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LABORATORY FOR ENERGY-RELATED HEALTH RESEARCH

DAVIS, CALIFORNIA 95616
(916) 752-1340 (FTS 453-1340)

3/21/80

#23

March 18, 1980

Dr. Brian K. Grimes, Director
Emergency Preparedness Task Group
Office of Nuclear Reactor Regulation
Nuclear Regulatory Commission
Washington DC 20555

Dear Dr. Grimes:

Enclosed is my evaluation of "Infant Mortality Changes following the Three Mile Island Accident," by E. J. Sternglass.

I have not addressed his statements about the Millstone and Connecticut Yankee reactors since they do not pertain to the Three Mile Island releases. Furthermore, they appear to be similar to statements he has made in the past that have been addressed by others.

I hope my comments are of some assistance to you.

Sincerely yours,

Steven A. Book

Steven A. Book, Ph.D.
Associate Research Physiologist

SAB:nh

cc w/encl: Dr. R. Gotchy, NRC
Dr. M. Parsont, NRC
Dr. M. Goldman, LEHR

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Comments on "Infant Mortality Changes following the Three Mile Island Accident" by E. J. Sternglass

Steven A. Book

Laboratory for Energy-Related Health Research
School of Veterinary Medicine
University of California, Davis, California 95616

In the paper "Infant Mortality Changes Following the Three Mile Island Accident" by E. J. Sternglass, the author claims an increase in infant mortality followed releases of radioactivity from the Three Mile Island accident. He considers the alleged increase in infant mortality to result from (1) the concentration of iodine-131 in fetal thyroid glands and (2) a consequent increase in immature and underweight infants that died of respiratory distress.

In my opinion, the claims presented in that paper are without substance and are based on fallacious arguments that violate basic concepts in areas of physiology, radiation biology, and epidemiology.

The basis for that paper appears to be that a higher incidence of respiratory distress occurs in immature infants. On page 4 it is stated that "even a small degree of retardation in development due to a reduced output of growth hormone [sic] by the thyroid gland during the last three to four months of intrauterine development would be expected to increase the risk of death due to respiratory insufficiency immediately after birth. Failure of the critical lung surfactant to be produced in adequate amounts can therefore lead to respiratory problems and death as a result of damage either to the thyroid or the pituitary gland which in turn controls the thyroid's output of growth hormone [sic] (17)."

In terms of basic physiology, that paragraph makes little sense. Growth hormone, of course, is not secreted by the thyroid gland. It is secreted by specific cells of the anterior pituitary gland and is involved in generalized growth and metabolism, but it has no particular target organ. Thyroid glands secrete the thyroid hormones, tetraiodothyronine (thyroxine) and triiodothyronine, required for normal development of bone and nervous tissue. I have been unable to find any reference to thyroid hormone or growth hormone involvement in production of lung surfactant. Even reference 17 cited in that paper does not implicate either of those hormones, but instead demonstrates the need for a functional pituitary-adrenal gland axis for normal lung development. Regardless of the endocrine involvement in lung development, it

appears the relationship of thyroid and surfactant production basic to the author's claims is questionable.

In stating that the reduced output of thyroid gland would lead to retarded development and an increased likelihood of death from respiratory insufficiency, the author requires us to accept the notion that his proposed "immaturity" is the same as prematurity with all its attendant problems (including respiratory distress). While that acceptance may sound reasonable at the outset, prematurity is a different situation altogether.

Another area that is basic to the claims of that paper has to do with the increased radioiodine concentration of the fetal thyroid and the consequent dose. Estimates of fetal thyroid dose are not large enough, however, to result in effects on the thyroid gland.

While it is true that the fetal thyroid gland begins to function early in gestation, it is not true that the fetal thyroid doses can be 100 times those of adults. In general, the fetal thyroid concentration of ^{131}I would be up to about 10 times the maternal thyroid concentration, and the subsequent dose similarly within one order of magnitude. The proposed 100-fold difference results from Eisenbud's observation (reference 3) of a 10-fold higher dose rate in a fetal thyroid than in children, based on a single unusual case. Elsewhere in the same paper, however, Eisenbud mentions that children have doses about twice those of adults. Hence, according to those values, fetuses would have (10 x 2 =) 20 times the dose of adults, not 100. Furthermore, since those data were from fallout ^{131}I, the assimilated ^{131}I was primarily from ingested milk; from inhaled ^{131}I, as the author mentions on page 2 of the Three Mile Island paper, the dose to adults was the same as for infants (about 0.01 rad by inhalation), and the dose to fetuses, therefore, would be about (1 x 10 =) 10 times that to adults. The dose to the fetal thyroid, then, would appear to be no more than about 0.1 rad.

The author assumes another 0.1 rad to the thyroid from other radionuclides including ^{3}H, ^{134}Cs, ^{137}Cs, ^{140}Br, and ^{89}Sr (pages 2 and 3). I am not aware of the release of any radionuclides other than of isotopes of xenon, iodine, and perhaps krypton from Three Mile Island; hence, doses from those other radionuclides would be unlikely. Furthermore, even were those radionuclides released, they are not concentrated by the thyroid gland, and they would not have resulted in any appreciable thyroid dose.

The reduction of growth observed in exposed Marshallese is cited as being supportive of the author's arguments. While there were definite thyroidal effects resulting from the exposure of The Marshallese to weapons fallout, the doses were many orders of magnitude greater than doses from Three Mile Island, and effects were manifested years after exposure. Thyroid doses to children less than 10 years of age were from 810-1150 rads from various radioiodines (+ 175 rads to the total body) in the group that demonstrated the most thyroid effects. Children that received 275-450 rads to the thyroid (+ 69 rads, total body) demonstrated fewer effects. Children exposed to 60-95 rads to the thyroid, principally from ^{131}I (+ 14 rads total body) showed no thyroid effects. Only 1 of 3 exposed in utero to 175 rads from external gamma plus up

to an estimated 3000 rads to the thyroid demonstrated thyroid lesions. Based on these numbers and many others available in the scientific literature, the estimated 0.1 rad to the fetal thyroid is not likely to result in thyroid dysfunction. That dose, I emphasize, is many orders of magnitude lower than doses at which immediate thyroid effects have been observed.

The cause and effect relationship that is professed assumes ^{131}I and thyroid effects to be the cause and infant mortality to be the effect. As demonstrated above, the cause as presented appears not to be a cause. As I will discuss below, the effect similarly appears not to be an effect.

Changes in infant mortality do not appear to be the proper parameters for addressing biologic effects, since infant mortality involves a number of uncertainties. I suspect that most of the monthly changes discussed in the paper are not real changes. Rather, they reflect normal variability. There are too many fluctuations in the small numbers utilized to draw meaningful conclusions from them. For example, in Fig. 5, monthly rates are seen to increase from ~1.1% mortality in March to ~1.8% in July, a change purported to be of even more significance because of the lower mortality of summer months. However, a look at infant mortality for July for the past 10 years shows a range of 1.3 to 2.1% infant mortality. Furthermore, I suspect that the changes in national infant mortality for which the author blames nuclear weapons testing are related instead to changing practices in neonatal medicine in the 1950's and 1960's, particularly as they relate to premature births and the likelihood of survival. The presence of a correlation does not necessarily mean cause and effect.

I do not find the paper "Infant Mortality Changes Following the Three Mile Island Accident" by E. J. Sternglass to be scientifically valid. The paper attempts to show how low doses from ^{131}I to the thyroid caused increased infant mortality due to respiratory distress of immature infants. It fails to do so in a reasonable manner, and, therefore, appears to me to be without scientific merit.

#4

LETTER REPORT

PATHWAYS OF IODINE-131 TO MILK FOLLOWING THE THREE MILE ISLAND INCIDENT

D. A. Baker
R. G. Schreckhise
J. K. Soldat

June 1983

Pacific Northwest Laboratory
Operated for the U.S. Department of Energy
by Battelle Memorial Institute

PATHWAYS OF IODINE-131 TO MILK FOLLOWING THE THREE MILE ISLAND INCIDENT

D. A. Baker, R. G. Schreckhise, and J. K. Soldat

SUMMARY

A study sponsored by the U.S. Nuclear Regulatory Commission was conducted by Pacific Northwest Laboratory to determine the pathway of iodine-131 observed in some of the milk samples collected from the surrounding area following the incident at the Three Mile Island Number 2 nuclear power station on 28 March 1979. Information from the utility, its consultants, and other governmental agencies were collected and studied. Various potential pathways were considered, and finally two were selected for detailed study: inhalation and ingestion. Using the mathematical procedure of convoluting the air concentrations near the animal locations with the impulse response functions representing the two pathways in the animals, it was found that for the three farms selected for study the most probable pathway was air inhalation, at least in the first ten days after the accident. Finally, a simple model was developed to estimate peak milk concentration of iodine-131 after an accidental release into the atmosphere from the inhalation. The model utilizes the ambient air concentration averaged over the time of the release.

STATEMENT OF THE PROBLEM

In the days immediately following the accident, which occurred on the morning of 28 March 1979 at the Three Mile Island 2 nuclear power plant near Middletown, Pennsylvania, iodine-131 was detected in milk from cows and goats residing on farms in the surrounding area. Since this was very early in the spring, the cows and goats were essentially "not on pasture," but were being fed stored feed and silage. Thus, it was assumed at the time that these animals could not derive any iodine-131 from the normal air-grass pathway. So why was there measurable iodine-131 in their milk? The problem then was to determine the probable pathway for this iodine and to develop a suitable model or revise existing NRC models to account for this phenomenon.

The ensuing sections of this report discuss our investigations and proposed solution to this problem.

INFORMATION COLLECTED AND SITE VISIT

Information from various organizations was provided by our NRC project representative, Sarbes Acharya. Other information was also received from William E. Riethle, Supervisor, TMI Environmental/Impact Assessment Group, of the GPU Service Corporation during a site visit 30-31 October 1979. It consisted of the results of their monitoring program during the period after the accident which had been compiled by their consultant, Bruce Newman of Porter-Gertz Consultants, Ardmore, Pennsylvania. Also included were measurements of iodine-131 in air, vegetation and milk. In addition, descriptive material was furnished on their meteorological programs, iodine-131 release rates, animal husbandry practices in the region, goat and cow census data, and a description of the procedure for measuring iodine-131 in milk. The latter information was provided by Drs. Hewitt W. Jeter and John David Martin of Teledyne Isotopes, Westwood, New Jersey.

During the site visit, Mr. Ron Laufer, the person responsible for collecting milk samples for the GPU monitoring program, arranged for us to meet the owners and tour two farms: the Becker farm which had the highest concentration of iodine-131 in cows' milk found by the utility monitoring program, and the Hardison farm where iodine-131 in goats' milk was detected (Figure 1 shows locations). Figures 2 and 3 show the variation with time of the iodine-131 concentrations in milk from the Becker and Hardison farms, respectively. Data from the Alwine and Fisher farms (Figures 4 and 5) were also analyzed; however, these farms were not visited. From conversations with Mr. Becker and Mrs. Hardison, we found that although the animals were not on pasture for their essential nourishment, they did have access to pasture at the time of and immediately following the accident. Therefore, it was possible for the animals to graze on the standing dead vegetation. However, later in the month the goats' pasture "greened up" according to Mrs. Hardison. The goats were then eating fresh pasture in addition to their usual winter diet of stored feed which probably accounts for the sudden increase in iodine-131 concentrations in their milk during the latter part of April (see Figure 3).

During the afternoon of 30 October 1979, we visited with Mr. John Collins of the NRC at the site. The next morning we visited with Ms. Margaret A. Reilly and Mr. Don Beaver of the Pennsylvania Department of Environmental Resources at Harrisburg in order to get more opinions on the problem.

POTENTIAL PATHWAYS INVESTIGATED

The pathways that could have been responsible for iodine-131 measured in the milk were:

1. drinking water
2. silage, in the case of the cows
3. pasture (limited but accessible)
4. inhalation.

Drinking water was pretty much ruled out as a source of iodine-131 because well water was being used. The cows did have access to a small creek during their time on pasture; however, the owner (Mr. Becker) said they didn't use this much.

The Becker cows were given a combination of silage (chopped corn stalks) and stored grain. The silage was loaded from a closed silo to an open farm wagon, then taken to a conveyor where it was transferred to covered eating troughs. Since the cows took only about 30 minutes to eat the silage, and because only the top of the silage could be deposited with iodine, this pathway was not considered a potential route.

The limited pasture available to the animals was deemed to be the most probable pathway. Inhalation seemed unlikely. This was particularly true because the whole-body counting results of the people in the surrounding area (except plant workers) showed no detectable levels of iodine-131 in their bodies (Auxier, 1979, p. 26). However, we did not rule out inhalation entirely since the farm animals and the people were not in the same place at the same time and the reduced air infiltration provided by snug houses may have been a factor in the negative whole-body counting findings.

CONVOLUTION STUDY

To determine if inhalation or grass ingestion was the dominant pathway, a simple convolution model was developed. The air concentration was the input function, and two different retention functions were used as the impulse-response function. The output which represented the variation of milk concentration with time was then cross-correlated with the measured values of milk/iodine-131 concentration. Graphs of the input, impulse and output functions are shown in Figures 6 through 8.

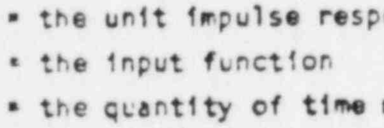
Convolution is a method of determining the output of a linear system as a function of time where the input function as well as the unit impulse response function (IRF) is known. The method was developed for electrical circuits, but can be generalized to any linear system. The convolution process is described in the relation:

$$V_2(t) = \int_0^t V_1(t - \tau)\, U(\tau)\, d\tau \qquad (1)$$

where

$V_2(t)$ = the output function, as a function of time, t
$U(\tau)$ = the unit impulse response function (IRF) of the system
$V_1(t - \tau)$ = the input function
τ = the quantity of time measured backward from any time t

Essentially, the convolution process is a weighting process in which each value of V_2 is determined by each past value of V_1 suitably weighted by the IRF. Take, for example, the decaying exponential function as the impulse response function:



$$U(t) = e^{-\lambda t}$$

Here V_2 is mainly influenced by recent values of V_1 while "very old" values of V_1 have very little effect on V_2.

Consider the case of a constant input rate as an example:

$$V_1(t) = I$$

then the form of the convolution integral becomes:

$$V_2(t) = \int_0^t I\, e^{-\lambda t}\, d\tau$$

Evaluating the integral gives,

$$V_2(t) = I\,\frac{(1 - e^{-\lambda t})}{\lambda}$$

which is the familiar equation that describes the buildup of a tracer in a system where the input is constant and the loss is a first-order rate constant.

The added advantage of the convolution integral is that it can also be evaluated numerically. This is very useful for instances where the input is not constant or cannot be described adequately by a mathematical expression. This is basically the type of input we have with the available iodine-131 air concentration data. A very good description of convolution with examples and numerical methods of computation can be found in Cooper and McGillem (1967).

For the inhalation pathway, the impulse response function is just:

$$U_A(t) = e^{-\lambda_A t} \qquad (2)$$

where

λ_A = the effective loss constant of iodine-131 in the cow (d^{-1}). It includes both the biological loss constant and the radiological decay constant.

The IRF for the ingestion pathway is dependent on two exponentials:

$$U_{IG}(t) = C \frac{e^{-\lambda_E t} - e^{-\lambda_A t}}{\lambda_E - \lambda_A} \qquad (3)$$

where

C = a constant dependent on the rate of contaminated grass eaten by the cow, the fraction of this grass which is contaminated, and the fraction of iodine-131 ingested which is transferred to the milk.

λ_E = the effective environmental loss constant of iodine-131 on the pasture grass (d^{-1}). It includes both the weathering or field-loss constant and the radiological decay constant.

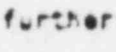
In the discussion to follow, we will refer to these effective loss constants by their equivalent effective half times in days which are related by:

$$T = \ln 2/\lambda$$

Hoffman and Baes (1979) have made a study of the variation in the effective environmental loss half time T_E. They have found from the literature that T_E is quite variable with a range of between 2.8 to 5 days with a mean of 4.5 days. However, Shaeffer (Miller et al., 1979, p. 44) presents values of T_E, estimated from releases of iodine-131 at Dresden and Monticello that vary from 0.94 to 4.6 days with a mean of 2.9 days. Since these releases should be more like the actual reactor releases than

experimental setups, we have assumed for the following discussion a T_E value of 3 days.

For the effective animal half life, T_A a value of around one day has been frequently given in the literature. Garner and Russell (Russell, 1966, p. 304) give a value of half life of 16 hours for the first part of the decay curve. A value of one day was found from the CERT experiments (Hawley et al., 1964, p. 59). Values for Dresden and Monticello releases given by Shaeffer (Miller et al., 1979, p. 44) give T_A values ranging from 0.59 to 2.18 days with a mean of 0.66 days. For the same reason as above for selecting T_E, a T_A value of 0.7 day was used.

The air concentration measurements (Figures 6A, 7A, 8A) that were obtained at locations closest to the three farms studied were convoluted with each of the two impulse response functions (i.e., inhalation and ingestion). The resulting output functions then were compared to the actual measured values of milk concentration. Figures 6 thru 8 show these plots. By general appearance, it is argued that at least for the first ten days or so after the accident, the output curve from the inhalation impulse response function "  " was more similar to the plot of actual data than that from the ingestion pathway. The output functions (Figures 6b,c - 8b,c) do not, however, show the concentration quantitatively, since correction factors would have to be applied. However, the graphs do show the concentration variations qualitatively.

As a further argument, the cross correlations of the output data from the convolutions were made with the actual milk concentration data for the first ten days for comparison. The usual Pearson product moment correlation coefficient was obtained using the MINITAB statistical computing system (Ryan et al., 1976). Table 1 shows these results which furthers the argument that the inhalation pathway was the predominant mechanism in which radioiodine was transferred to cows' milk in the ten days (at least) succeeding the accident.

INHALATION PATHWAY MODEL

To further test the inhalation hypothesis, data to derive an inhalation-to-milk transfer factor were obtained from a report by Stuart C. Black and

TABLE 1. [illegible]ults of Cross Correlation of the Output Values [illegible]om the Convolution with Mea[illegible]ured Values of Iodine-131 Milk Concentrations

Farm	Pathway: Ingestion of Grass	Pathway: Air Inhalation
Becker	-0.371	0.949
Alwine	0.208	0.972
Fisher	0.586	0.887

Delbert S. Barth on some of their experiments carried out at the EPA Environmental Monitoring and Support Laboratory near Las Vegas (Black and Barth, 1975) and a personal communication from Paul Voillequé in which he gave us some data on an inhalation experiment he carried out during one of the CERT experiments near Idaho Falls. He found that, in an experiment of over one-hour duration, his cows inhaled a total of 0.74 Ci of iodine-131 from an integrated air concentration of 520 μCi-s-m^{-3}. The peak milk concentration following inhalation was 1400 pCi/L with a 2-1/2 day half-life.

Let us define a parameter, F_I, which relates the peak concentration of iodine-131 in milk (pCi/L) derived from air intake[a] to the integrated air concentration (pCi-s-m^{-3}):

$$F_I \equiv \frac{\text{Peak Milk Concentration}}{\text{Integrated Air Concentration}} \quad m^3\text{-}L^{-1}\text{-}s^{-1} \tag{4}$$

Then from the data of Voillequé given above and that of Black and Barth (1975, p. 15) we can calculate a value for F_I. Table 2 shows these experimental data and the results of the calculation.

(a) This term from Black and Barth includes inhalation as well as ingestion of iodine-131 occurring when the cow licks its muzzle, nares or surrounding objects.

TABLE [illegible] [illegible]
[illegible] Experimental Data

[illegible]	[illegible] Conc. [illegible]	Integrated Air Conc. [illegible]	[illegible]	Reference
[illegible]	1.4E3	5.2E6	[illegible]-6	[illegible]
HAYSEED	[illegible]	3.2E8	1.8E-[illegible]	Black & Barth, [illegible]
ALFALFA	[illegible]	3.3E8	6.6E-6	Black & Barth, [illegible]
[illegible]	1.1E3	1.6E8	7.4E-6	Black & Barth, [illegible]
MICE*	3.6E3	1.3E8	2.7E-5	Black & Barth, [illegible]

* The iodine was in the form of a gas during this experiment.

A regression line may be drawn between the two points: the median, x_{50}, and the 99th percentile, x_{99}, which are calculated from original data in the following manner (Hoffman and Baes, 1979, pp. 7-9).

$$x_{50} = x_m = \exp(\mu), \text{ and} \quad (5)$$

$$x_{99} = \exp(\mu + 2.326\,\sigma) \quad (6)$$

where μ and σ are the population mean value and standard deviation respectively. For calculational purposes we will assume the estimates of these values are the same. Then

$$\mu = \left(\sum \ln x_i\right)/n, \text{ and}$$

$$\frac{\sum_1 (\ln x_i)^2 - \left(\sum_1 \ln x_i\right)^2/n}{[illegible]}$$

Also the most probable value is given by

$$X_p = \exp(\mu - \sigma^2), \text{ and} \tag{9}$$

the mean value by

$$\bar{X} = \exp(\mu + \sigma^2/2) \tag{10}$$

Table 3 shows the F_I factors sorted in ascending order along with their cumulative probabilities calculated using the relationship $(i - 0.375)/(n + 0.25)$ recommended by Gosslee and Mitchell (1972, p. 4) for a small number of data points, n. The particle size is given for the Black and Barth experiments. It is interesting to note that F_I and particle size is inversely related.

TABLE 3. Sorted F_I Values with Their Corresponding Cumulative Probabilities and Particle Size

i	F_I	Cumulative Probability $(i - 0.375)/5.25 \times 100\%$	Particle Size (μm)
1	1.8E-6	12	23
2	2.7E-6	31	--
3	6.6E-6	50	2
4	7.4E-6	69	0.13
5	2.7E-5	88	(Gas)

Figure 9 is a log normal plot of these experimental data points. From the regression line and equations (9) and (10), we find that the median (50 percentile) value is 5.8×10^{-6}, the mean is 1.0×10^{-5}, and the most probable is 1.9×10^{-6} $m^3\text{-}L^{-1}\text{-}s^{-1}$.

From the utility data on TMI air concentrations measured at three sites over two days from noon of 29 March to noon of 31 March, air concentrations for the three farms were estimated. Air concentrations of iodine-131 at the Falmouth Observation Center and Goldsboro monitoring sites were extrapolated to that of the Becker, Alwine and Fisher sites respectively using curves of dilution factor versus distance found in Auxier (1979, pp. 111, 113). Since no air monitoring of iodine-131 was performed at the farms, all monitoring sites had to be chosen that were nearly in the same direction as the respective farm sites. The F_I was then calculated for the three farms (Table 4) from the measured milk concentrations on the 31st (assumed to be the peak). Note that the missing data value for peak milk concentration on 31 March has been extrapolated from the succeeding days and from the values at the other farms.

The concentration of iodine-131 in milk or other animal products from air inhalation is usually insignificant compared to that resulting from ingestion of pasture grass contaminated by the same air. However, for the special circumstance when animals obtain all their nutrition from uncontaminated stored feed or silage, while having access to the outdoor air, inhalation may be a primary pathway of iodine-131 from air to milk.

The peak concentration in milk in pCi/L may be estimated from air inhalation knowing the integrated air concentration in $pCi\text{-}s\text{-}m^{-3}$ at the location of the animals. Hence,

$$C_{mp} = F_I \int_0^T \chi(t)\, dt \qquad (pCi/L)_{Peak} \qquad (11)$$

where

C_{mp} = the peak iodine-131 concentration in milk (pCi/L)

F_I = the air to milk transfer factor as defined previously

$\chi(t)$ = the iodine-131 air concentration as a function of time at the pasture (pCi/m^3)

T = the duration of the release

TABLE 4. Iodine-131 Inhalation Transfer Factor, F_I, and Percentile for Three Farm Sites Assuming Inhalation Pathway Only

Site	Location[1] (Mi)	Site	Location[1] (Mi)	$\bar{x}$[2] (pCi/m^3)	Ext. $\bar{x}$[3] For Farm (pCi/m^3)	IAC[4] ($pCi\text{-}s/m^3$)	Peak Conc. (pCi/L)	F_I ($m^3\text{-}L^{-1}\text{-}s^{-1}$)	Percentile[6] %
Becker	1.6/140°	Falmouth	2.3/159°	20.1	31	5.4E6	20	3.7E-6	34
Alwine	1.1/65°	Obs. Center	0.4/86°	20.3	4.8	8.3E5	8.5	1.0E-5	71
Fisher	3.5/294°	Goldsboro	1.6/253°	23.9	7.4	7.3E5[5]	7.4	1.0E-5	71

(1) The first figure represents miles from plant; the second figure, degrees from true north with respect to reactor.
(2) Sample averaged over 2 days (noon of 29 March to noon of 31 March)
(3) Extrapolated from $\bar{x}$ at reference measurement site
(4) Integrated air concentration (over 2 days)
(5) Extrapolated
(6) The intersection of F_I with the regression line of Figure 9

When $\chi(t)$ is not specifically known, an average may be used, $\bar{\chi}$, with the time T to obtain an approximate integrated air concentration. In that instance:

$$C_{mp} = F_I \bar{\chi} T \qquad (pCi/L)_{Peak} \qquad (12)$$

CONCLUSION

To conclude, we feel that the primary pathway of radioiodine to the cows' and goats' milk during the first week or so after the TMI 2 accident was through inhalation; a smaller portion may have been contributed by ingestion from sporadic grazing on the dry pasture available to the animals at the time primarily for exercise, especially after the middle of April when new pasture growth commenced.

RECOMMENDATIONS

In order to more easily determine the pathway to milk of radioiodine released into the atmosphere after an accident at a nuclear power plant, we recommend that the NRC consider the following enhancements to an applicant's monitoring program:

- Place iodine air sampling stations at locations of grazing animals. The filters of these samples should be analyzed at least daily.

- Pasture vegetation should be sampled and analyzed at these locations.

- Place real time thyroid counters on selected cows as soon after the accident as possible and have the data telemetered to a central command post at the site such as the Emergency Operations Facility (EOF).

In addition, we feel that these recommendations, if carried out successfully in the hours and days succeeding an accident, would further the validation of pathway models presently used by the NRC and the nuclear industry.

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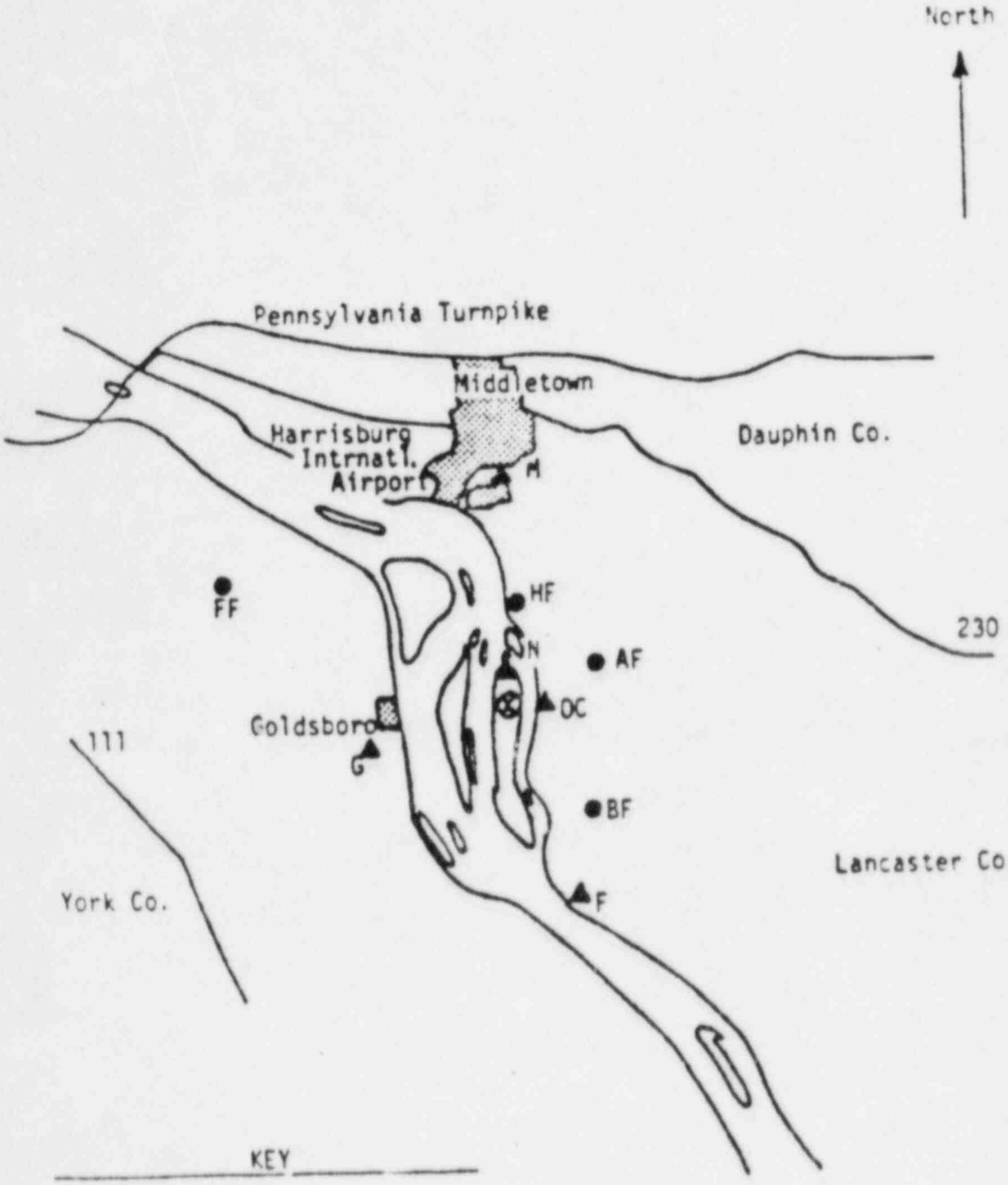
FIGURE 1. Locations of Farms and Iodine Air Sampling St tions



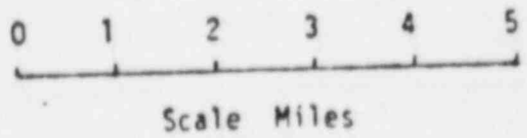
KEY

● Farm Sites:

AF - Alwine Farm
BF - Becker Farm
FF - Fisher Farm
HF - Hardison Farm

▲ Air Sampling Stations:

M - Middleton
F - Falmouth
OC - Observation Center
G - Goldsboro
N - North Weather Station



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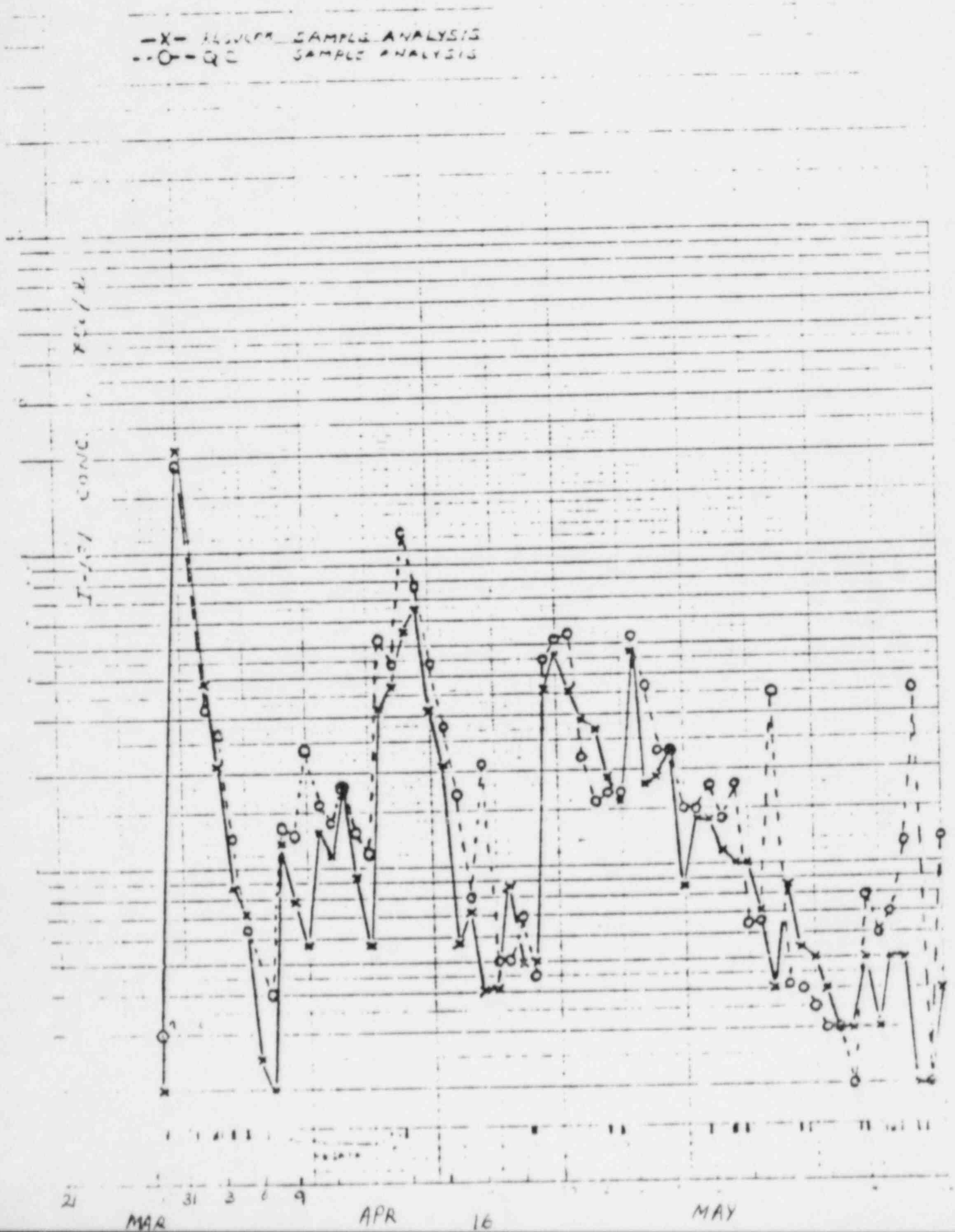


SAMPLE ANALYSIS
QC SAMPLE ANALYSIS
CONC.
21
31
3
6
9
MAR
APR
16
MAY

FIGURE 3. Hardison Farm (Goats') Milk Concentration

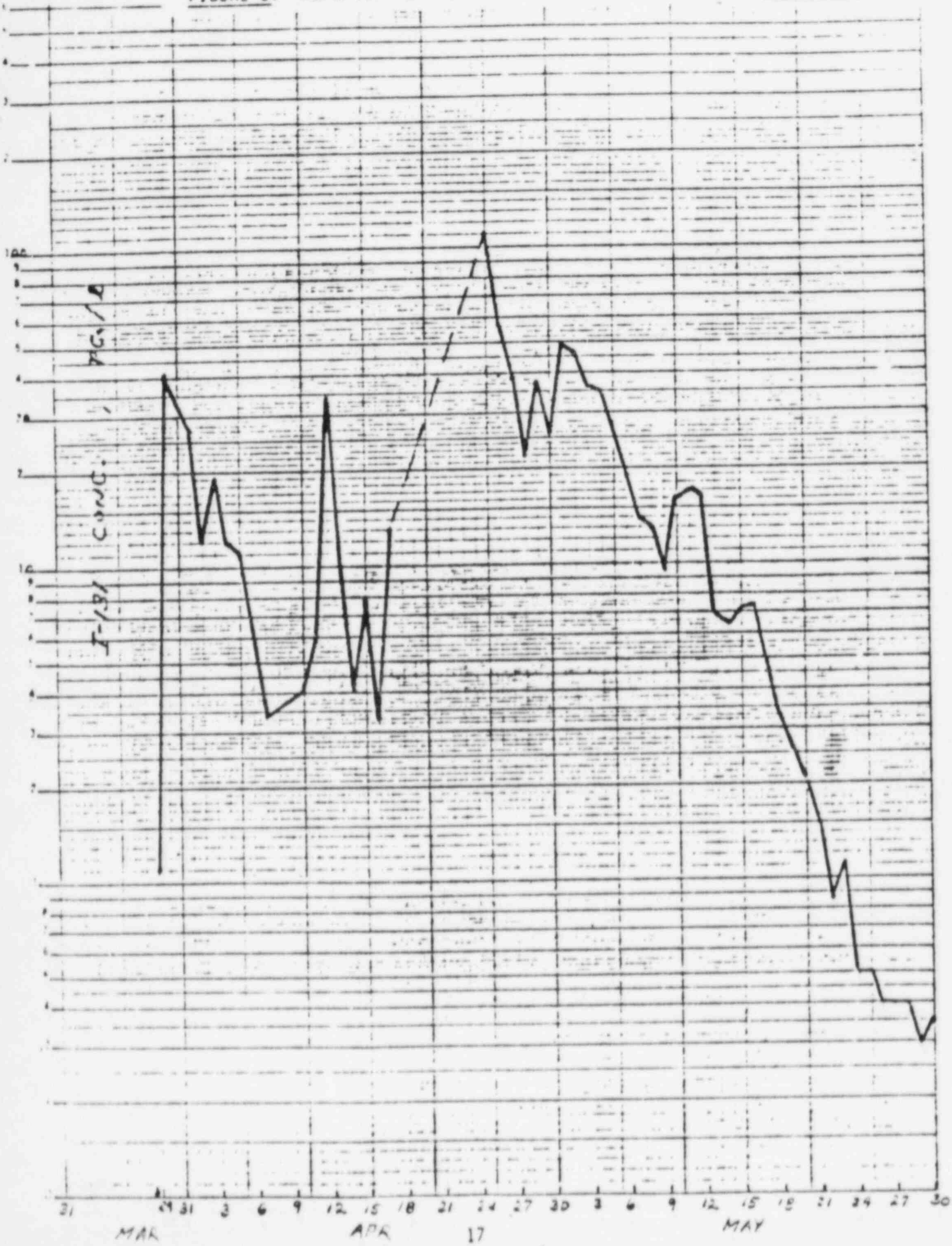


FIGURE 4. Alwine Farm Milk Concentration

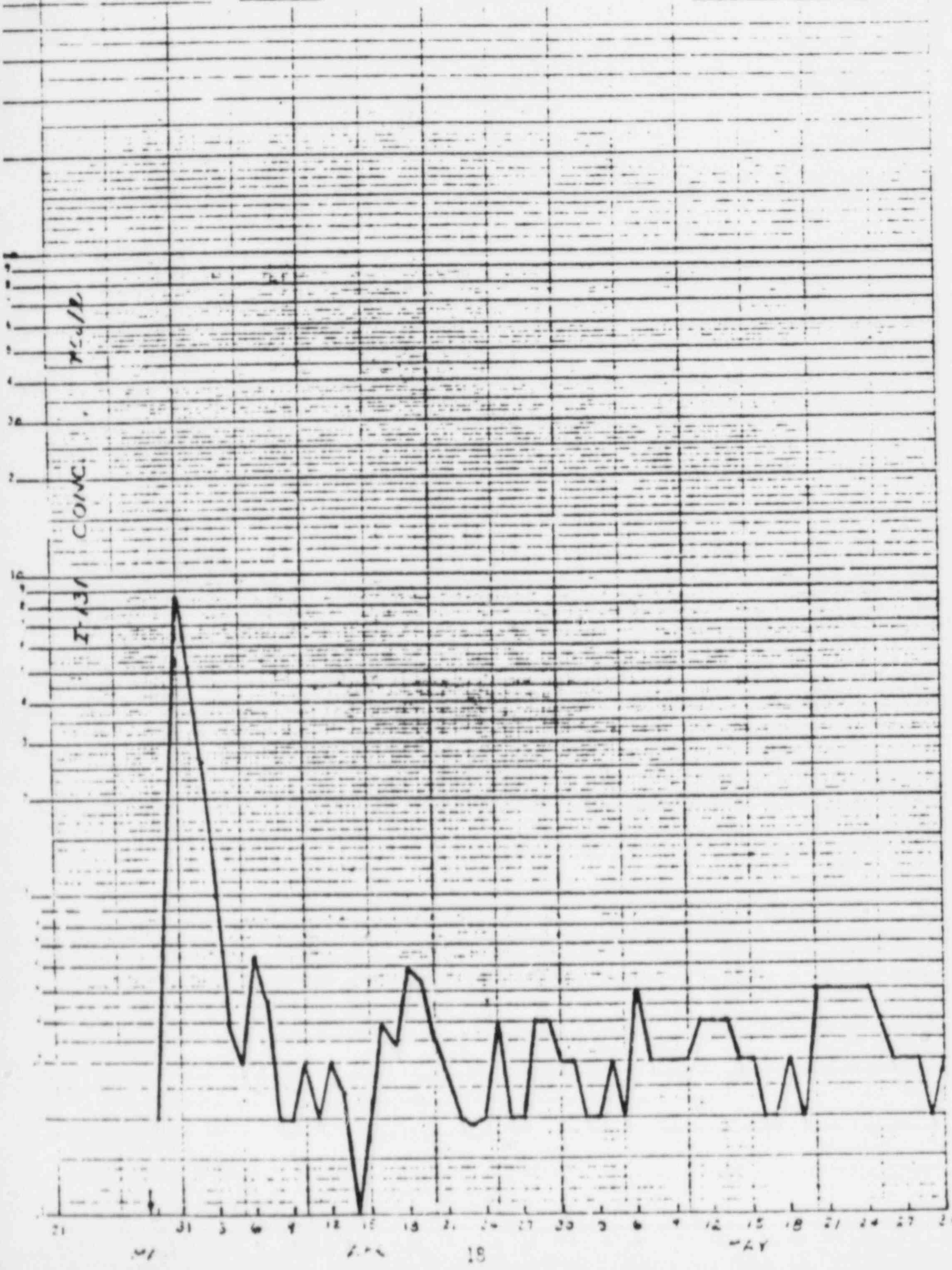


FIGURE 5. Fisher Farm Milk Concentration

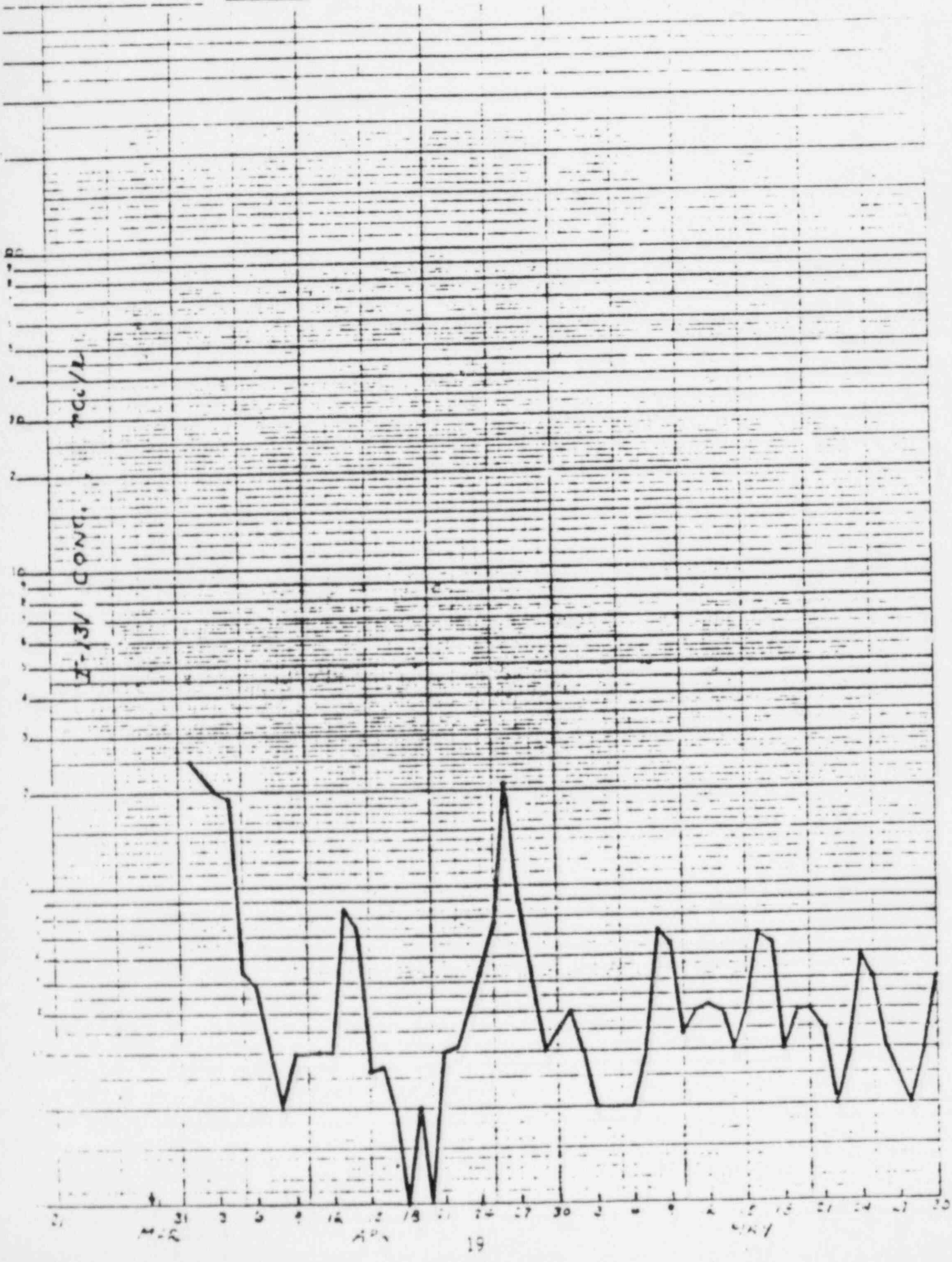


FIGURE 6a. Measured I-131 Air Concentration at Falmouth Substation. Input Function for Convolution

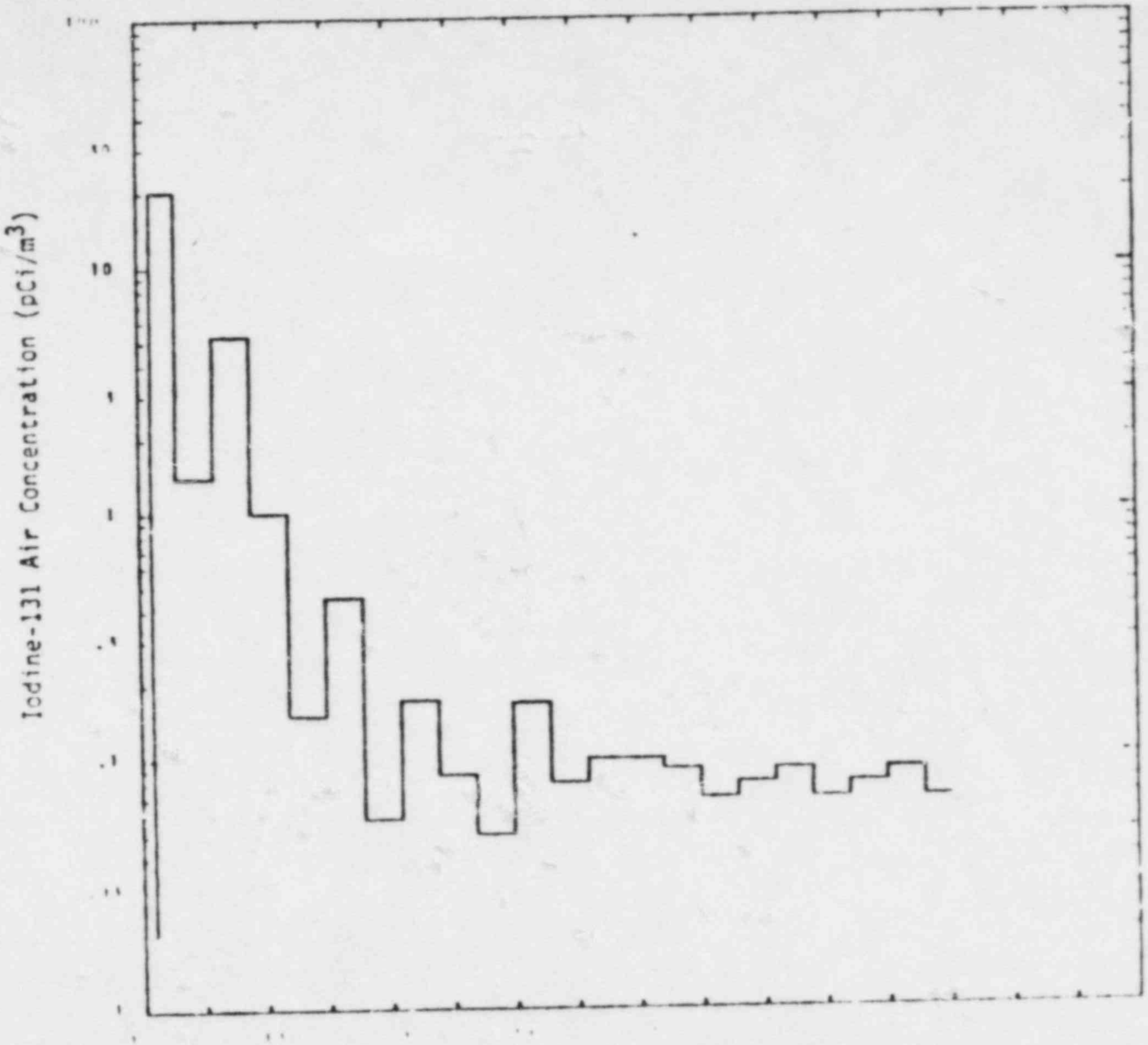


FIGURE 6b. Inhalation Pathway Output Function. Convolution of Falmouth Air Concentration with Equation 2.

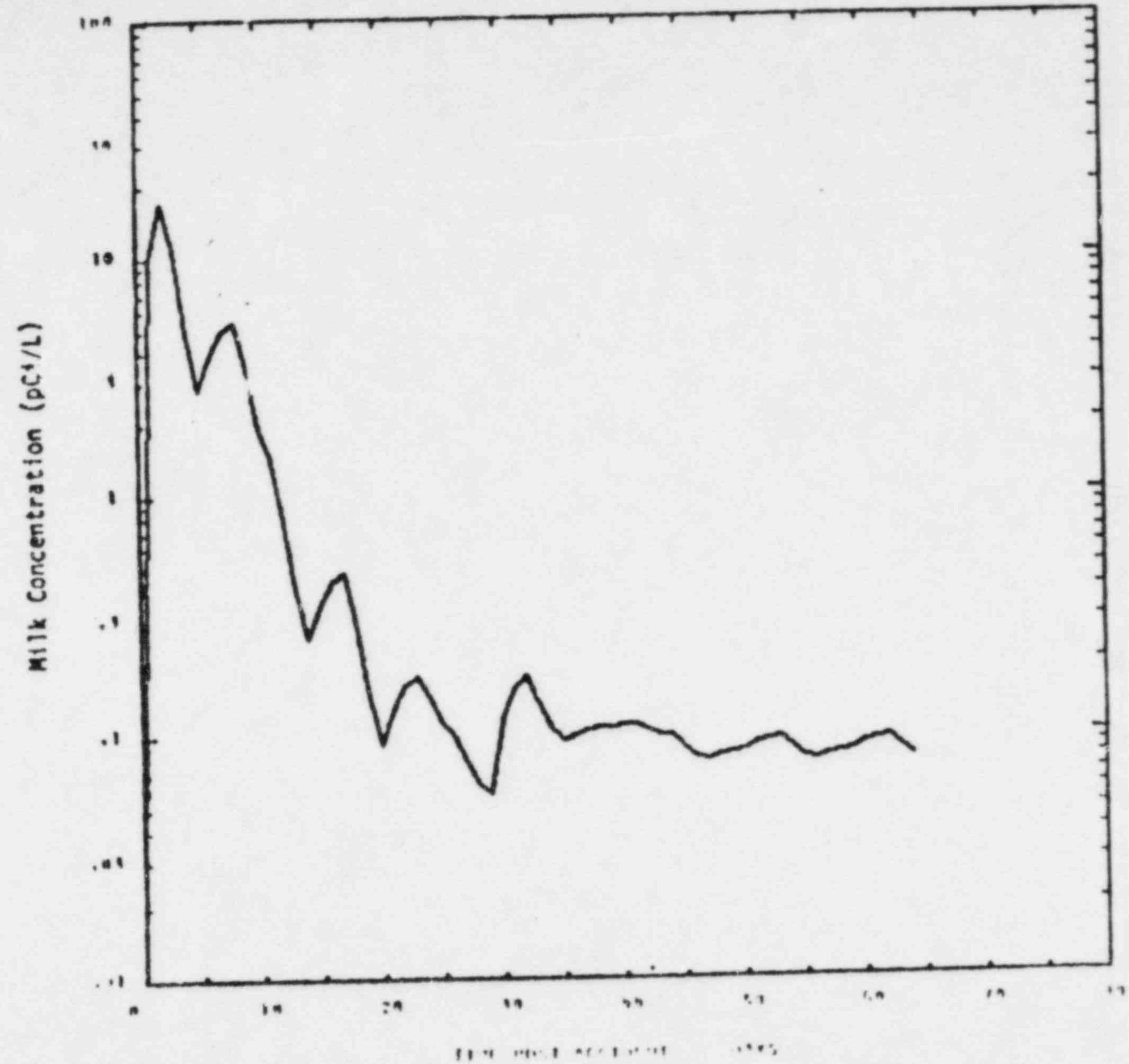


FIGURE 6c. Ingestion Pathway Output Function. Convolution of Falmouth Air Concentration with Equation 3.

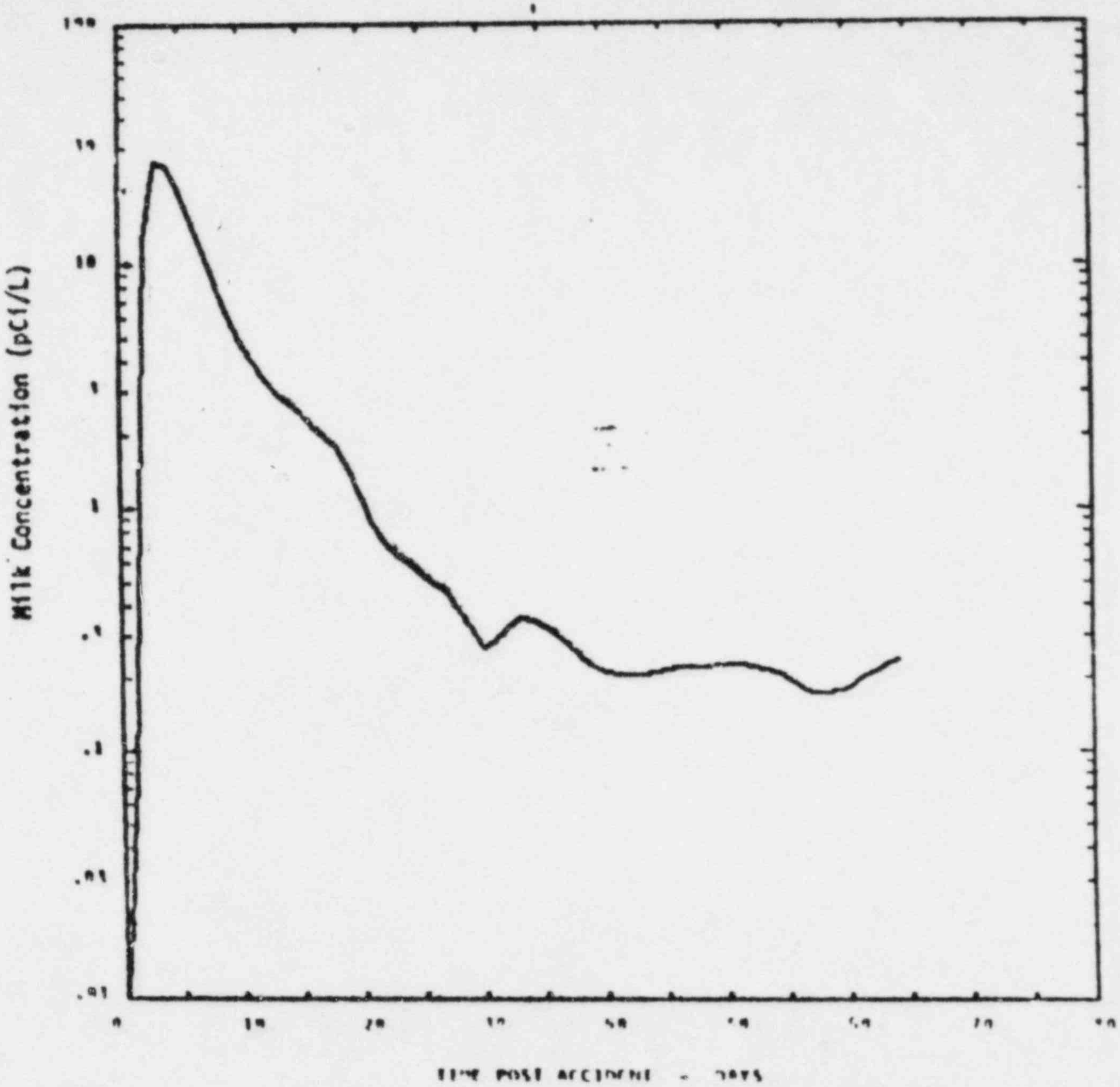


FIGURE 6d. Measured I-131 Milk Concentrations from Becker Farm

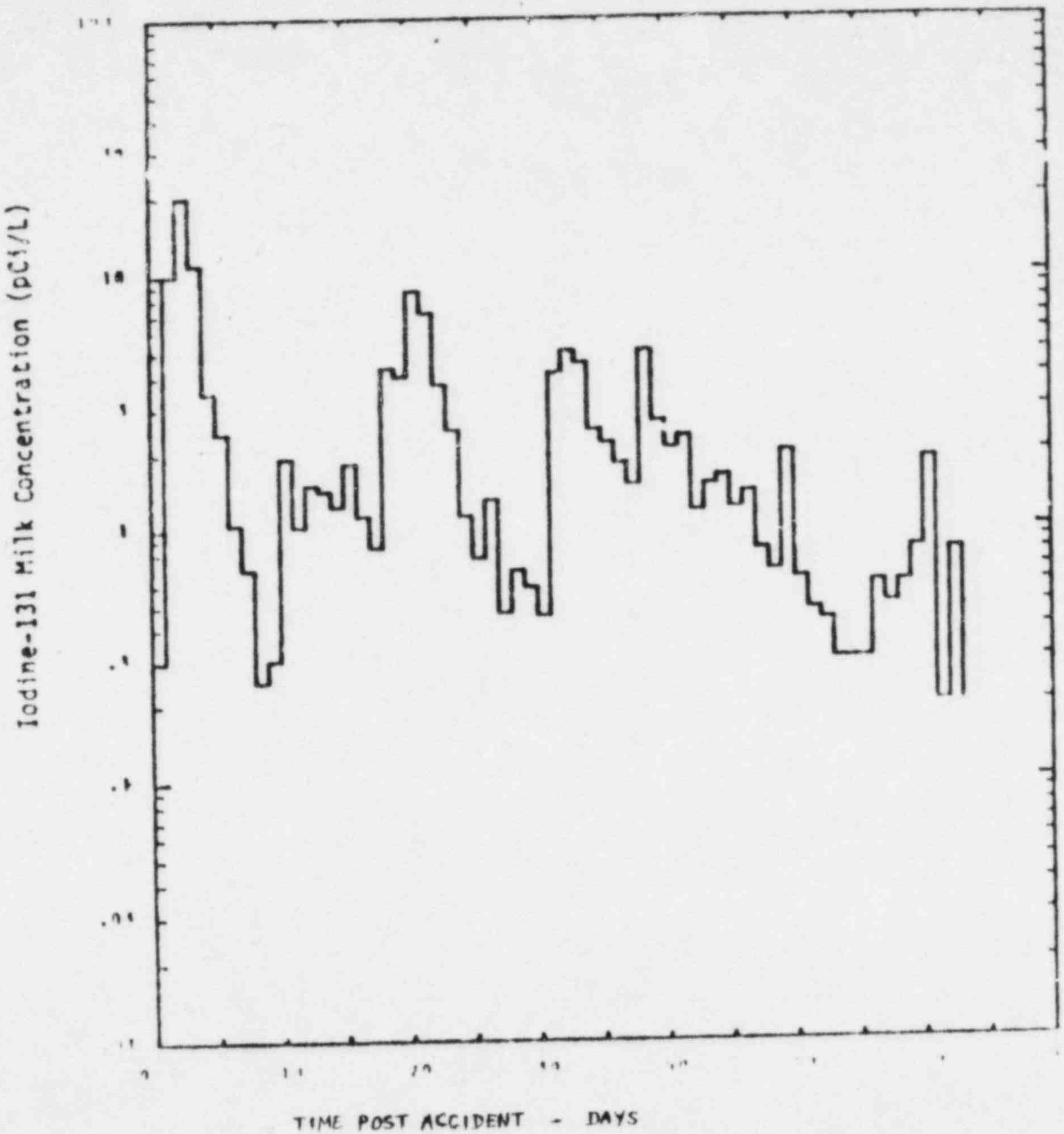


FIGURE 7a. Measured I-131 Air Concentration at Observation Center. Input Function for Convolution.

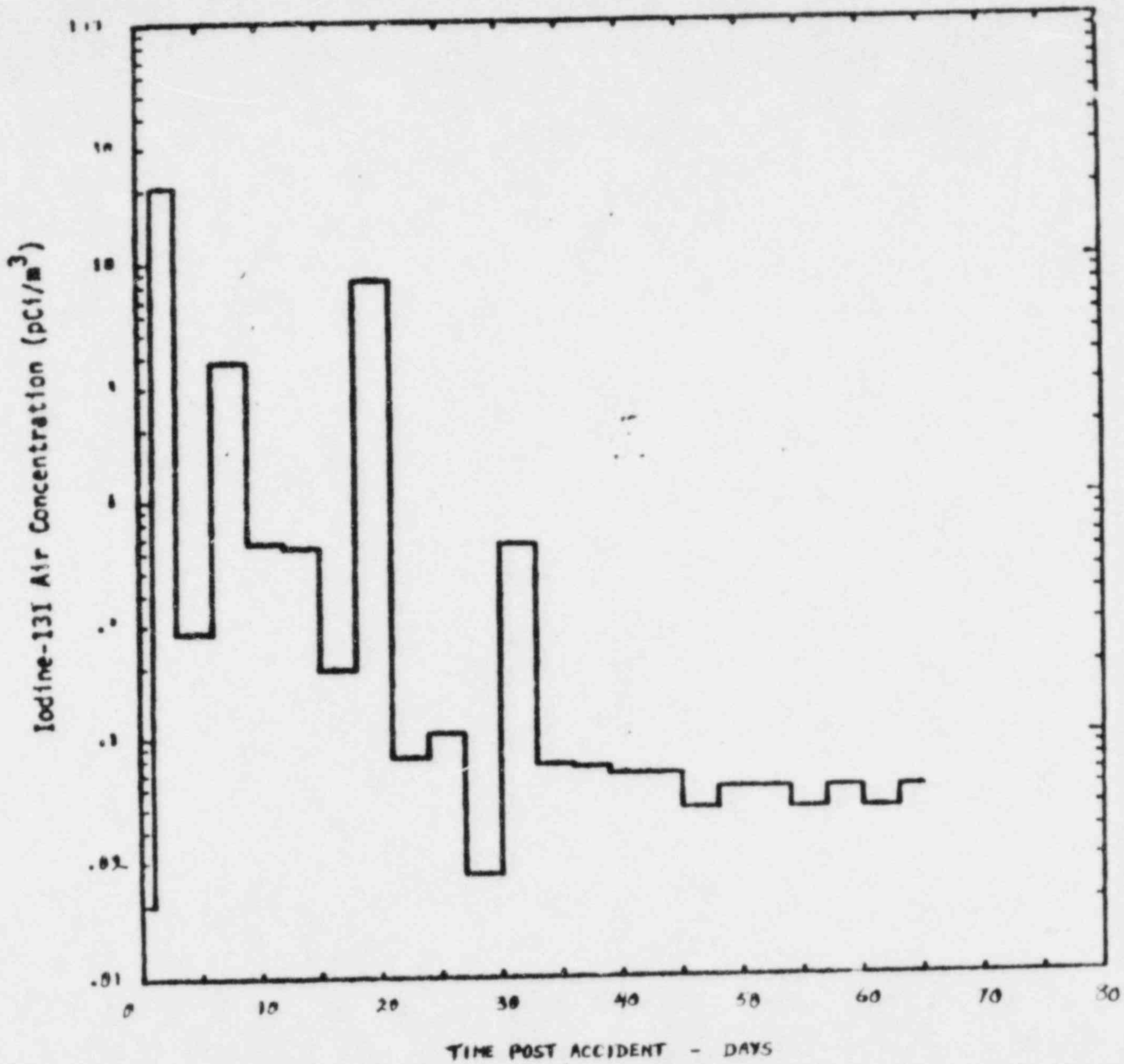


FIGURE 7b. Inhalation Pathway Output Function. Convolution of Observation Center Air Concentration with Equation 2.

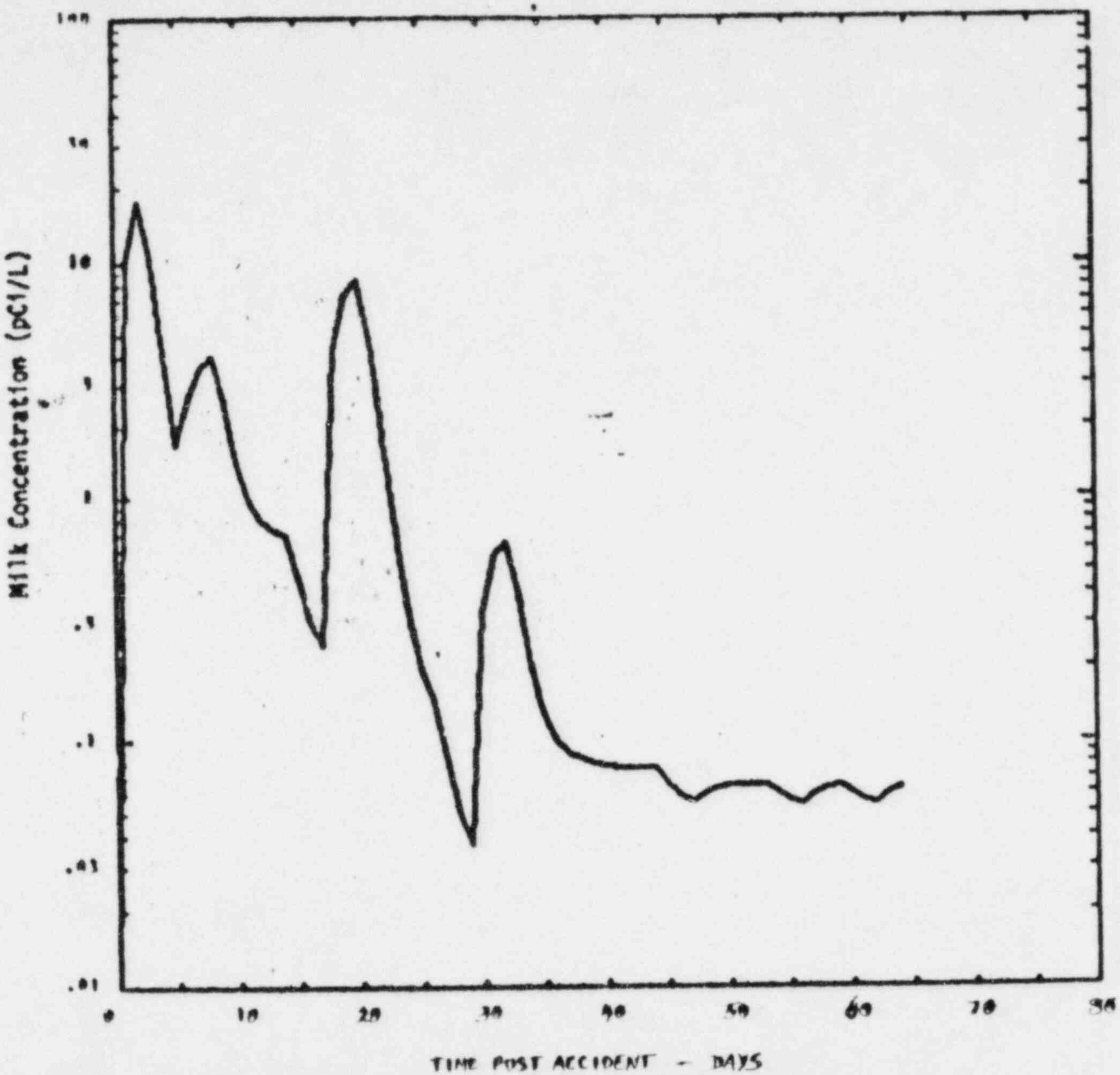


FIGURE 7c. Ingestion Pathway Output Function. Convolution of Observation Center Air Concentration with Equation 3.

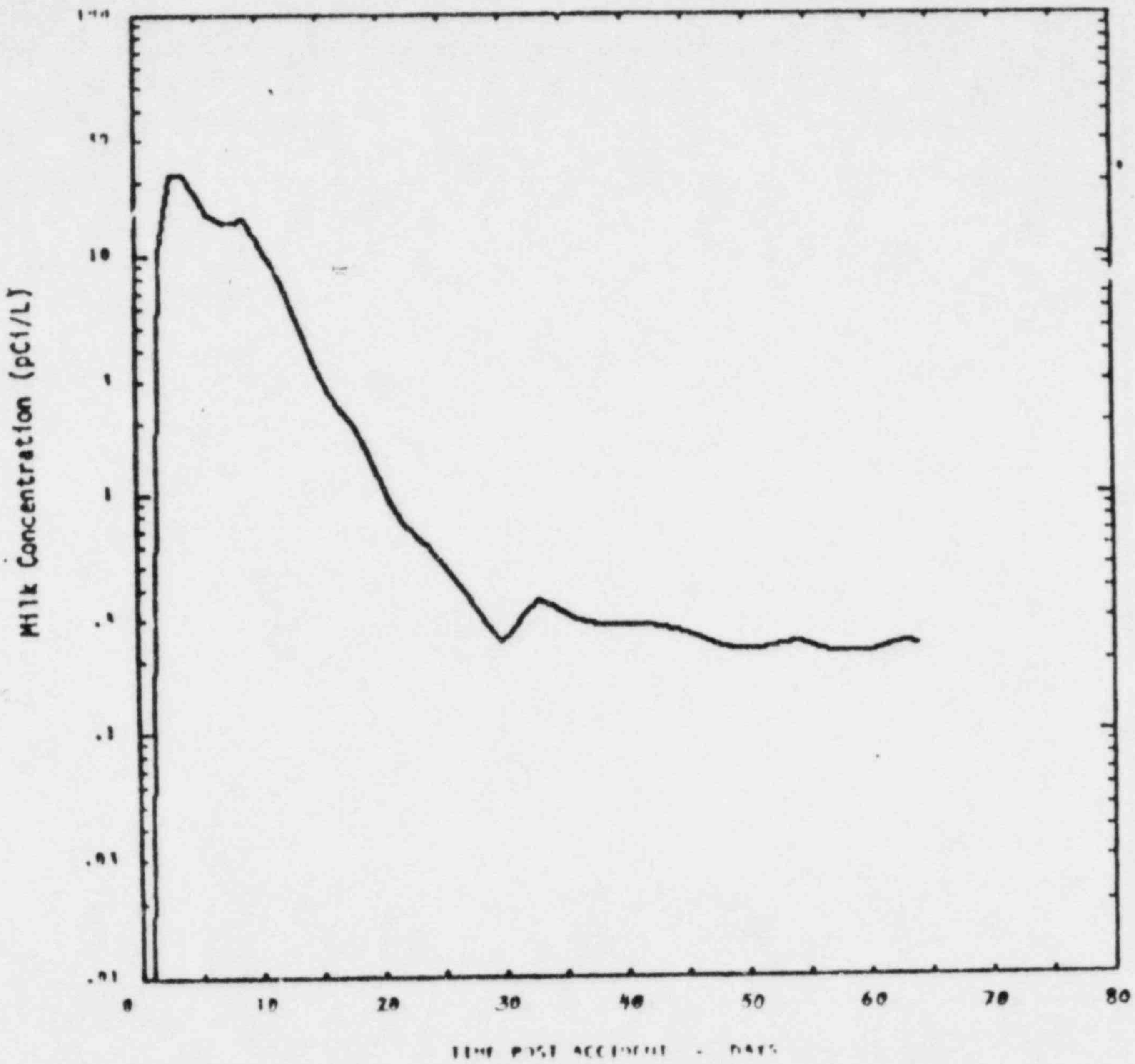


FIGURE 7d. Measured I-131 Milk Concentrations from Alwine Farm

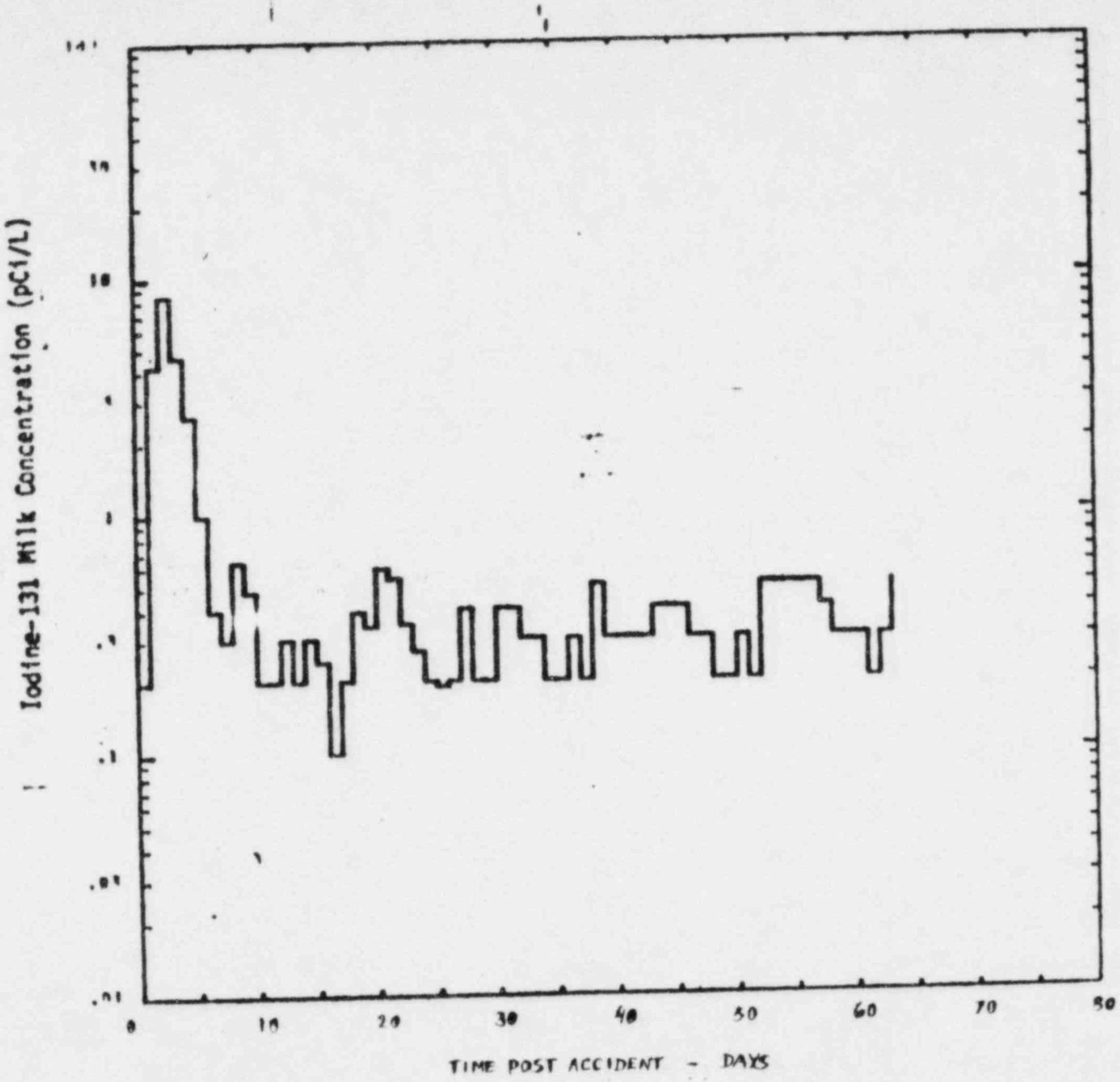


FIGURE 8a. Measured I-131 Air Concentration at Goldsboro Air Station. Input Function for Convolution.

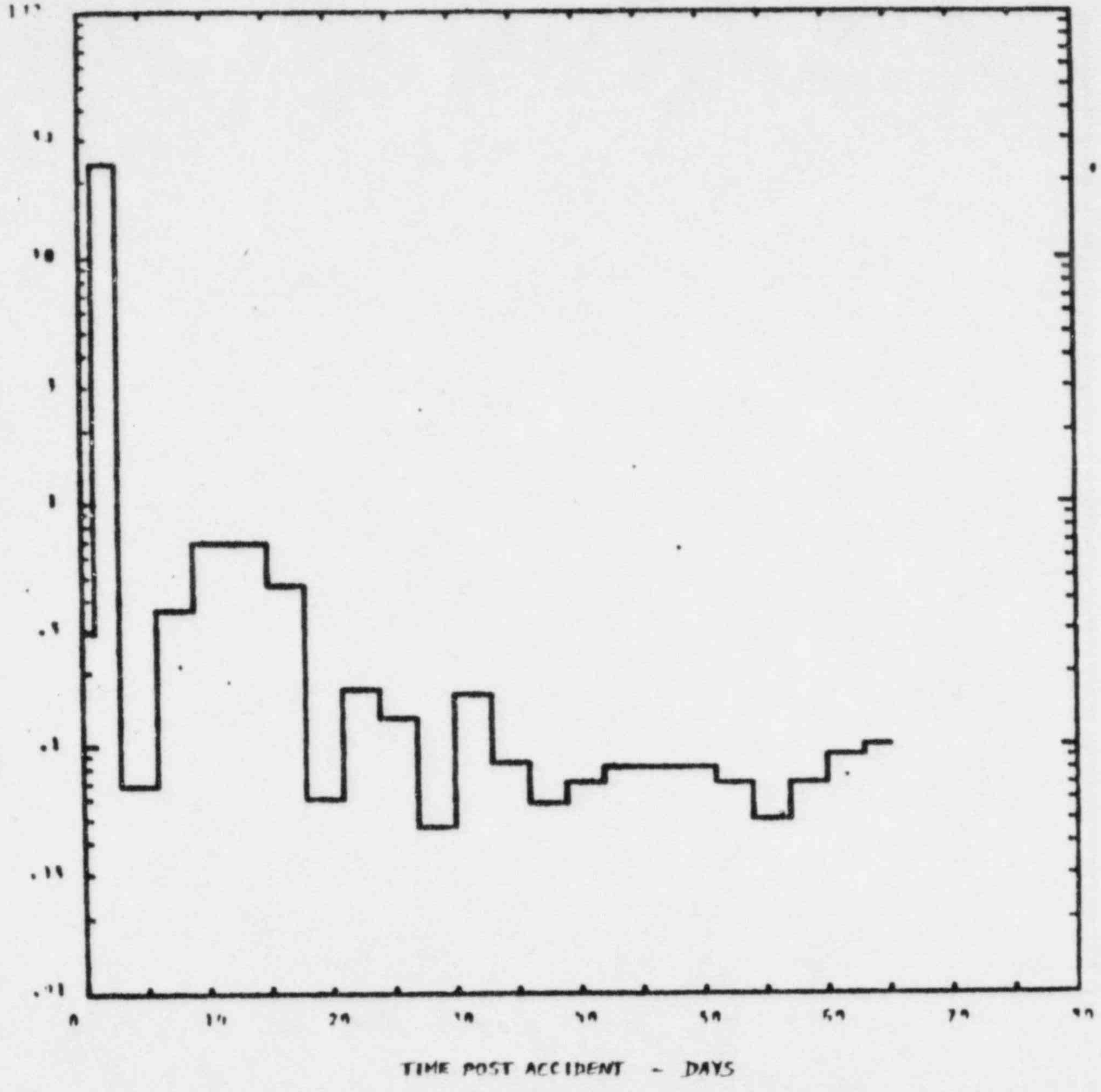


FIGURE 8b. Inhalation Pathway Output Function. Convolution of Goldsboro Air Station Air Concentration with Equation 2.

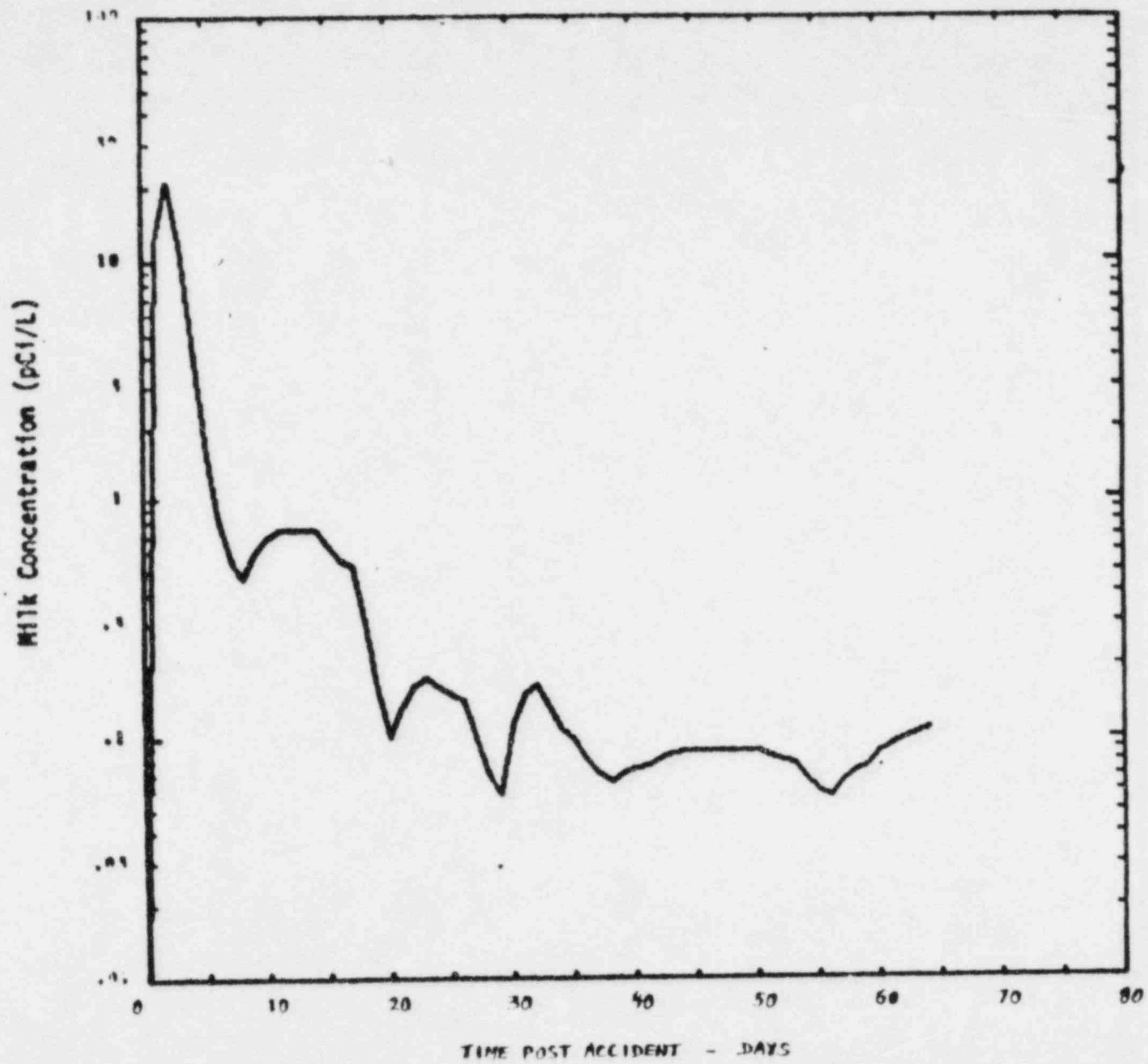


FIGURE 8c. Ingestion Pathway Output Function of Goldstone Air Station Air Concentration with Equation 1

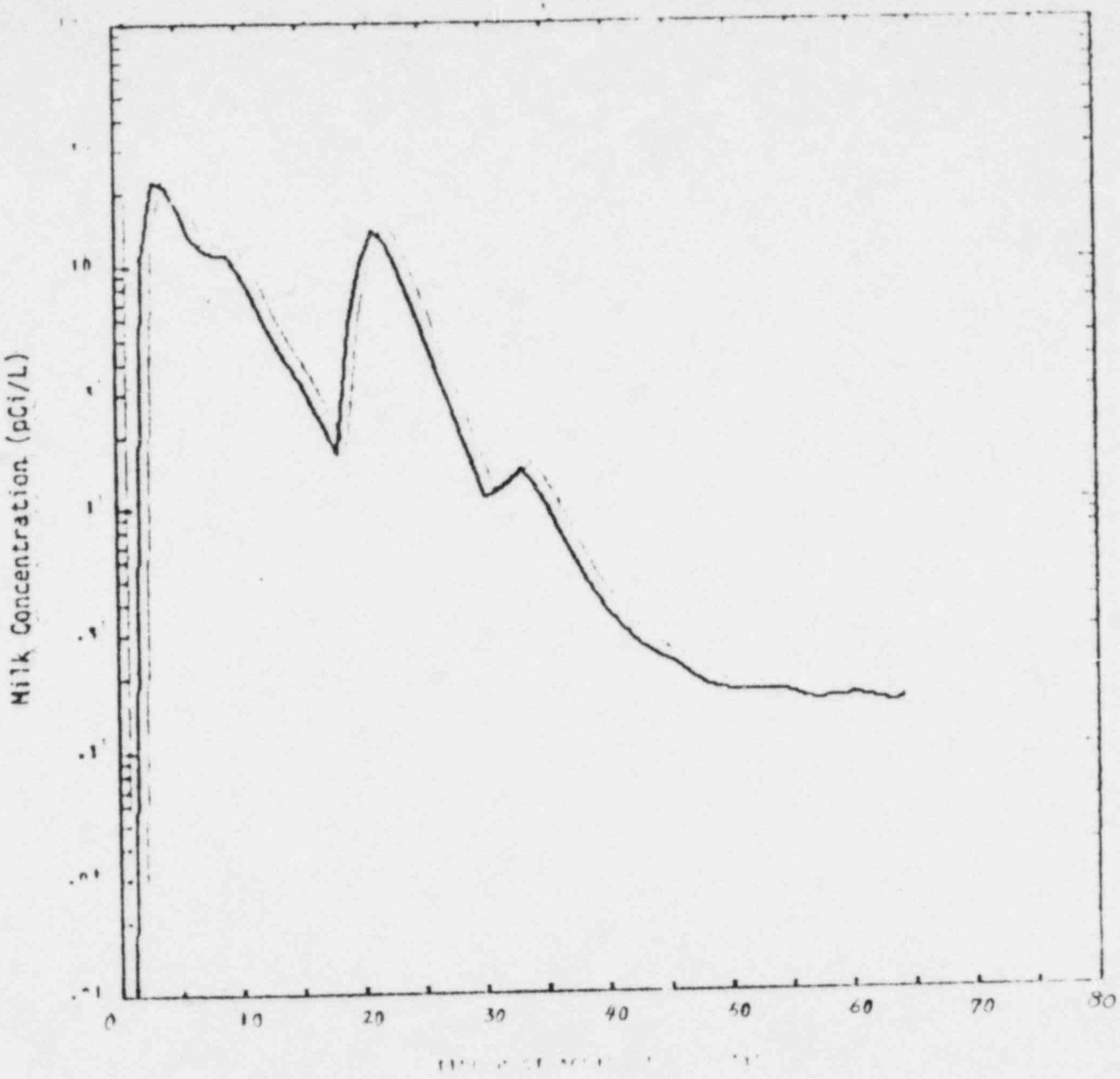
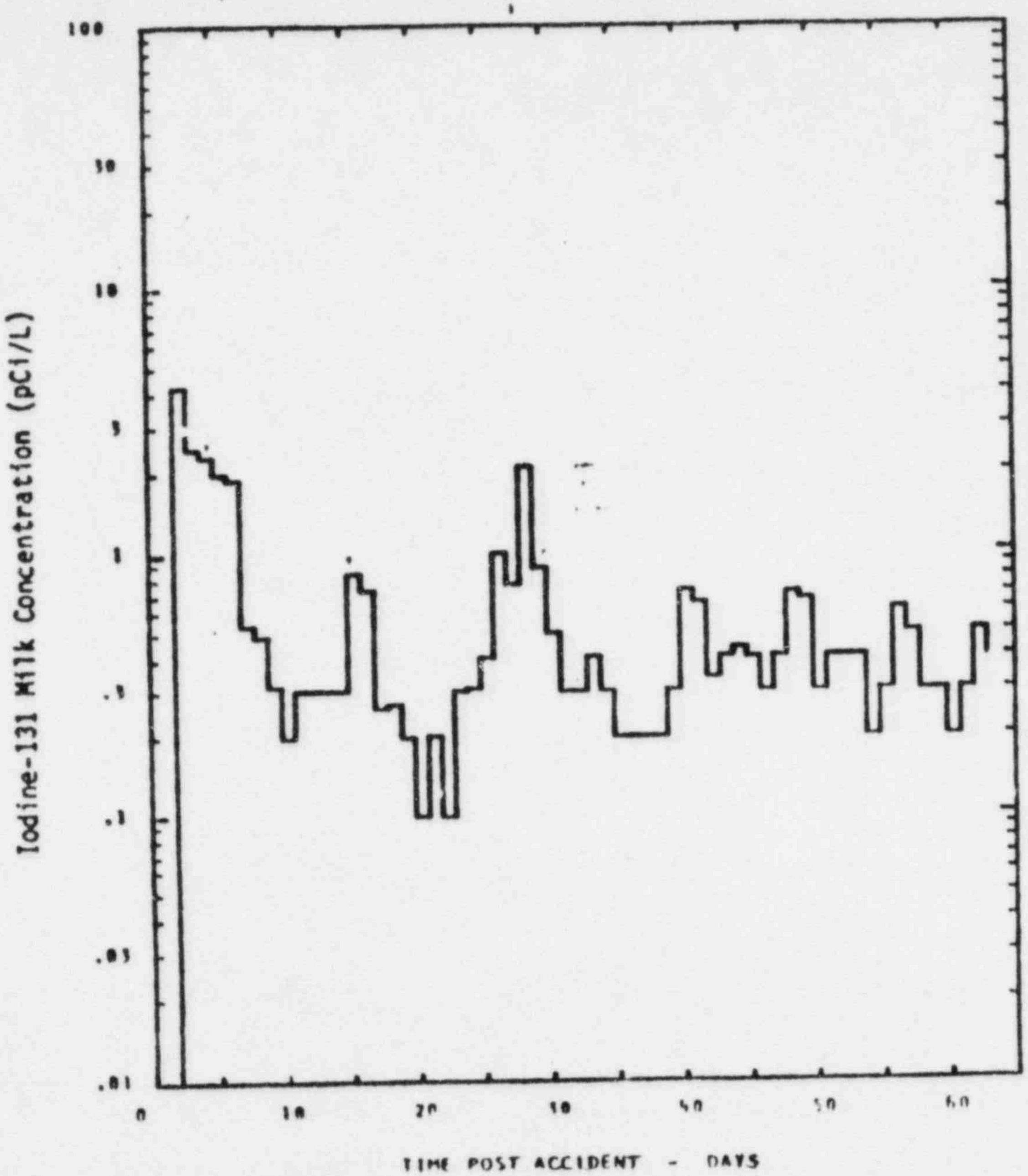


FIGURE 8d. Measured I-131 Milk Concentrations from Fisher Farm



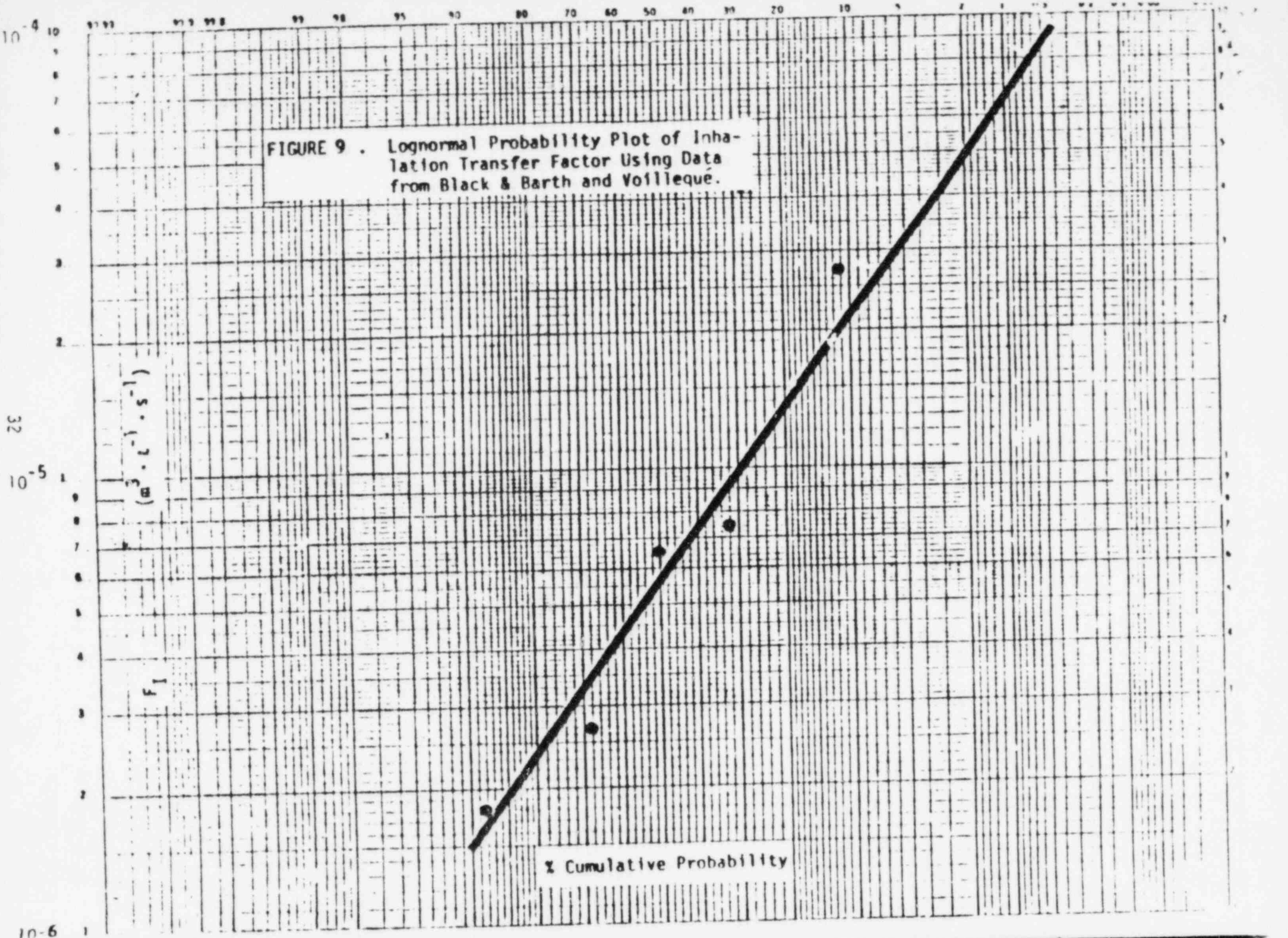


FIGURE 9 . Lognormal Probability Plot of Inhalation Transfer Factor Using Data from Black & Barth and Voillequé.