533 | Full term | . 54 | . 68 |

nent molars, according to cohort, are presented in table 5. Similar to the findings for deciduous teeth, only small differences in average number of DMF teeth or surfaces were found between cohorts. The average scores for DMF surfaces were only slightly higher than the corresponding mean scores for DMF teeth, indicating that most first permanent molars experiencing decay had only one affected surface. As for deciduous teeth, there may be a slight downward trend in mean scores for DMF teeth and surfaces with longer prenatal exposure to fluorides.

We calculated a coefficient of $-0.05$ as the correlation between average number of DMF teeth and length of prenatal exposure to fluoridated water. For DMF surfaces, $r$ equaled $-0.04$. These values are of the same magnitude as for deciduous teeth.

From these data, it can be concluded that the relation, if any, between prenatal exposure to fluoride and prevalence of caries in deciduous cuspids and molars and permanent first molars is of very low degree and has no practical significance in the prevention of decay.

## Discussion

Except for the children in group A, who were born during the 3 months preceding fluoridation of the water in Minneapolis, all children in this study had consumed optimally fluoridated water since birth. The findings of this investigation, therefore, do not provide information on the caries-inhibiting effect of exposure to fluorides only prenatally. It is conceivable that the developing teeth were protected by prenatal exposure to fluorides but that the effect was masked by the subsequent postnatal use of fluoridated water. If there were a masked prenatal effect, it would have little practical significance and be only of academic interest.

The findings of the Minneapolis study concerning deciduous teeth are essentially in agreement with those of Carlos and co-workers (*17*) in Newburgh, N.Y. The similarity in study designs may account, in part, for the consistent findings. In both studies, a relatively short period of time elapsed between serial examinations, virtually eliminating the potential uncontrolled variables that can be introduced when several years intervene between two comparative examinations. In the latter situation, changes in diet, oral hygiene practices, professional preventive care, and use of therapeutic dentifrices can influence the measured variable (dental caries experience) and thus confound interpretation of the results.

Aside from one equivocal report by Feltman and Kosel (*30*), no studies have been reported on the efficacy of dietary supplements of fluoride for pregnant women. The recommendation for their use during pregnancy has been based on the lack of evidence indicating harmful effects to the mother or the developing fetus and the prospect that benefits may accrue to the child's deciduous teeth. This rationalization constitutes a weak justification for the use of dietary supplements of minerals. The few clinical studies that report dental benefits to children whose mothers consumed fluoridated water during pregnancy had served as a basis for the wide-scale promotion by the pharmaceutical industry of prescribing dietary fluorides for pregnant women. The Minneapolis data do not support this practice.

Dietary supplements of fluoride taken once a day are rapidly cleared from the body (*31*, *32*). There is reason to believe, therefore, that they would be less effective than fluoridated water, which is consumed throughout the day and thus maintains a more constant blood-fluoride level. Certainly, more proof of efficacy is needed before dietary supplements of fluoride can be recommended for gravid women as a sound public health procedure. The Food and Drug Administration, recognizing that insufficient evidence existed to support claims for benefits from prenatal use of preparations containing fluorides, recently banned the marketing by manufacturers of these products offered for prenatal decay prevention (*33*).

## Summary and Conclusions

Five cohorts totaling 2,509 children of the same age (7 years, 2 through 4 months), all with essentially the same postnatal exposure to optimally fluoridated water but with different patterns of prenatal exposure, were compared for prevalence of dental caries in their decidu-

ous cuspids and molars and first permanent molars.

The data indicate that there were no meaningful additional benefits from the maternal ingestion of fluoridated water if the offspring also ingested the water from birth. The results cast serious doubts on the benefits to be derived from dietary supplements of fluoride to pregnant women.

#### REFERENCES


(1) Arnold, F. A., Jr., Likins, R. C., Russell, A. L., and Scott, D. B.: Fifteenth year of Grand Rapids fluoridation study. J Amer Dent Assoc 65:780–785, December 1962.

(2) Brown, H. K., and Poplove, M.: Brantford-Sarnia-Stratford fluoridation caries study. Final survey. J Canad Dent Assoc 31: 505–511, August 1965.

(3) Ast, D. B., Smith, D. J., Wachs, B., and Cantwell, K. T.: Newburgh-Kingston caries-fluorine study. XIV. Combined clinical and roentgenographic dental findings after ten years of fluoride experience. J Amer Dent Assoc 52: 314–325, March 1956.

(4) Knutson, J. W.: An evaluation of the Grand Rapids water fluoridation project. J Mich Med Soc 53:1001–1006, September 1954.

(5) Russell, A. L., and Hamilton, P. M.: Dental caries in permanent first molars after eight years of fluoridation. Arch Oral Biol 6 (special supp.): 50–57 (1961).

(6) Weaver, R.: Fluorine and dental caries. Further investigations on Tyneside and in Sunderland. Brit Dent J 77: 185–193, Oct. 6, 1944.

(7) Grainger, R. M., and Coburn, C. I.: Dental caries of the first molars and the age of children when first consuming naturally fluoridated water. Canad J Public Health 46:347–354, September 1955.

(8) Klein, H.: Dental caries (DMF) experience in relocated children exposed to water containing fluorine. II. J Amer Dent Assoc 33:1136–1141, September 1946.

(9) Backer Dirks, O., Houwink, B., and Kwant, G.: Some special features of the caries preventive effect of water-fluoridation. Arch Oral Biol 4 (special supp.):187–192, August 1961.

(10) Dale, P. P.: Prenatal fluorides: The value of fluoride during pregnancy. J Amer Dent Assoc 68:530–534, April 1964.

(11) Zipkin, I., and Babeaux, W. L.: Maternal transfer of fluoride. J Oral Ther 1:652–665, May 1965.

(12) Schlesinger, E. R.: Dietary fluorides and caries prevention. Amer J Public Health 55: 1123–1129, August 1965.

(13) Kraus, B. S.: Calcification of the human deciduous teeth. J Amer Dent Assoc 59:1128–1136, December 1959.

(14) Arnold, F. A., Jr., Dean, H. T., Joy, P., and Knutson, J. W.: Effect of fluoridated water supplies on dental caries prevalence. Public Health Rep 71:652–657, July 1956.

(15) Blayney, J. R.: A report on thirteen years of water fluoridation in Evanston, Illinois. J Amer Dent Assoc 61: 76–79, July 1960.

(16) Blayney, J. R., and Hill, I. N.: Evanston dental caries study. XXIV. Prenatal fluorides—value of waterborne fluorides during pregnancy. J Amer Dent Assoc 69: 291–294, September 1964.

(17) Carlos, J. P., Gittelsohn, A. M., and Haddon, W., Jr.: Caries in deciduous teeth in relation to maternal ingestion of fluoride. Public Health Rep 77: 658–660, August 1962.

(18) Tank, G., and Storvick, C. A.: Caries experience of children one to six years old in two Oregon communities (Corvallis and Albany). I. Effect of fluoride on caries experience and eruption of teeth. J Amer Dent Assoc 69:749–757, December 1964.

(19) U.S. Public Health Service: Fluoridation census, 1965. Washington, D.C. Mimeographed.

(20) U.S. Weather Bureau: Climatic summary of the United States; supplement for 1951–60. [Pamphlet] U.S. Government Printing Office, Washington, D.C., 1964, p. 45.

(21) U.S. Public Health Service: Public Health Service drinking water standards. PHS Publication No. 956. U.S. Government Printing Office, Washington, D.C., 1962, p. 8.

(22) Horowitz, H. S., Law, F. E., Thompson, M. B., and Chamberlin, S. R.: Evalution of a stannous fluoride dentifrice for use in dental public health programs. I. Basic findings. J Amer Dent Assoc 72: 408–422, February 1966.

(23) Horowitz, H. S., and Lucye, H. S.: A clinical evaluation of stannous fluoride in a prophylaxis paste and as a solution. J Oral Ther. In press.

(24) Miller, J., Hobson, P., and Gaskell, T. J.: A serial study of the chronology of exfoliation of deciduous teeth and eruption of permanent teeth. Arch Oral Biol 10: 805–818, September-October 1965.

(25) Toverud, G.: Kariesetiologi og profylakse. [Ekstrakt av Forelesninger 1946 og 1947] Oslo, 1947.

(26) Fulton, J. T., Hughes, J. T., and Mercer, C. V.: The life cycle of human teeth. University of North Carolina School of Public Health, Chapel Hill, 1964, pp. 7–18.

(27) Christensen, G. J., and Kraus, B. S.: Initial calcification of the human permanent first molar. J Dent Res 44: 1338–1342, November-December 1965.

(28) Boller, R. J.: Fetal morphogenesis of the human

dentition. J Dent Child 31 (2d Quarter): 67–97 (1964).
(*29*) Council on Dental Therapeutics: Value of fluoride administered to expectant mothers. American Dental Association, Chicago, Mar. 5, 1962.
(*30*) Feltman, R., and Kosel, R.: Prenatal and postnatal ingestion of fluorides: Fourteen years of investigation; final report. J Dent Med 16: 190–198, October 1961.
(*31*) Hennon, D. K., and Muhler, J. C.: Clinical use of fluorides. J Indiana Dent Assoc 41: 88–95, March 1962.
(*32*) Muhler, J. C., Stookey, G. K., Spear, L. B., and Bixler, D.: Blood and urinary studies following the ingestion of single dosages of fluoride. J Oral Ther 2: 241–260, January 1966.
(*33*) Food and Drug Administration: Statements of general policy or interpretation, oral prenatal drugs containing fluorides for human use. Federal Register, Oct. 20, 1966.

# PUBLICATION ANNOUNCEMENTS

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*Annual Report of the Pinellas County Health Department for 1965.* 1966; 26 pages. Pinellas County Health Department, Fifth Street and Second Avenue North, St. Petersburg, Fla.

*The Emergence of the American Welfare State.* Reprinted from Vol. XVIII, No. 2, January 1965, McCormick Quarterly. By Charles G. Chakerian. 1966; 11 pages. McCormick Theological Seminary, 800 West Belden Avenue, Chicago, Ill. 60614.

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