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Nuclear Regulatory Commission
Before the Atomic Safety and Licensing Board

In the Matter of Three Mile Island Unit 1
Metropolitan Edison Company, et. al
Docket 50-289

Testimony of Bruce Molholt Regarding Aamodt Contention 4 (EP-1)

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THE BIOLOGICAL EFFECTS OF THE ACCIDENT AT TMI

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The crowning achievement of medical science over the past two decades has been to understand the molecular basis of many chronic diseases. Especially in the realm of genotoxicity, we now understand the cause and effect relationships between exposures to carcinogens, mutagens and teratogens and the development of cancers, birth defects and fetal malformations in human populations. Except for the latter, which may be apparent immediately after birth, these chronic genotoxic effects may take a generation or more to become manifest. Therefore, after any event which releases genotoxic agents, such as the commercial nuclear accident at Three Mile Island Nuclear Station Unit 2 on March 28th, 1979, it is premature, at any time before the passage of one generation to state "Noone died (as the result of the event.)"

Genotoxic events take place in two distinct phases: genotypic and phenotypic. Genotypic changes are actual alterations of chromosomes such that the genetic code is altered leading to mutations and consequent alterations in behavior patterns of afflicted cells. Phenotypic changes are the changes we see, that is the observable manifestation of what has happened at the chromosomal level. These observable phenotypic changes may take decades to become apparent. If the genotype of a germline cell has been altered, a potential birth defect has been created which will become manifest in the next generation, or possibly in several generations. On the other hand, if a somatic cell has been altered, it may develop into a cancer, which development may take 20 - 40 years to become manifest. Only one stage of human existence is "immediately" responsive to genotoxic effects, the fetal stage when the individual grows from an initial single cell, the zygote, to several trillion cells in the newborn.

These considerations make insults to the fetus or newborn especially germane in consideration of human health effects of the accident at TMI. We will not know carcinogenic or mutagenic effects for another generation. We must look to fetal and neonatal populations at present for some barometer of human health effects. As we shall see, the admonition that "Noone died at TMI" may be not only premature considering the chronic nature of radiation-induced adverse health effects, but also ignorant of statistically significant increases in infant mortalities and neonatal events at the time of the accident.

In this breakdown of potential adverse health effects emanating from the TMI-2 reactor at the time of the accident, we will examine the following three types of evidences:

1) Examination of infant mortalities and neonatal hypothyroidism in people residing near TMI at the time of the accident.

2) Examination of animal data collected from samples taken near TMI at the time of the accident.

3) Estimates of radionuclide releases from the reactor at the time of the accident.

Although 187 radionuclides were manufactured in TMI-2 during its 90 days of operation, these considerations will only concern radioisotopes of the element iodine.and of krypton-85.

The Pennsylvania Department of Health (DOH) has reported infant mortalities within 5 and 10 miles of the TMI reactor for the April through September intervals in 1977, 1978 and 1979. As may be seen from Table 1, these infant mortality statistics show a significant increase in 1979, the year of the TMI accident, as compared to the two previous years. There were approximately 14 excess infant mortalities in April-September 1979 within 10 miles of the TMI reactor. It is estimated that of the 31 infant mortalities within the 10 mile radius during that period that 25-26 were <u>in utero</u> at the time of the accident. It would be interesting to know the thyroxin and thyroid stimulating hormone levels of these infant mortalities at birth.

The State DOH has repeatedly attempted to dismiss these infant mortalities as due to the accident at TMI. If one contrasts DOH and national infant mortality statistics obtained from the Department of Health and Human Services (HHS), 85 infant mortalities appear to be missing for 1979 from the DOH statistics.

Similarly, there were increased cases of neonatal hypothyroidism in the area of the Three Mile Island nuclear reactors following the accident in March 1979 at Unit 2. Neonatal hypothyroidism became checked as a birth defect in every Pennsylvanian newborn in the summer of 1978. Statistics are thus available for all births recorded during the 9 months before and after the accident at TMI. Comparison of these cases (Figure 1) shows that a cluster of new cases occurred in seven counties downstream or downwind from TMI at the time of the accident. These comparisons are summarized in Table 2. As control populations, the counties west of Harrisburg, comprising geographically the majority of the State, showed 7 cases of neonatal hypothyroidism before the TMI accident

and seven cases after. Similarly, Philadelphia, the five county area, showed 6 cases before the accident and six cases after. Of the 10 excess cases in Pennsylvania following the accident, all occurred in Lancaster, Lebanon, Schuylkill, Berks and Lehigh Counties. Including the cases in Dauphin and Carbon Counties which occurred prior to the accident, these counties had 4 cases of neonatal hypothyroidism during the 9 months before TMI and 14 cases after, a statistically significant increase at the 95 per-cent confidence level. The six cases of neonatal hypothyroidism in Lancaster County occurred in 2700 live births, whereas the expected frequency is 1/4300 live births, 1/10th the actual value seen. Neonatal hypothyroidism may be due to many different insults to the developing thyroid gland, including contamination with radioactive iodine (iodine-131, iodine-132). As we will see below, these two isotopes of iodine were released in significant quantities from the reactor at TMI during the course of the accident. One result of hypothyroidism in the developing fetus is insufficient cranial development which manifests itself as a form of cretinism leading to mental retardation of afflicted individuals.

That sufficient iodine-131 was released from the TMI-2 reactor during the accident in March and April of 1979 has been shown in the studies of the Department of Biology at Millersville State College which employed wild field vole populations in the vicinity of Three Mile Island. Field voles captured within 1.9 km of the reactor had maximal iodine-131 contents of 4 nCi/g, adjusted to April 9th. These iodine-131 levels have been substantiated in rabbit and field vole thyroid samples from the same period by studies conducted by the University of Missouri. These three sets of studies are summarized in Table 3.

It is clear from the above results that sufficient iodine-131 contaminated the environment around TMI to induce hypothyrodism <u>in utero</u>. Official estimates of iodine-131 release were 14-15 curies. However, there are several arguments which indicate that the 4 nCi/g I-131 levels are minimal (Table 4). Furthermore, Takeshi has estimated that the iodine-131 releases are 5,100 to 64,000 curies (Table 5). Hence, there is supportive evidence from animal studies and from noble gas-to-iodine-131 releases that sufficient iodine-131 was released from TMI-2 to induce hypothyroidism in humans.

Field voles have not only been employed for monitors of I-131 contamination, but also for monitors of krypton-85 contamination during the venting of the TMI-2 containment facility June 30-July 14th, 1980. It had been predicted that krypton-85 would accumulate in fat tissues due to its lipophilicity. Studies conducted at Millersville State College indicate that Kr-85 accumulated in testicular fat of field voles captured during the venting operation.

Although krypton-85 as a noble gas is chemically inert, it is by no means biologically inert. Due to its lipophilicity, it also accumulated in breast tissues of humans exposed to the gas during the intentional venting of the TMI-2 containment facility. Figure 2 shows the track length of beta particles emitting from krypton-85 superimposed upon micrographs of human breast tissue. It is obvious that target ductal and lobular epithelial cells are well within the range of beta decays emanating from fatty deposits in human breast tissue. These considerations are compounded by the fact that ductal and lobular cells of the human breast are among the most radiosensitive of human tissues.

Figure 3 summarizes early stages in carcinogenesis at the cellular level. Cells become cancerous after undergoing two separate genetic events (initiation) followed by promotion steps which include cellular differentiation and proliferation. Long-lived radionuclides may trigger both genetic events in the same sub-population of cells, making them more insidious carcinogens than chemical compounds. Local tissue damage by radioactive decay may also promote carcinogenesis through ablation and restoration of damaged tissue. Hence, radionuclides are ultimate carcinogens, able to initiate both required genetic events and the first step of promotion.

The steps in the sequence of Figure 3 are cumulative, that is they need not follow each other closely in time. Exposures to low-level radiation at yearly intervals could suffice to mitigate each step. Hence, populations under duress from local discrete episodes of radionuclide pollution enhance their carcinogenic risk at each subsequent release of radionuclides. In addition, since radiation is immunosuppressive, and 99 percent of all budding tumors are rejected by the cellular immunity system, further radiation exposures will allow even more tumors to break through this immunologic barrier. These considerations are especially germane to the population residing near TMI who have received genotoxic insults from radiation periodically as a result of the accident in March and April 1979, the venting of the containment facility in June and July 1980 and who will continue to receive further exposures to radionuclides as a result of subsequent phases of the cleanup operation.

Summary

There is reason to suspect that adverse human health effects have been initiated both by the accident at Three Mile Island and by the intentional venting of krypton-85 gas the following summer. Data supporting this contention derives from the following observations which were contemporaneous with the accident:

1) Within a 10 mile radius of TMI in the 6 months following the accident there were 31 infant mortalities, whereas 17 were expected.

2) Within the seven county area downwind or downstream from TMI at the time of the accident, neonatal hypothyroidism cases increased from 4 to 14 in equivalent 9 month periods.

3) Sufficient iodine-131 was found in wild animal populations (field voles and rabbits) to account for hypothyroid induction.

4) Iodine-131 releases from TMI were 5100 to 64,000 curies during the course of the accident.

These results concentrate on the fetal period of human existence since it is by far the most radiosensitive (Figure 4). It will be 20 - 40 years before we can know of cancers and birth defects induced as a result of the accident.

Similar evidence has come from potential adverse health effects which may have been induced during the intentional krypton venting. Field voles collected during that time displayed krypton-85 in their testicular fat. Beta particles leaving these krypton-85 atoms could induce birth defect-causing mutations in local spermatogenic tissues. Similarly, breast cancers could have been induced by beta decay from krypton-85 atoms trapped within fatty deposits of the human breast.

The population residing near Three Mile Island has been exposed to genotoxic radionuclides upon at least two separate occasions and are predisposed to further radionuclide-induced carcinogenesis or mutagenesis.

Table 1

INFANT MORTALITIES WITHIN 5 OR 10 MILES OF TMI

5 mile radius

	1977	1978	1979
no. deaths	3	1	7
rate/1000 births	6.7	2.3	16.1

10 mile radius

	1977	1978	1979
no. deaths	20	14	31
rate/1000 births	10.5	7.2	15.7

1977-78 average v. 1979

	1977-78	1979
no. deaths, 5 mi.	2	7
no. deaths, 10 mi.	17	31 *

*Difference significant at $p < 0.05$.

Table 2

NEONATAL HYPOTHYROIDISM

During the nine months before and after the accident

Geographic Area	Before	After
Pennsylvania west of Harrisburg	7	7
Five county area of Philadelphia	6	6
Rest of Pennsylvania	4	14 *
Total	17	27
Downwind TMI (Dauphin, Lebanon, Berks, Schuylkill, Lehigh, Carbon)	2	8 *
Downstream TMI (Lancaster County)	2	6

* Difference significant at $p < 0.05$.

Table 3

SUMMARY OF ANIMAL STUDIES - IODINE-131 IN THYROIDS

	Site	Number	Mean (per thyroid)	Max (per thyroid)	Mean (per gram*)	mrem
	Field Voles					
April 6-16th	12.9 km NE	20	0.0 ± 0.8 pCi	-	-	
	2.3 km E	22	2.2 ± 1.1	-	723 pCi	82.5
	1.9 km NE	18	5.6 ± 1.2	11.4	1866 (4/9)	210
					*assuming 3 mg/thyroid	
April 25th	0.8 km E	1			0.53 (4/25) (1500?)	
	Rabbits					
April 24th	1.6 to 4.8 km NE	5?*			644 (4/9) 161 (4/24)	72.5
	*composite sample not counted until May 8th					
	Cow Milk				Max (/ml)	
					36 fCi	
	Goat Milk				41	
	Isopleths					
	1.9 km NE					18

1866 pCi/g in field vole thyroids is conservative:

1) Maximal values were 3800 pCi/g (3.8 nCi/g).
2) Thyroids may weigh less than 3 mg.
3) Calculations are to April 9th when maximal I-131 releases were March 28 - April 1st.
4) Only I-131 calculated (early I-132 mrem may have exceeded I-131, they are synergistic)
5) Fetal thyroids 10-200 X affinity for iodine.

Table 4

NEONATAL HYPOTHYROIDISM AND IODINE-131 AT TMI

CONSIDERATIONS

1. 4 nCi/g max in voles, 1.9 km NE
2. ---> 8 nCi/g (4/9 ---> 4/1.79)
3. ---> 80 nCi/g from equivalent I-132 (I-132 9X I-131)
4. ---> 16000 nCi/g because fetal thyroid 10-200 X uptake I-131 (16 μCi/g)
5. 50% suppression of rat thyroid with 13 μCi/g I-131
6. therefore, potentially hypothyroid-inducing dose released at TMI

Table 5

RELEASE OF IODINE-131 AT TMI (Takeshi)

A. Calculation from I-131 and noble gas releases on April 20th

1. I-131 release rate = 1.4 μCi / sec
2. noble gases released at 4,700 μCi / sec
3. I-131 / noble gases = 1 / 3400
4. Extrapolation to March 28th: I-131 / noble = 1 / 8800
5. Total noble gases released = 45×10^6 Ci
6. Therefore, total I-131 released = $45 \times 10^6 / 8800$

   = 5100 Ci

B. Calculation from NUREG-0600 (NRC Office of Inspection and Enforcement, Aug. 1979 "Investigation into TMI accident")

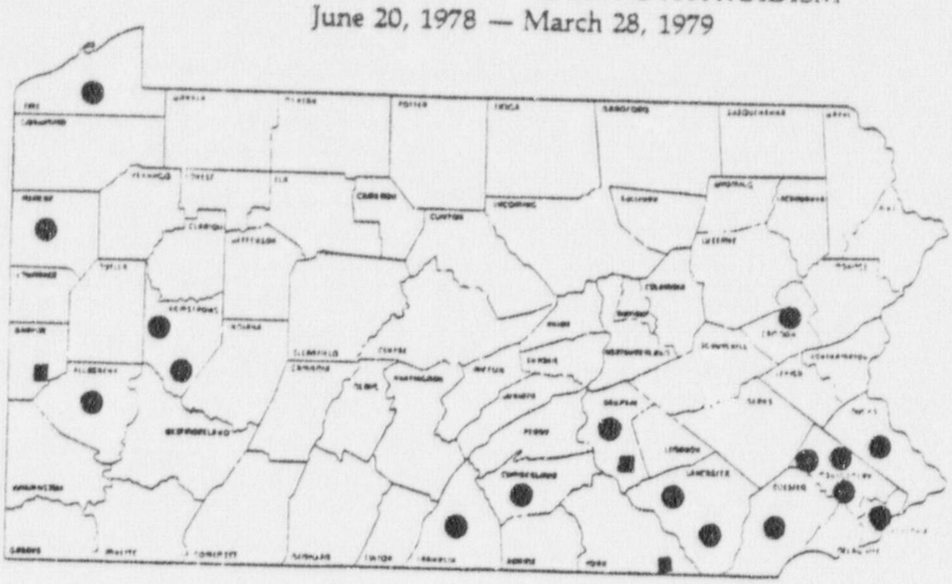
1. I-131 releases began 7 a.m. March 28th
2. I-131 to noble gas release ratio then = 1 / 700
3. Maximal iodine releases occurred within a few hours
4. Therefore, I-131 release = $45 \times 10^6 / 700$

   = 64,000 Ci

C. Admitted release of I-131 = 14-15 Ci

Figure 1

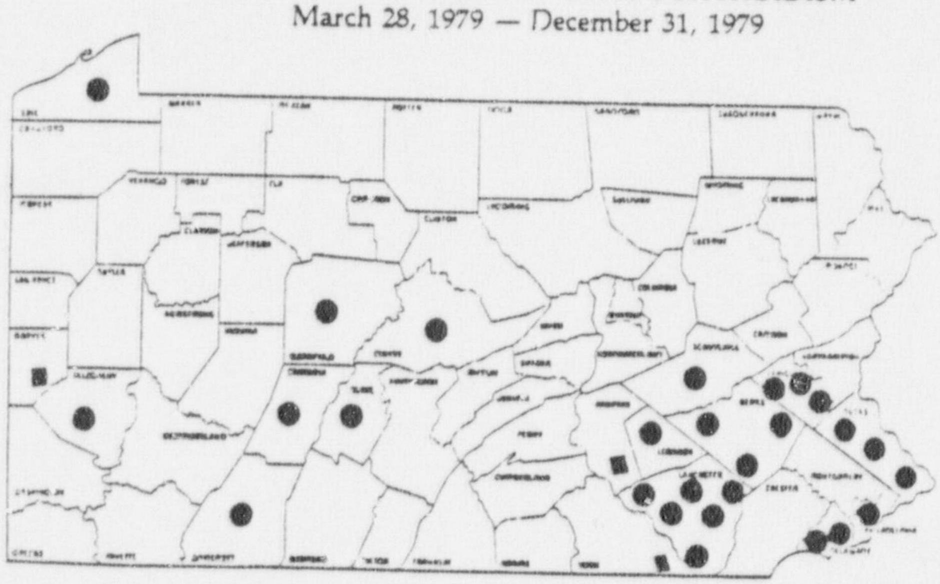
INCIDENCE OF NEONATAL HYPOTHYROIDISM
June 20, 1978 — March 28, 1979



● Neonatal Hypothyroidism Case
■ Nuclear Power Plant

PENNSYLVANIA
Cases Reported By
Department of Health

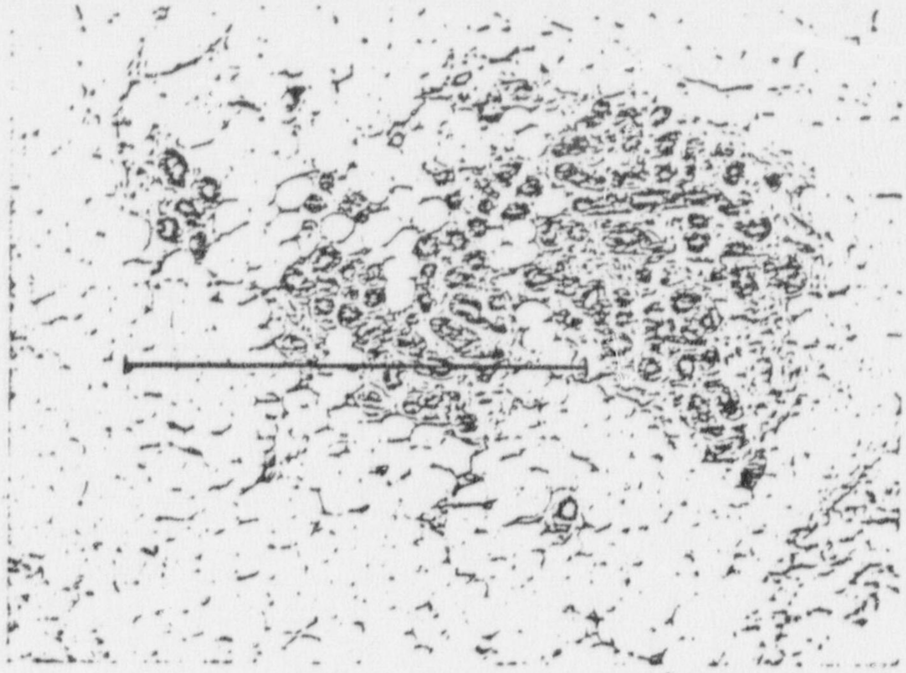
INCIDENCE OF NEONATAL HYPOTHYROIDISM
March 28, 1979 — December 31, 1979



● Neonatal Hypothyroidism Case
■ Nuclear Power Plant

PENNSYLVANIA
Cases Reported By
Department of Health

Figure 2



Normal breast epithelial cells (above) and carcinoma cells (below) showing juxtaposition with fatty areas (clear globules). The bar indicates 2.5 mm, the track length of the krypton-85 beta particle.

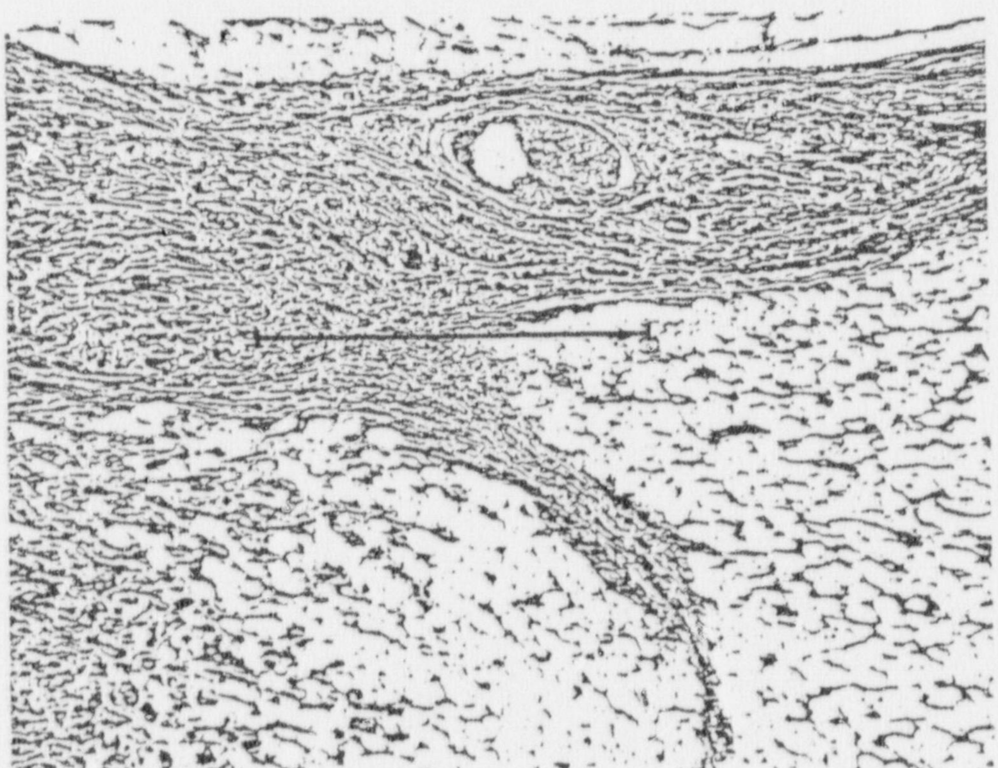
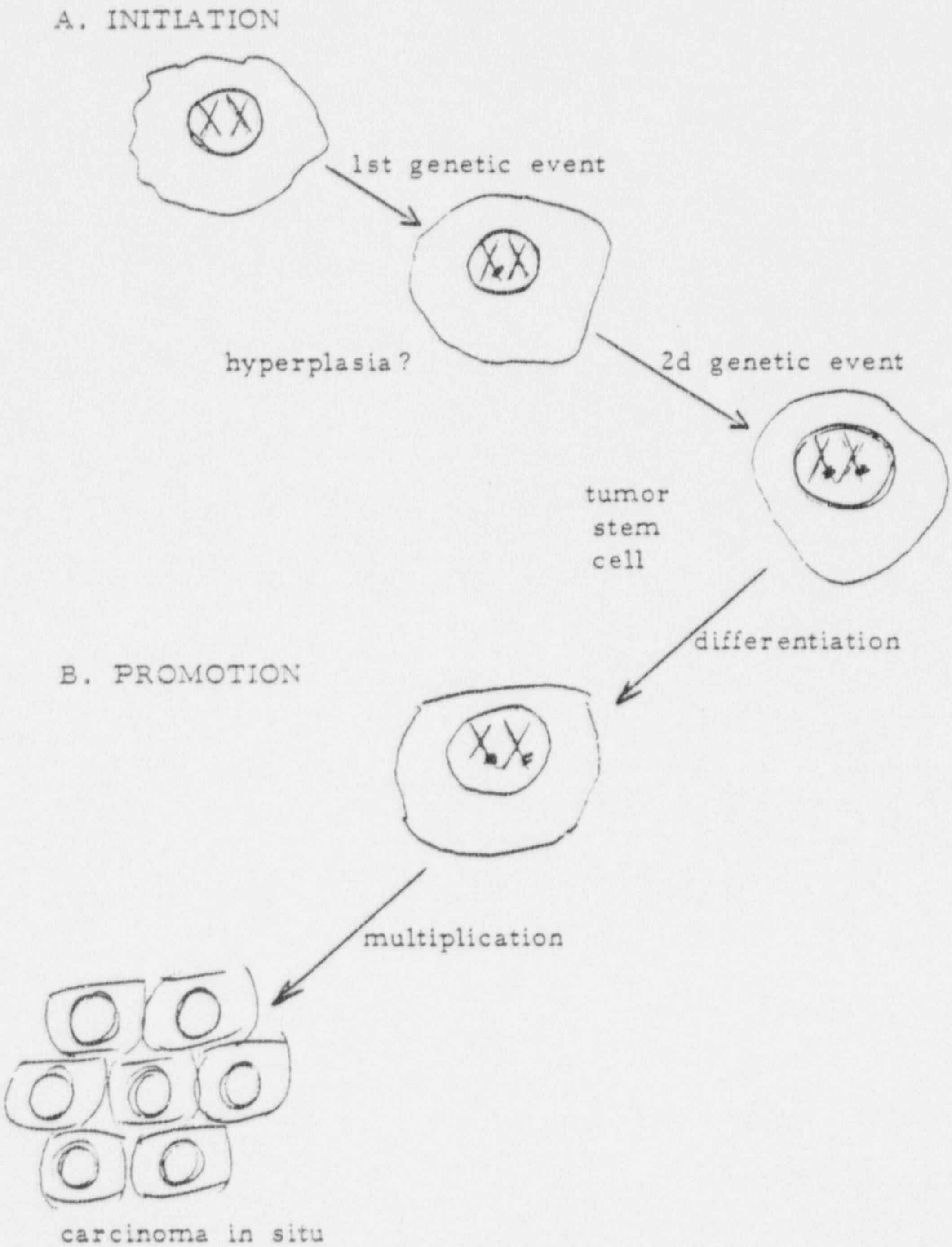


Figure 3

STEPS IN CARCINOGENESIS



RELATIVE RADIATION SENSITIVITIES OF VARIOUS TISSUES

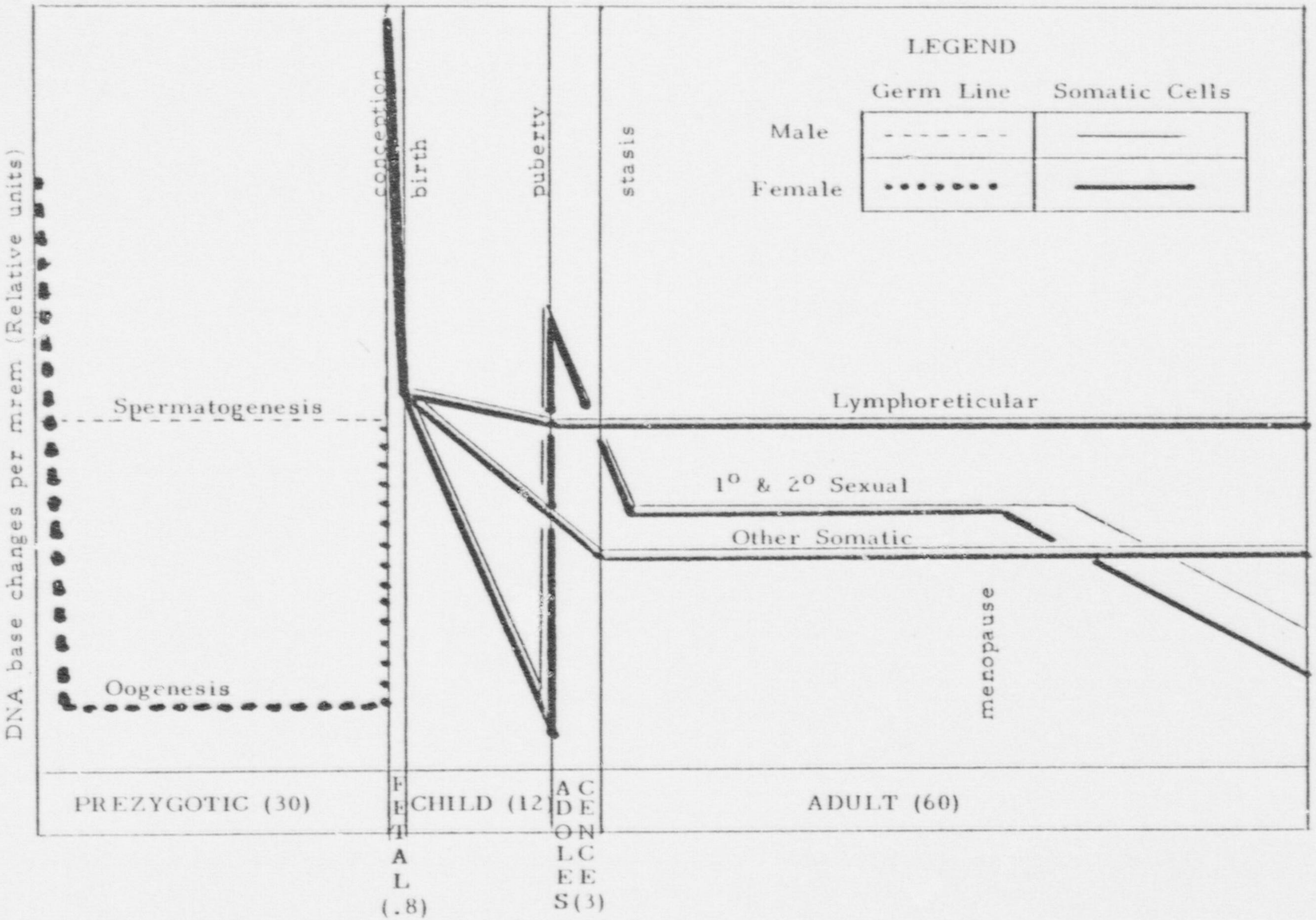


Figure 4

TMI AND THE POLITICS OF PUBLIC HEALTH*

by
Gordon K. MacLeod, M.D., F.A.C.P.**

A near nuclear catastrophe occurred just over a year and a half ago at Three Mile Island (TMI). It was an accident that just couldn't happen--after all, nuclear power plants were built so safely they could not possibly affect the public's health.

Your being here reflects a concern on your part for the health impact of that accident, especially as it could relate to the nuclear power plant at Indian Point. And, I commend you for that concern.

Shortly before the accident at TMI, I was persuaded by the new Governor of Pennsylvania to take a two-year leave of absence from the University of Pittsburgh to become Pennsylvania's Secretary of Health. I accepted the appointment in the spirit that public service is both a privilege and an obligation--even though the Pennsylvania Health Department was rated by one colleague as 50th out of 50. It needed to be restructured. After all, it had no place to go, but up.

Just twelve days after being sworn in as Secretary of Health, I had to face the TMI accident, the likes of which had never been thrust upon a public health officer anywhere in the world. More than any other single event, TMI pointed up very abruptly--almost explosively--the deficiencies in at least one of the Nation's Health Departments.

Bureaucratic and political decisions had carved up the Pennsylvania Department of Health over the past 10 to 15 years. One effect of the politics of public health can be seen in chart #1 which shows the decreasing percentage of total state dollars allocated to the health department.

When I was called about the accident early in the morning of March 28, 1979, what did I find? There was not even a book on radiation medicine in the Department. Worse yet, the medical library had been completely disbanded two years previously for budgetary reasons. There was no Bureau of Radiation Health in the Health Department. That was in the Department of Environmental Resources. No Occupational Health. It also was in the Department of Environmental Resources--with no physicians on its staff. No Office of Mental Health. That was in the Department of Welfare. Nor did the Governor have the capacity to bring together Pennsylvania's scattered health resources. Despite the intense mental stress during the TMI crisis, no one from the office of mental health ever sat in on a briefing session with the Governor. In short, there was little or no capacity within the Department of Health--nor elsewhere in the state--to deal with the extraordinarily serious health problems facing us.

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*Prepared for presentation at a Conference on Public Health and Public Policy sponsored by the Albert Einstein Medical Center on November 22, 1980 at Columbia University International Affairs Auditorium, 420 West 118th Street, New York [illegible]

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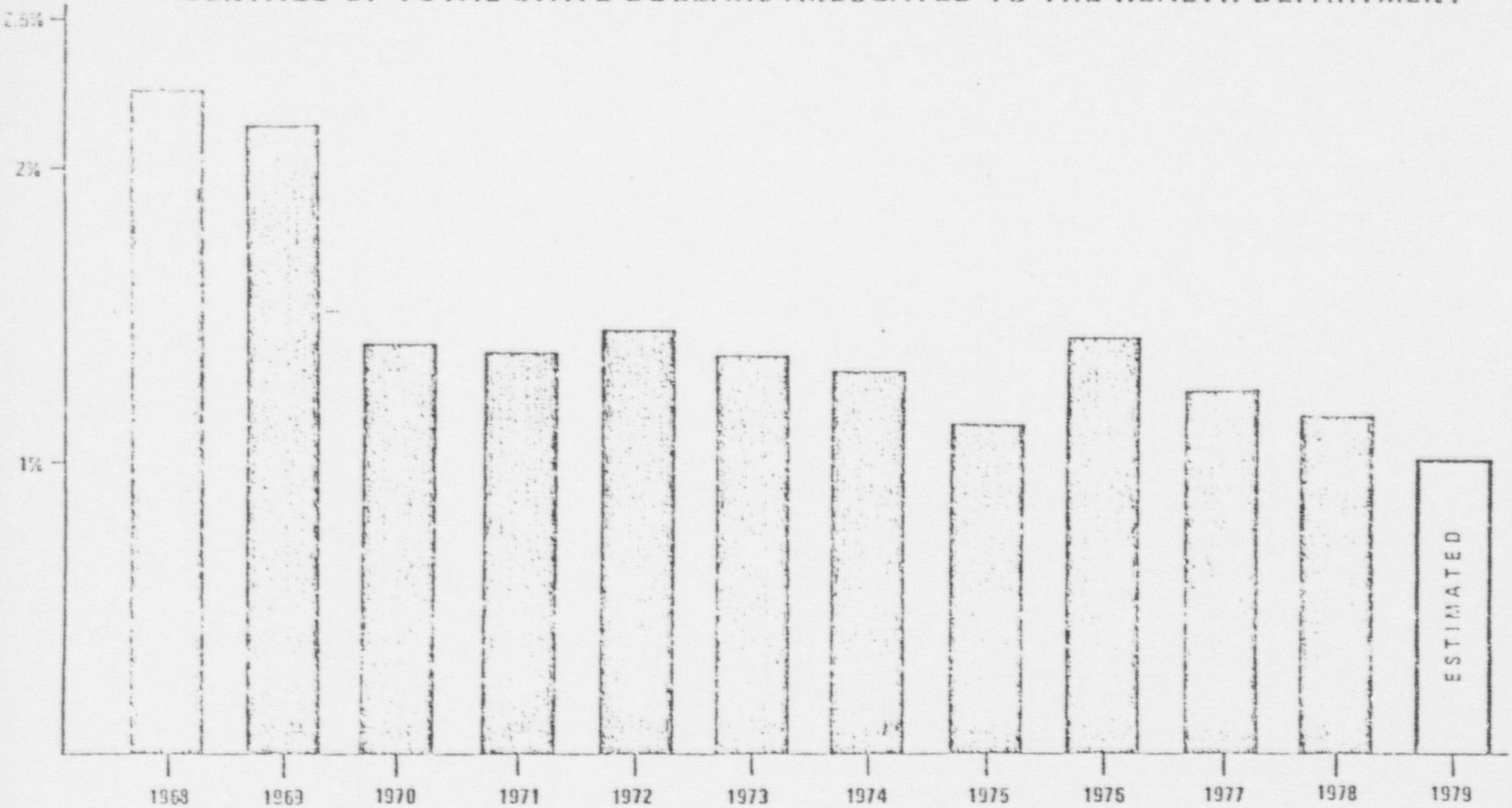
PERCENTAGE OF TOTAL STATE DOLLARS ALLOCATED TO THE HEALTH DEPARTMENT*



*From 1968 to 1978 the state budget increased from 2.016 to 5.865 billion dollars.

SOURCE: Bureau of Fiscal Management, Pennsylvania Department of Health

The management of the accident was left--and continues to be left--all too much to engineers and physicists--another political decision. There was little or no physician input by state and federal government into the many decisions made prior to the accident. There was little or no physician input into decisions made in the days immediately following the accident. And I am sorry to say, there remains little or no physician input into decisions made even today relative to the frenetic attempts to clean up radioactive debris from the accident.

In fact, radiation health protection was, is, and probably will be downplayed until we have a nuclear accident with visible signs of radiation damange or until citizens served by state and local health departments become sufficiently concerned to do something about radiation protection.

Let's briefly review the extent to which public health is being ignored in this nuclear age. First let's go back in history--to the 1950s. Some of you will recall the near ecstasy with which Americans welcomed the Nation's commitment to nuclear energy. Much of the ecstasy was spawned by the promise that we had embarked on a new mission--"atoms for peace"--it was called. And the product of that mission would be clean, safe and cheap energy for all. And we believed that promise. And until recently, we knew that nuclear energy was safe. Or I should say, we <u>thought</u> it was safe. This total assurance of safety still prevails in many quarters today. But that assurance of safety was shattered by TMI with potentially serious consequences in the days and months following March 28, 1979.

For years, we had been so thoroughly imbued with the idea of safe nuclear power that we hadn't even allowed ourselves to think about a nuclear accident--let alone prepare for one. Even though we have had nuclear reactors in Pennsylvania for years, there was no regularly employed physician expert in radiation medicine in the health department, nor anywhere else in Pennsylvania state government for that matter. We were not prepared to grapple with the health consequences of TMI's nuclear radiation exposure, because the state health department had done no prior planning for such an event.

Shortly after the onset of the accident, I proposed the establishment of a Division of Radiation Health within the Department. Regrettably--the Governor's office rejected this proposal on the basis of considerations having nothing whatever to do with health status--yet another political decision. Denial of my proposal to staff a Division of Radiation Health has stripped public health in Pennsylvania of its mandate to protect the health of its people and to prevent disease--particularly in case of a nuclear accident.

What we hoped to do was to assist physicians and hospitals statewide in addressing a vast array of technical issues--moral dilemmas if you will--that have been thrust upon the medical profession by a technology that has outstripped previous guidelines and concepts governing our professional behavior.

Let us briefly consider the function of state and local health departments in planning and directing responses to radiologic emergencies. The primary role of state and local health departments is the protection of the public's health. In case of radiation exposure, health departments must give paramount attention to its effects of humans, and they must also work cooperatively with those agencies that collect data and measure contamination of air, water, soil, food--affecting our homes, our children, our crops, our animals,

et cetera. In emergency planning for a radiation emergency, the responsibility of health departments is to ensure that there are sufficient professional and organizational resources at the state or local level to meet whatever health emergencies may occur. Such planning involves an inventory of both public and private resources and the initiation of appropriate steps to ensure their availability in an emergency. Health professional resources are essential and must be readily available for a radiological emergency plan to work. In planning for a radiation emergency, the first question to ask is what are the immediate and long-term effects on the public's health. This requires pre-planning using baseline data available to most health departments. Here we can call upon morbidity data and vital statistics: primarily live births and infant death rates and hypothyroidism and cancer rates. In addition, pre-planning requires knowledge of what health data are to be collected, of the demographic characteristics of the population at risk, as well as the availability of hospitals, physicians, other medical and logistical resources needed to handle a radiologic emergency. Such baseline reference data should be collected around each nuclear facility even before it is installed--and in the case of Indian Point, certainly before it resumes operating. In essence, then, preplanning for a radiation emergency provides a data base for decision making before, during, and after the emergency.

At this point, let me state my position clearly. Although I am unalterably opposed to nuclear warfare, I do not have a bias against nuclear power. While many of you may not share my position toward nuclear energy, I am sure we would all agree that we need to be prepared in case of a radiation emergency. I personally believe that nuclear power can be made relatively safe if we don't ignore the public health lessons of the past. In other words, I believe that health professionals have a responsibility--and society the right--to know how radiation and the production of nuclear energy affects the human body and its behavior insofar as we know it.

But, what do we know? We know that diagnostic radiation has been a boon to mankind--but it can result in fetal abnormalities. We know that therapeutic radiation has prolonged life in many instances--but it can result in radiation sickness, increased bleeding, and infection. And, to date, we know that nuclear power plants have been relatively safe--but they can have accidents which result in physical and psychological damage over a wide geographic area. What's more, we know from the United Nations Scientific Committee on the Effects of Atomic Radiation that the most important effect of "low doses of radiation is the occasional induction of malignant diseases."

Even with all we know, there is much we don't know. We don't know much about age-related responses to low dose radiation exposure, we don't know much about the response to one-time exposure versus continuous exposure, and, most importantly, we don't know very much at all about the pathological response to the interaction of low dose radiation with other exposures.

Without any professionally competent radiation medicine personnel in Pennsylvania's Department of Health, we were--and still are--ill-equipped to respond to the effects of either low or high dose radiation exposure from a nuclear reactor accident. In any state or locale at risk of exposure to a nuclear reactor accident, a radiological health unit must now be deemed essential to public health activity.

Any radiological emergency response plan must not overlook population density for at least 20 miles and maybe up to 50 miles around existing or proposed nuclear reactor sites. In light of Pennsylvania's lack of preparedness for a nuclear accident even after TMI, the public health and safety of millions of New Yorkers could be at risk if an accident were to occur at Indian Point. As a first step toward developing an emergency response plan, every state and local health officer in the vicinity of a nuclear reactor should learn the clinical effects of acute ionizing radiation doses.

Chart #2 shows radiation effects at various levels of exposure, but does not include acute radiation thyroiditis or fetal effects. Nor does it show long term effects on the thyroid gland, the genetic apparatus, or the life span. Nor does it show such defects as reduced body size, microcephaly, and mental retardation. Nor does it show the long term carcinogenic effect leading to solid tumors and leukemia.

During the accident, the maximum estimated radiation dose off site was calculated to be less than 100 millirem. Because of this very low level of exposure, I still believe we may not be able to measure radiation effects on the exposed population even though in April 1979 the Secretary of the then Department of Health, Education and Welfare announced there would be between one and ten fatal cancers and the same number of non-fatal cancers in the exposed population to TMI.

In face of repeated human error at the reactor site and elsewhere, even with my limited knowledge of radiation effects, I was loathe to accept engineering estimates of the safety of TMI.

On day two of the accident, I recommended, and on day three urged the Governor in the strongest possible terms to call for the departure of pregnant women and young children from an area within five miles of the TMI plant. But--and I hope you grasp the significance of this--the state's radiation physicists and nuclear engineers did not agree with my recommendation. Like so many other technicians responsible for monitoring the adverse effects of technology, they trust themselves into the position of making medical and public health judgments. On day two, non-medical judgment was accepted in this matter, which indeed should have been decided on no other basis but the public's health. Finally, it took the endorsement of the Nuclear Regulatory Commission's Chairman, Joseph Hendrie, a nuclear engineer, to convince the Governor to issue his directive for the departure of pregnant women and young children one day later.

Isn't that interesting. A physician and public health professional warns of potential health impacts of radiation and the warning goes unheeded. Not until a nuclear engineer supports the warning is it accepted. Certainly an Alice in Wonderland approach to sound public health decisions.

The dominance of engineering and physics over health did not flag after the accident. Six months after the accident, the Governor called together several state officials to plan for the clean-up procedure at TMI. Nobody from the Department of Health was present. Because no one from the Department of Health was invited.

SUMMARY OF CLINICAL EFFECTS OF ACUTE IONIZING RADIATION DOSES

	Dose (rem)					
	0-100	100-200	200-600	600-1000	1000-5000	Over 5000
Vomiting	None	5-50%	50-100%	100%	100%	100%
Onset of vomiting	--	3 hr.	2 hr.	1 hr.	30 min.	30 min.
Principal organ system affected	None	----Hematopoietic tissue		------------	Gastrointestional tract	Central Nervous System
Signs	None	Moderate leukopenia	--Severe leukopenia;-- purpura; hemorrhage; infection; epilation		Diarrhea; fever; electrolyte disturbance	Convulsions; tremors; ataxia; lethargy
Critical post-exposure period	--	--	-------4-6 weeks------		5-14 days	1-48 hr.
Prognosis	Excellent	Excellent	Good	Guarded	Hopeless	Hopeless
Convalescence	None	Weeks	Months	Long	--	--
Deaths	None	None	0-80%	80-100%	90-100%	90-100%
Time til death	--	--	-------2 months-------		2 weeks	2 days
Cause of death	--	--	--Hemorrhage; infection-		Shock	Respiratory failure; cerebral edema

From: Eisenbud: Environmental Radioactivity, 1973.

My recommendation for pregnant women and young children to leave the area was based upon several considerations. To begin with, we were dealing with too many unknowns--not the least of which stemmed from conflicting reports of levels of radiation emanating from the plant. Further, we were committed to exercising extreme caution in the interest of the public's health.

Most people knew enough to be concerned about congenital malformations and cancer that could result from radiation. But what the public did not know--and doesn't really know yet--is that radioisotopes of iodine are among the most abundant by-products of nuclear fission. Nor did pregnant women know that high levels of radioactive iodine can have such a devastating effect on the fetal thyroid gland which, if destroyed, can cause cretinism with severe mental retardation in infants in the first few months of life.

There was another threat present at TMI--perhaps a more significant threat than possible thyroid damage, which is treatable. As health officials, we were fully aware of the rising level of concern--in some cases outright panic--among the general public as conflicting reports of radiation fallout mounted in the news media. We knew full well that fear of radiation--however unreasonable that fear may have been--could cause health impacts as damaging as actual radiation itself. Such fear can produce real psychological, if not, physiological, change.

More than a year after the accident, a Hershey Medical Center study reported a surprising persistence of anxiety among a large part of the population near TMI. The health effects of this distress included increases of 113 percent in the number of persons using sleeping pills and 88 percent in those using tranquillizers. Also 14 percent used more alcohol and 32 percent smoked more cigarettes. Eloquent testimony, indeed, to the stress that grew out of the atmosphere surrounding TMI.

I have gone into some detail on the reasons for removing young children and pregnant women from the TMI area because I think it, better than any other example--points up a dangerous attitude toward public health--an attitude that surfaced all too often during the accident at TMI. And that attitude--which still persists--says that engineering expediency takes precedence over public health.

I found it interesting that in testimony before the President's Commission on TMI, one of the state's radiation physicists expressed strong opposition to the idea that radiation health activities should fall under the aegis of the Pennsylvania Department of Health. And to date it's position has prevailed as I shall explain later. He told the Commission that because a doctor would be in charge, he, as a radiation physicist in charge of radiation protection within the Department of Health, would have to be nice to doctors who operate x-ray equipment. Apparently he never reflected upon the far greater likelihood of radiation physicists and nuclear engineers being nice to their counterparts working at nuclear power plants. I cite this example simply because I think it exemplifies another political problem--an example of bureaucratic jealousy without regard for health care.

Despite the consequences of radiation both to the human body and the spirit, such deficiencies are not limited to state government. Early in the accident, we urged the federal government's Nuclear Regulatory Commission to

provide the on-site services of a physician expert in the field of radiation health. I persisted in my request to NRC's Harold Denton, only to be informed for several days that NRC just could not find such a person. I was told, and I think this is significant, that NRC had no physicians on its staff, much less a physician schooled in the field of radiation medicine. No physicians directly employed by NRC--again a sad commentary on the priority given to public health matters involving nuclear regulation.

Our job--and I don't have to tell you this--was to prevent the harmful effects of any possible radiation, not simply allow fallout to occur and then have to depend on medical therapy to correct the consequences of exposure.

Another example of the lack of public health emphasis in the aftermath of TMI is how little attention has been paid to the feasibility of stockpiling potassium iodide as a protective agent against radioactive iodines. Although I had prepared lengthy testimony on this matter for the President's Commission, it was almost entirely preoccupied with the nuclear engineering and evacuation logistics--in fact I wasn't asked a single question about potassium iodide as an effective preventive medicine against radioactive iodine. When I inquired into the reason for this glaring omission, the Commission Chairman Dr. John Kemeny, wrote me that the Commission just didn't have time to discuss potassium iodide. Apparently, too many important non-health issues took up its time.

But let me tell you the potassium iodide story. We know that a large release of radioactive iodine into the atmosphere from a nuclear power reactor would result in the public's inhaling or ingesting amounts which could produce acute, continuing, or late thyroid effects. These effects range from mild thyroiditis to hypothyroidism to benign thyroid neoplasms, nodules, and cancer. Fetal hypothyroidism associated with cretinism is of particular concern due to the inverse relationship between iodine uptake and age. Apart from evacuation and shelter to protect against radioactive iodine by the thyroid gland present the most complete protection against the hazards of inhaling or ingesting I-131. Potassium iodide (KI) was found to be eminently suitable for thyroid blocking purposes.

For two or more years prior to TMI, the Department of Environmental Resources in Pennsylvania had been frustrated in obtaining stockpiles of potassium iodide from the federal government. I think it interesting to speculate on whether the Department of Health would have met with earlier success if the responsibility for the matter had been lodged there rather than in the Department of Environmental Resources.

Immediately after the accident, the State's Department of Environmental Resources again requested potassium iodide from the federal Department of Health, Education and Welfare. It took more than 60 hours after that just for the then Secretary of DHEW to give the order to initiate steps to ship potassium iodide to Harrisburg. Five days after the accident began the first shipment of 11,000 little brown vials arrived:

--6,000 of those 11,000 vials were unlabeled;
--Each dropper yielded only one half the correct dosage;
--The droppers did not fit the vials; and
--Many of the vials contained hairlike filamentous material and other particulate matter.

Seven days after the accident began, we received instructions from Secretary Califano to administer potassium iodide to all workers at the site and to distribute it to all residents within a ten-mile radius. This, again, was a recommendation based upon a total lack of understanding of the real problem. By this time, we had received assurances from the NRC that the reactor was progressing toward a cold shut down. The very next day, the politics of public health must have prompted Secretary Califano to appear before Senator Kennedy to fault the state for not administering the drug.

An important factor in obtaining satisfactory blocking of radioactive iodine uptake is the speed with which KI is administered following exposure to radioiodine. Within 30 minutes after oral administration, KI blocks the uptake of 99% of I-131 for 24 hours. Standard uptake curves demonstrate that the bulk of the radioactive iodine from a single exposure will have entered the thyroid within 10 to 12 hours; little benefit may be expected by blocking beyond that time. Significant benefit (a block of 50%) is attainable during the first 3 to 4 hours.

Other factors militated against the administration and distribution of potassium iodide:

1) The known incidence of side effects such as iodism--a severe skin rash--plus thyroid diseases; and an occasional cardiac death in the elderly has been reported--presumably from excess potassium and
2) The likelihood of precipitating unnecessary panic among the populace after 6 days of intense stress and strain.

Thus, we avoided a fiasco similar to the swine flu mass immunization where the U.S. Center for Disease Control attempted to prevent an epidemic that never existed. And we were ready to respond instantly to real and immediate public health problems. But had we experienced the massive fallout that many people feared would result from the accident during the first few days, we would have been without the only proven effective preventive medicine against the accumulation of radioactive iodine in the thyroid gland, even as we are today.

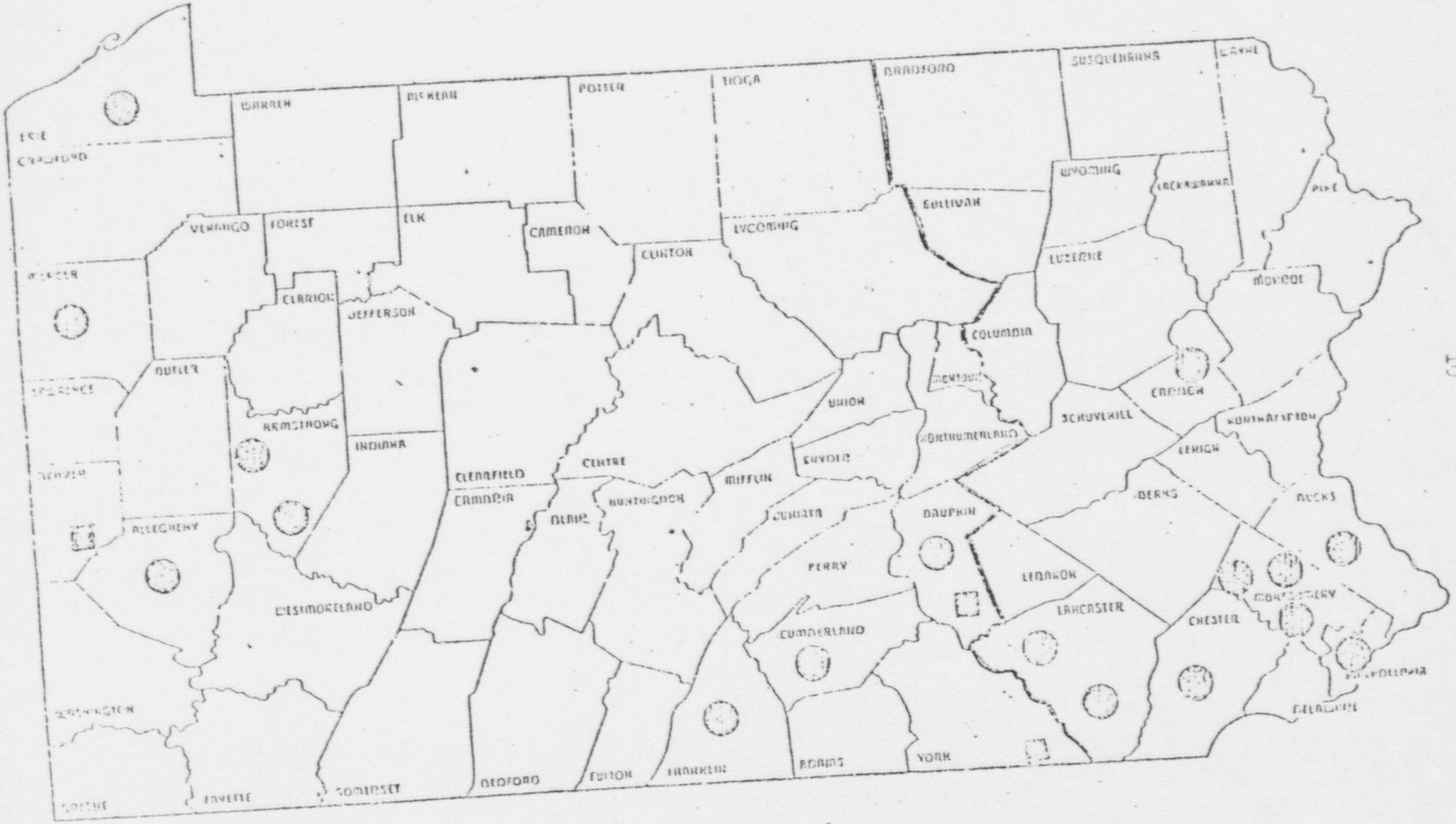
Let us consider a moral issue at stake many months following the nuclear accident--the right to know the truth. Four months after I had resigned as Secretary, I was called about an unusual cluster of thyroid deficiency cases in Pennsylvania. I immediately notified state health officials. After several days of refusal by the Health Department to alert the public to this preventable illness in newborns, I asked a reporter to look into the matter in order to discover and treat any cases missed in screening.

In mid-1978, the state health department fortuitously began screening newborns for thyroid deficiency. Seventeen cases of depressed thyroid function were reported and treated in the nine months prior to the accident. Surprisingly, the number of cases jumped to 27 during the nine months following the accident. The increase was concentrated downwind of the reactors at TMI and Peachbottom. Of interest, the number of cases downwind has remained elevated during 1980. Prevailing winds are west to east. (See charts #3, 4 and 5)

Since the TMI accident, major concentrations of thyroid deficiency cases have occurred in Lancaster County--immediately downwind of the counties containing TMI and Peachbottom reactors. These cases are many times more than what would be expected in Pennsylvania. Even though the numbers are small,

INCIDENCE OF NEONATAL HYPOTHYROIDISM

JUNE 20, 1978 - MARCH 28, 1979



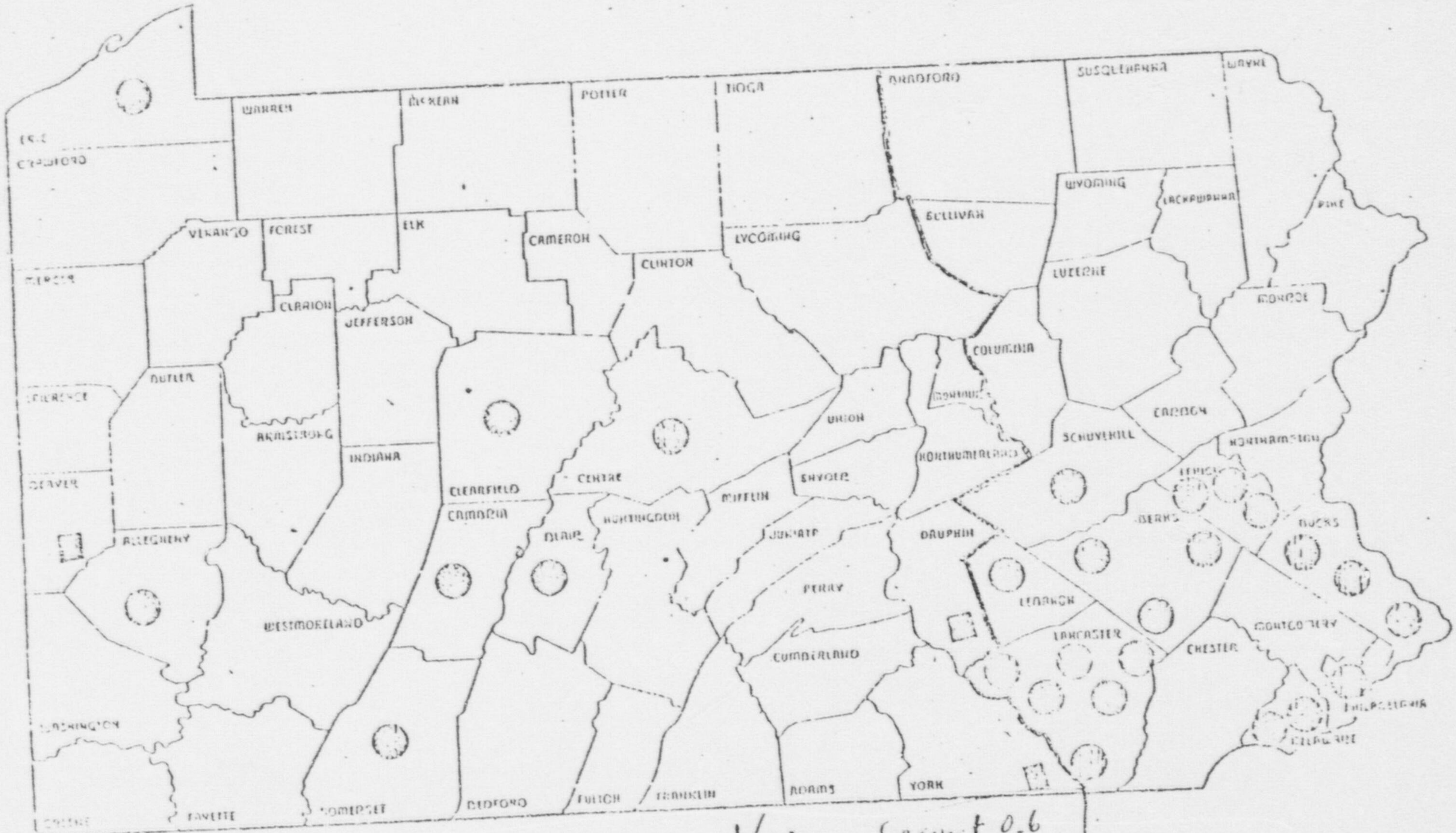
NEONATAL HYPOTHYROIDISM CASE

PENNSYLVANIA

CASES REPORTED BY

INCIDENCE OF NEONATAL HYPOTHYROIDISM

MARCH 28, 1979 - DECEMBER 31, 1979



1/9300

6/2700

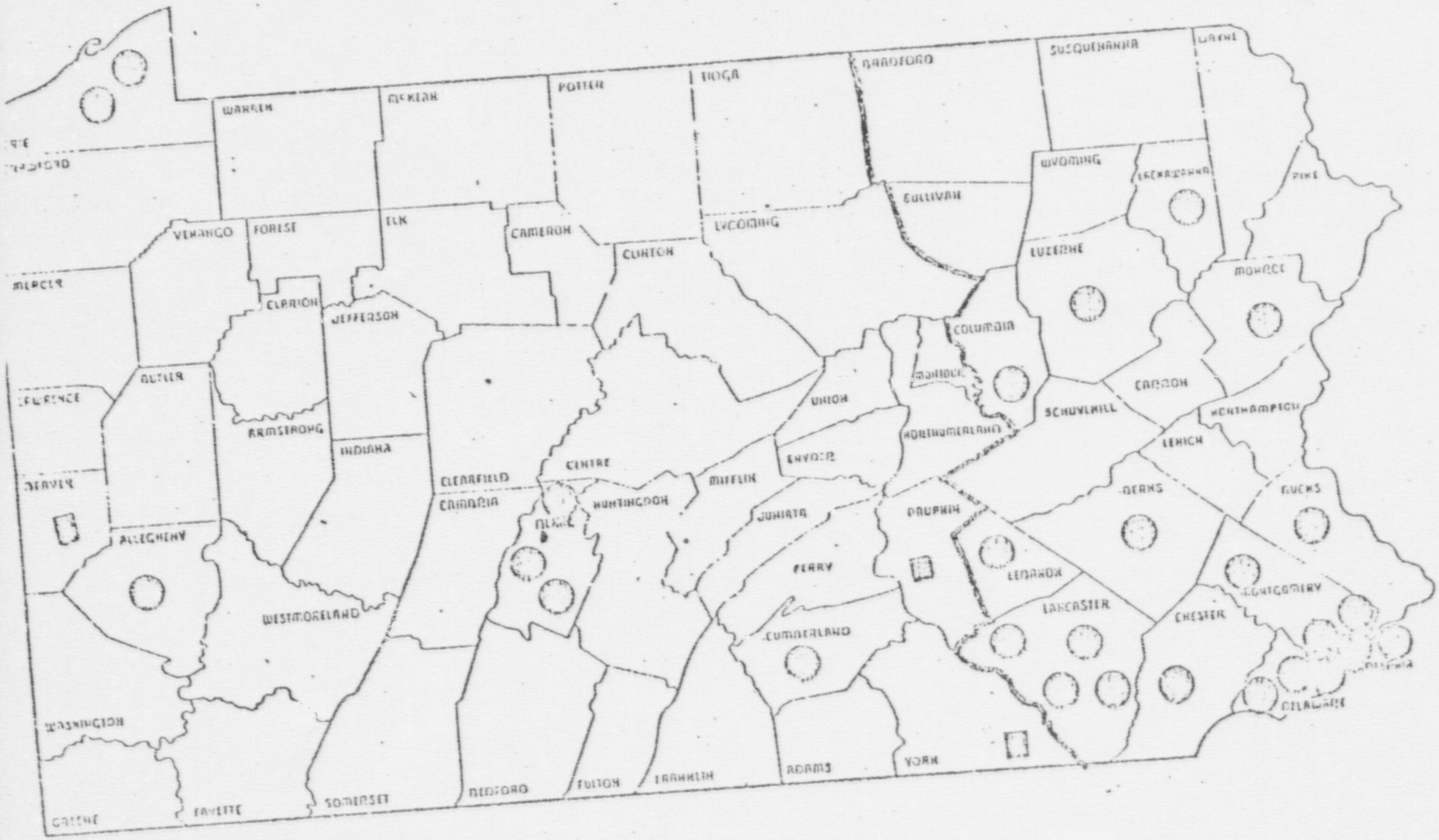
expect 0.6
actual 6

○ NEONATAL HYPOTHYROIDISM CASE

PENNSYLVANIA
CASES REPORTED BY

INCIDENCE OF NEONATAL HYPOTHYROIDISM

JANUARY 1, 1980 - AUGUST 31, 1980



PENNSYLVANIA

Neonatal Hypothyroidism Case

Nuclear Power Plant

Cases Reported by Department of Health

they warrant further epidemiological investigation whether from inbreeding, from environmental causes such as chemical contamination of water and food, or from industrial or medical radiation sources. It's premature to blame them on radioactive iodine but it cannot yet be ruled out. (See chart #6)

Once again, the politics of public health prevailed. The Health Department has refused to examine the relationship of the TMI accident to this dramatic increase in hypothyroidism. (See chart #7)

It was our good fortune that there was no immediate loss of life from TMI, but lack of adequate communication with the public during and after the accident led to a loss of confidence in our public officials. Public health is now in jeopardy of losing its credibility as health data become available and are not released or are carelessly compiled.

Since my resignation as Secretary of Health, several concerned health department employees have called me repeatedly to complain that abnormal health data were not being made available to the public. More than a year after the accident at TMI, I released long overdue infant mortality statistics which I was told were not going to be released by the Health Department. My public announcement prompted the state to release infant death rates within 72 hours. These data should have been made public months before.

Regrettably some were convinced by Health Department statements that these data showed conclusively that the nuclear accident at TMI had caused no damage to unborn children resulting in birth defects or death during infancy. While this is entirely possible, it is premature to conclude this from the Pennsylvania Health Department data. For in its eagerness to report no increase in infant deaths, the Health Department data unfortunately were unreliable. They were flawed by errors and internal inconsistencies. (See charts #8 and 9)

Without careful compilation of data, many mathematical impossibilities were found in the data released by the Health Department. For 10 miles around TMI, the total fetal death rate for January is lower than the two components which comprise the total. Also, the neonatal and infant death rates for October and November vary, even though the two components (Harrisburg and outside Harrisburg) which should comprise the total are identical. Furthermore, tabulation of neonatal and infant death rates for Harrisburg during 1979 are identical during the entire year. We are led to believe then that all infant deaths in Harrisburg, i.e., those in the first year of life, occurred neonatally in the first 28 days of life. In chart #9, the second quarter neonatal death rate is higher than the same quarter infant death rate even though chart #8 shows the two rates during the same period to be identical.

Yet to be explained is why five-and-ten mile infant death rates during the six months following the accident climbed sharply compared to the same period in previous years. The increase could be a statistical variation or could be induced by cause. Some of these findings are statistically highly significant and surely warrant careful attention if not further study. Later data reveal, increases in neonatal deaths after the accident may be even more significant than increases in infant deaths.

Five-mile rates were 6.7, 2.3 and 16.2 infant death rates per thousand live births for time frames from April through September in 1977, 1978 and 1979, respectively. Within ten miles--including Harrisburg--death rates were

RATIO OF NEONATAL HYPOTHYROIDISM TO NEWBORNS IN PENNSYLVANIA

	Western PA	Eastern PA
June 20, 1978 to March 28, 1979	1 : 6,805	1 : 6,564
March 28, 1979 to December 31, 1979	1 : 7,955	1 : 3,021
January 1, 1980 to August 31, 1980	1 : 7,231	1 : 2,746

All newborn hypothyroid cases reported by Pennsylvania Department of Health since initiation of neonatal hypothyroid screening program, June 20, 1978 until August 31, 1980.

FETAL, NEONATAL, AND INFANT DEATH RATES BY MONTH: PENNSYLVANIA, TEN MILE TMI AREA COMMUNITIES (INCLUDING AND EXCLUDING HARRISBURG CITY) AND HARRISBURG CITY, JANUARY - DECEMBER, 1979

Reported by Pennsylvania Department of Health

Month	Geographic Area				Geographic Area			
	Pa.	TMI (10 Mile)	Harrisburg City	TMI (Excl. Hbg.)	Pa.	TMI (10 Mile)	Harrisburg City	TMI (Excl. Hbg.)
	Fetal Death (Total) Rates				Fetal Death (Excluding Abortions) Rates			
January	24.4	29.9	31.9	33.3	15.3	26.9	21.5	33.3
February	22.3	23.4	45.0	10.6	12.5	23.4	45.0	10.6
March	25.4	21.9	48.5	9.2	13.1	19.8	39.2	9.2
April	23.3	16.5	32.3	9.5	13.0	16.5	32.3	9.5
May	25.1	11.2	9.3	12.0	15.5	8.5	-0-	12.0
June	22.7	9.3	19.2	4.5	13.4	9.3	19.2	4.5
July	20.8	13.4	29.4	7.4	12.9	13.4	29.4	7.4
August	22.8	15.3	20.6	13.1	13.3	15.3	20.6	13.1
September	22.8	18.2	44.2	4.6	13.2	18.2	44.2	4.6
October	21.0	18.8	42.1	8.9	11.7	18.8	42.1	8.9
November	20.4	21.3	42.1	12.8	12.3	21.3	42.1	12.8
December	24.6	6.5	-0-	9.4	15.3	6.5	-0-	9.4
Jan. - Dec.	23.0	17.1	30.6	11.1	13.4	16.3	28.2	11.1
	Neonatal Death Rates				Infant Death Rates			
January	9.8	21.5	33.0	17.1	14.8	21.5	33.0	17.1
February	10.0	10.3	28.3	-0-	13.7	13.7	28.3	5.4
March	8.2	19.2	40.8	9.3	11.5	22.4	40.8	14.0
April	10.7	20.1	33.3	14.4	14.1	20.1	33.3	14.4
May	9.8	17.0	18.9	16.3	14.1	17.0	18.9	16.3
June	10.7	18.7	39.2	9.1	13.2	18.7	39.2	9.1
July	10.0	5.4	10.1	3.7	11.9	10.8	10.1	11.1
August	7.3	6.2	-0-	8.8	10.2	12.5	-0-	13.3
September	9.8	12.3	9.3	13.9	12.9	15.4	9.3	18.5
October	9.9	9.6	11.0	9.0	13.7	12.7	11.0	9.0
November	10.5	6.2	22.0	-0-	14.8	9.3	22.0	-0-
December	10.9	13.1	31.3	4.8	16.1	19.6	31.3	9.5
Jan. - Dec.	9.8	13.2	23.0	8.9	13.3	16.1	23.0	11.5

[illegible] Figures for 1979 are provisional.

BR213

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--PENNSYLVANIA CAPITOL UPDATE--

(HARRISBURG) -- A COMMITTEE TO INVESTIGATE HYPOTHYROIDISM IN PENNSYLVANIA MET FOR THE FIRST TIME TODAY... BUT THE RADIATION EFFECTS OF THE THREE MILE ISLAND ACCIDENT WERE NOT ON ITS AGENDA.

DOCTOR DONALD REID -- HEAD OF THE 11-MEMBER PANEL -- SAID THE GROUP WILL NOT DISCUSS EITHER THE PRO OR CON SIDE OF WHAT MAY OR MAY NOT HAVE OCCURRED SINCE THE THREE MILE ISLAND ACCIDENT.

HE SAID THE MEDICAL EXPERTS WILL CONDUCT AN UNBIASED OBJECTIVE STUDY OF WHY 34 PENNSYLVANIA INFANTS SUFFERED FROM HYPOTHYROIDISM IN 1979.

THE DISORDER IS A BIRTH DEFECT THAT, IF LEFT UNTREATED, CAUSES MENTAL RETARDATION. IT CAN BE CAUSED BY A VARIETY OF HEREDITARY AND ENVIRONMENTAL FACTORS... INCLUDING RADIATION.

THE COMMITTEE OF MEDICAL EXPERTS FROM PHILADELPHIA, PITTSBURGH, HERSHEY AND ATLANTA WAS FORMED AFTER 1979 FIGURES REVEALED SIX CASES OF HYPOTHROIDISM IN LANCASTER COUNTY.

ONE OTHER CASE HAS BEEN REPORTED THIS YEAR IN THE COUNTY... WHICH IS LOCATED NEAR THE CRIPPLED NUCLEAR PLANT.

REID SAID THE NUMBER OF CASES IS NOT UNUSUAL... BUT THE CLUSTERING IS.

AP-PX-0327 1400EST

B214

FETAL, NEONATAL, AND INFANT DEATH RATES BY QUARTER: PENNSYLVANIA AND TEN MILE TMI AREA COMMUNITIES, 1977 - 1979

Reported by Pennsylvania Department of Health

Year/Quarter	Fetal Deaths-Total[1]		Fetal Deaths - Excluding Abortions[2]		Neonatal Deaths[3]		Infant Deaths[3]	
	Pa.	TMI (Ten Mile)	Pa.	TMI (Ten Mile)	Pa.	TMI (Ten Mile)	Pa.	TMI (Ten Mile)
1977								
Jan.-March	28.0	17.7	13.4	16.6	10.7	12.4	14.7	14.7
April-June	25.2	18.3	12.4	9.5	11.1	8.5	14.4	11.7
July-Sept.	24.9	25.0	13.1	22.0	10.1	6.1	12.9	9.2
Oct.-Dec.	25.1	23.6	12.7	16.5	10.1	10.5	13.7	14.7
1978								
Jan.-March	26.3	15.9	13.0	12.8	9.9	8.6	14.3	14.0
April-June	28.0	27.3	[illegible]	15.0	11.1	7.6	14.0	9.8
July-Sept.	24.5	19.1	13.4	15.3	9.3	1.0	11.8	4.9
Oct.-Dec.	25.2	22.2	14.1	14.9	10.5	10.8	13.6	15.1
1979								
Jan.-March	24.1	23.2	13.6	23.1	9.3	17.2	13.3	19.4
April-June	23.7	12.7	14.0	11.2	10.4	18.6	13.8	15.5
July-Sept.	22.1	15.5	13.1	15.5	9.0	7.9	11.7	12.8
Oct.-Dec.	21.9	15.7	13.0	15.7	10.4	9.6	14.8	15.6

1) Death rates per 1,000 deliveries (live and fetal deaths).
2) Death rates per 1,000 deliveries (live births and fetal deaths - excluding abortions).
3) Death rates per 1,000 live births.

Notes: Data for 1979 are provisional.

10.5, 7.2 and 15.7 during like time frames. Statewide levels for the same three years were 13.9, 13.5 and 13.6, respectively. (See chart #10). The Health Department subsequently blamed the 1979 elevation above the statewide level in the ten-mile rate on high infant mortality among Harrisburg's blacks. But it is also know that minority data were present in 1977 and 1978 before the nuclear accident occurred. Had the infant death rate remained unchanged or even decreased, I trust those findings would have been well publicized.

According to a newspaper report, a Health Department spokesman also reported that a radiation effect--reversing the sex ratio among newborns--did not occur in the months after the accident. Unfortunately, the data necessary to draw this conclusion were not complete. The radiation effect is upon post-meiotic spermatogenesis after the TMI accident. Tabulation of baseline data for male-female sex ratios in exposed persons begins 50 days after exposure, prior to conception. Accordingly, a decrease in the proportion of male births from radiation can only be found by counting the sex distribution for about four weeks on either side of the 320th day (50 plus 270 days for normal gestation) after the March 28, 1979 accident. These data were not available when the story was released.

Also of concern is the omission of 88 infant deaths from Department of Health data. (See chart #11). An unexplained absence of 88 infant deaths from Pennsylvania Health Department data occurred in the third quarter of 1979. This reduced number of infant deaths is found by comparing it to the number listed in the Federal Government's Vital Statistics for 1979. This discrepancy raises further questions about the reliability of health department data.

In light of these many discrepancies, it is difficult, if not impossible, to understand how, in May of 1980, the Health Department could state, "After careful study of all available information we continue to find no evidence to date that radiation from the nuclear power plant resulted in an increased number of fetal, neonatal or infant deaths."

The release of inexact data once again reflects upon the lack of health input into planning for the next nuclear accident. And there has been no public correction of these many errors. Assuming that the mathematical impossibilities, tabulation of erroneous data, the presentation of incomplete data, and the State's reporting of 88 fewer infant deaths than that reported by the federal government were all accidents of compilation, or else carelessness, it must prompt us to urge state and federal health agencies to improve their efforts to collect health data before, during, and after nuclear accidents.

Although I do think the available data are non-conclusive in their present form, such unusual patterns of hypothyroidism and infant death rates following the nuclear accident at TMI warrant complete candor and disclosure, not delay and denial. A lack of confidence in these findings prompts me to urge an investigation of Pennsylvania health data following TMI by a group of qualified epidemiological investigators.

Unless departments of health are removed from the political arena, they will probably not be able to operate much differently from how we had to operate in response to TMI. For until health related activities of state government are brought under the wing of competent health departments, we

INFANT DEATHS PER 1,000 LIVE BIRTHS A/

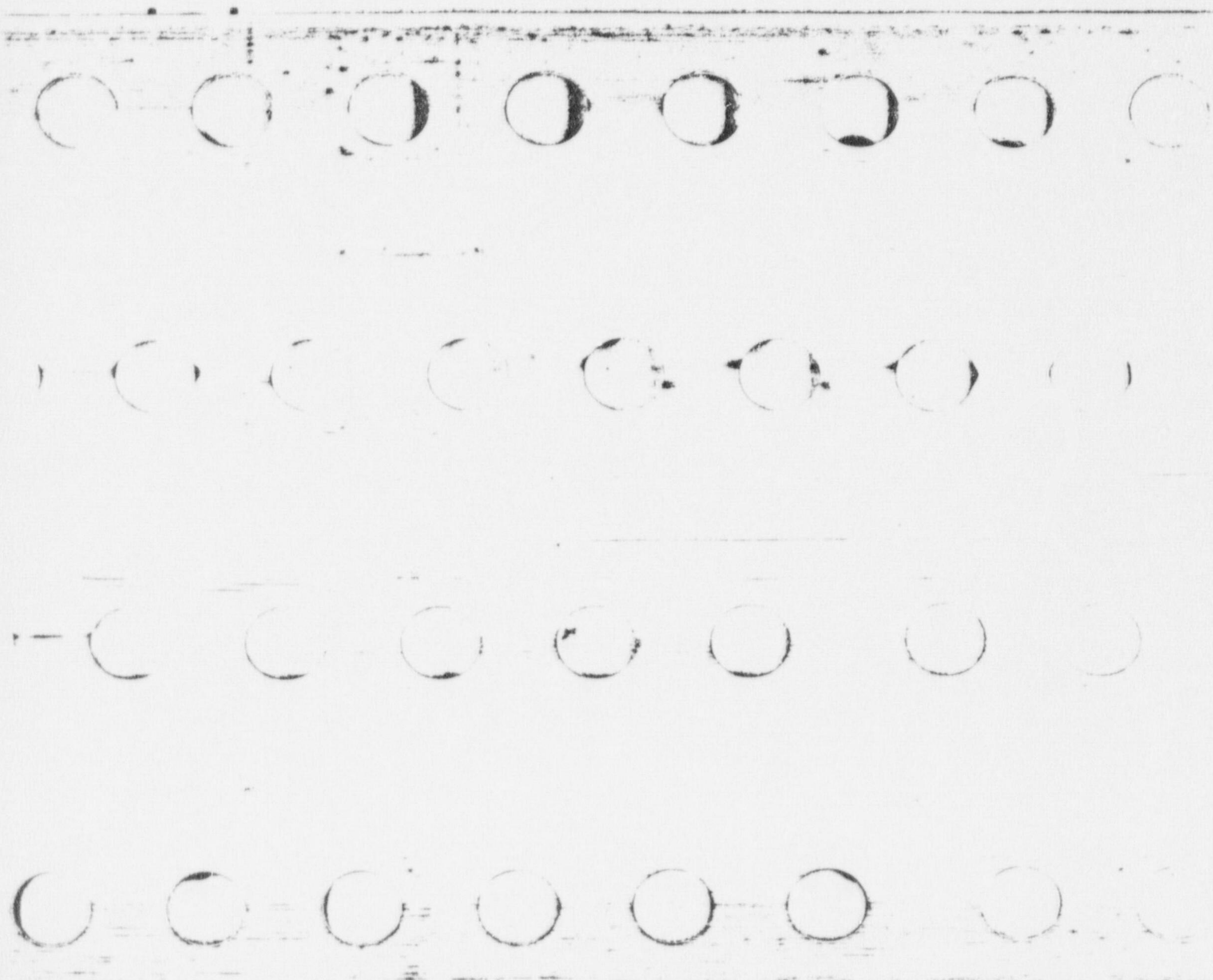
(ACTUAL DEATHS IN PARENTHESES)

	5 MILES FROM TMI	10 MILES FROM TMI	ALL PENNSYLVANIA
1977	6.7 (3)	10.5 (20)	13.9 B/
1978	2.3 (1)	7.2 (14)	13.5 B/
1979	16.1 (7)	15.7 (31)	13.6 C/

A/ APRIL THROUGH SEPTEMBER

B/ ANNUAL RATES

C/ JANUARY THROUGH SEPTEMBER 1979



NUMBER OF LIVE BIRTHS AND INFANT DEATHS REPORTED IN PENNSYLVANIA AFTER THREE MILE ISLAND ACCIDENT OF MARCH 29, 1979

A COMPARISION BETWEEN U.S. VITAL STATISTICS AND DATA FROM PA DEPARTMENT OF HEALTH (DOH)

	1979 D/HHS* Live Births	1979 D/HHS* Infant Deaths	1979 PA DOH** Infant Deaths	Excess (Deficit) of PA Over U.S.
April 1 - June 30, 1979	38,014	527	525	(2)
July 1 - Sept. 30, 1979	42,873	589	501	(88)
Oct. 1 - Dec. 31, 1979	38,303	565	567	2

*U.S. Monthly Vital Statistics Reports
D/HHS National Center for Health Statistics

**Data released by PA Department of Health, May, 1980

All 1979 figures are provisional

will continue to grope for direction and support in our efforts to address the health impacts of such an accident. But, perhaps there is a way that this can be done. (Perhaps there is a way that this can be done.) Perhaps there is a way to bring a common effort to whatever commitments to public health may remain in state government. I don't think it can be accomplished under the present structure.

The health industry has grown to such tremendous scope and size that it poses infinite temptations for political control. And any one individual entrusted with the responsibility of overseeing such a massive industry from within state government is going to be subjected to unbelievable political pressures from those who seek to share in such control. It is only fair to say the Governors who generally have no health professional qualifications are the victims of the politics affecting the health industry. There are just too many factions pulling and tugging at politicians for their own better interests. And these forces exert tremendous pressures on anyone trying to participate in a rational decision making process. That pressure is transmitted to secretaries of health who serve at the pleasure of the Governors.

In recent years, it has become fairly routine for new Governors--and sometimes new mayors--upon election to replace the entire administrative leadership in health departments. If, in fact, we are going to accept some degree of politicization of health departments, perhaps we should follow the trend in the executive and judiciary branches in Pennsylvania and elect a state surgeon general on the popular ballot--along with the Auditor General, and now, the Attorney General. As with the judiciary, a ten year term for the chief health officer could promote professional performance. Or perhaps health activities now conducted throughout state government could be placed under the direct control of a health commission selected by the Governor from a panel of physicians nominated for 5 to 10 year appointments by a statewide panel of medical school deans and leaders of medical professional societies. Because of the magnitude of the health care industry and the wide dispersion of public health services, including radiation health, the chief health officer cannot effectively operate professionally under the direct supervision of the Governor. Nor should the Governor be subjected to the constant political precsures exerted by special interests within and outside the health industry.

The reality of politics says that with high dollar volume, special interest pressures are exerted from a myriad of directions in both public and private sectors, the commitment to public health can quickly crumble in the process.

So now we come to the last serious issue. What happens in the event of any future nuclear accidents? How do we protect the public's health? Short of immediate cessation of all nuclear activity which I think is well nigh impossible, the nation must be prepared not to repeat the mistakes of TMI and to be far better prepared than Pennsylvania is at the present time. But let me warn you that the people of Pennsylvania are no better off today and perhaps worse off than they were the day before the radiation release at TMI on March 28. There is still no division of radiation health. No resources for health radiation preparedness. No potassium iodide for deployment in case of another accident.

The people of Pennsylvania are uniquely sensitized to the psychological stress of a nuclear accident. Were there to be another accident in any one of Pennsylvania's nuclear reactors in our present state of unpreparedness, irreparable psychological damage could occur. The lack of public health preparedness for another nuclear accident has been repeatedly cited--but to no avail. Based upon some of the lessons learned from TMI, let me suggest some items for consideration by public health departments in planning for a radiation emergency.

Every health department should have access to expertise in radiation medicine. One good model can be found at Massachusetts Institute of Technology, where a well regarded specialist in internal medicine is permanently appointed to chair a committee composed of radiologists, environmental health specialists, nuclear engineers, radiobiologists, nuclear medicine specialists, nuclear physicists, and radiation health physicists. A similar committee under state government auspices might be called upon to oversee health training programs for employees, community physicians, and other health care providers and be available to assist in the management of a nuclear accident were one to recur.

Every health department should be informed about preventive or protective programs in order to avoid or alleviate radiation induced diseases. In this regard, potassium iodide is recommended to be available for deployment and distribution to all persons at risk of exposure to a radiological emergency.

Both state and local health departments in the vicinity of a nuclear reactor should initiate or work cooperatively with the management of the plant to conduct radiation drills simulating radiologic emergencies. Through similar exercises our students have learned a great deal about disaster management.

Every health department should develop a radiological emergency response plan for the health aspects of a nuclear accident. The plan should encompass overall evacuation and special procedures for specific sectors of society, particularly institutionalized or home-bound patients.

Health departments should promote age-specific continuing health education programs pertaining to radiation for all age groups, especially for puberty and pregnancy.

Open communication with the medical community, the religious community and the general public will help to delimit psychological damage which we have seen result from the TMI accident. Above all, the public has the right to know the risks and dangers from a radiological emergency as well as from all other catastrophes that can affect the public health.

As soon as possible after the accident, it should be announced that health data shall be collected and released promptly to the citizenry affected by the emergency. The medical consequences, whatever they may be, should be promptly interpreted and further studies done as indicated. The public has become increasingly sophisticated in interpreting data about radiological emergencies.

Only through efforts to develop a sound system of public health throughout the country will we ever be able to address effectively the many health problems that could result from another nuclear accident. And unless we do address these problems--and address them now--we may well find our health to have deteriorated ten or twenty years hence pondering the same puzzles that are confronting us here today.

to appear in HEALTH PHYSICS

(August, 1981)

IODINE-131 IN THYROIDS OF THE MEADOW VOLE (*MICROTUS PENNSYLVANICUS*) IN THE VICINITY OF THE THREE MILE ISLAND NUCLEAR GENERATING PLANT

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Abstract

Meadow voles (*Microtus pennsylvanicus*) were trapped in the vicinity of Three Mile Island Nuclear Power Station between 6 April and 16 April 1979. Thyroids of voles caught 1.9 km from the reactor (Site III) contained significantly higher amounts of ^{131}I than those of voles caught further away. This is in agreement with Department of Energy predictions that this site was contaminated to a greater degree than the other two sites sampled. The highest level of ^{131}I detected from Site III was 11.4 pCi/thyroid and the mean for that site was 5.6 pCi/thyroid. The vole is proposed as a monitoring organism for ^{131}I contamination of ecosystems.

INTRODUCTION

The vole (Microtus spp.) is used extensively as a model organism for studying population dynamics (Kr74), nutrition (Ke78;Sh76;Sh75;Bar74;Br74;Sh74;Sh70), and bioaccumulation (Wi78;Ge77) because of its position in the terrestrial food chain, widespread distribution, abundance, small size and limited home range (Am69;Va69). These attributes also make the vole a good model for studies involving radionuclide pollution. The accident at the Three Mile Island Nuclear Plant (TMI) provided an opportunity to use voles to determine the extent and location of ^{131}I contamination of the environment.

Radiation from gas samples from the reactor containment building on 1 April 1979 showed that ^{131}I was one of the principle radionuclides released as a result of the accident at TMI. The amount of ^{131}I found in the containment building at that time was 6.3×10^{-2} microcuries/ml[2]. The first evidence of off site contamination by ^{131}I was detected in milk samples of domestic animals taken between 31 March and 4 April 1979. The maximum levels found in cow's and goat's milk were 36 and 41 pCi/L, respectively (Bat79). Our objective was to test the usefulness of (M. pennsylvanicus) as a monitoring organism by using this vole to determine the extent of ^{131}I contamination of the surrounding ecosystem by comparing these results to those obtained by conventional measures.

MATERIALS AND METHODS

The study area consisted of three sites in Dauphin County, Pennsylvania (Figure 1). Site I was 12.9 km NE of the damaged reactor and served as control. Site II was located 2.3 km E of the reactor, and Site III was 1.9 km NE of the reactor. The three sites contained similar meadow vegetation and had not been farmed for two years.

Sampling stations were placed at 2 m intervals along two 30 m lines separated by 8 meters. Approximately 240 m^2 were covered by the trapping grid at each site. Trapping was done between 6 April and 16 April, 1979 inclusively, and utilized two Sherman live traps baited with peanut butter at each sampling station. The traps were inspected and reset daily between 1000 and 1200 hours. Captured rodents were killed by diethyether and transported to the laboratory for analysis 24 to 48 hours after capture.

Each thyroid (< 4 mg tissue) together with a piece of trachea dissected microscopically was placed in 12 x 75 mm disposable culture tubes and analyzed for ^{131}I utilizing a Nuclear Chicago 1185 gamma counter with a 5 cm NaI scintillation crystal. Each sample was counted for ten minutes and background was determined over 600 minutes. Because of the number of samples from each site it was possible to make site to site comparisons. Counting efficiency was determined by counting absolute standards in the same geometry and matrix. Blanks for each sample were analyzed for ^{131}I content to

determine background counts. After subtracting the background from the count rate, the samples were corrected for ^{131}I decay, since the ^{131}I assays were conducted on different days during the trapping period. ^{135}Xe was among the radionuclides released from TMI and emits a Gamma of 360 KeV, which is within the window for ^{131}I. However, analyses of dissected thyroids taken from voles 24 to 48 hours after capture were specific for ^{131}I because ^{135}Xe is not concentrated in the thyroid, and because the half-life of ^{135}Xe is 9.2 hours and that of ^{131}I is 8.1 days.

RESULTS AND DISCUSSION

During the ten day trapping period, the number of voles caught was 20, 22 and 13 for Sites I, II and III, respectively. Only adult voles were captured, making age class comparisons impossible between the sites. Analysis for weight differences between sites, weight differences between sexes, and weight differences between site by sex revealed no significant effects. In addition, sex appeared to have no influence on the ^{131}I content of the vole thyroid.

Thyroids of voles from Site I contained no detectable ^{131}I, whereas those from Site III contained a significantly higher amount (Table 1). The highest level of ^{131}I detected at Site III was 11.4 pCi/thyroid, and the mean value obtained for this site was 5.6 pCi/thyroid. Analysis of variance revealed that the sites did differ with regard to

the content of ^{131}I in vole thyroids, $p < 0.01$. The Student, Newman, Keuls multiple-range-test, at $p \leq 0.01$, indicated that means for Sites II and III did not differ significantly, but that the ^{131}I in vole thyroids from Site III was significantly greater than for Site I. At $p \leq 0.05$ the content of ^{131}I in vole thyroids from Site III was significantly greater than that for animals from Site II. Analysis of variance is robust, but it assumes homogeniety of variance and normal distribution. Therefore, the data were tested for homogeniety of variance, skewness (g_1), and kurtosis (g_2). The calculated values of g_1 and g_2 for each site fell within allowable error and an F_{max} test found the variances to be homogeneous. To assure significance the data were analyzed again non-parametrically using the Kruskal-Wallis and Mann-Whitney tests. The results were essentially the same as those from parametric analysis. The between site differences in ^{131}I were significant at $p \leq 0.001$, and the Mann-Whitney test indicated that ^{131}I content of thyroids from Site III was greater than at either Sites I or II, $p \leq 0.01$ in both cases.

The ^{131}I content of thyroids from voles captured at Site III decreased during the sample period in a manner similar to that for the decay of ^{131}I. This would suggest that Site III received one dose of ^{131}I since such a decline would not be evident in a chronically exposed environment. Estimates of the distribution of ^{131}I released from TMI (Pa80) indicate that the major contamination of Site III occurred

between 28 March and 1 April 1979. Although the data describing the distribution of ^{131}I are in agreement with our findings insufficient vole data preclude a definitive statement. This points out the importance of taking successive samples from an environment thought to be contaminated by a single release of a radionuclide.

While no other systematic sampling of wild animals was undertaken in the vicinity of TMI, some scant evidence is in support of our findings. A vole captured 0.8 km east of TMI on 25 April 1979 was found to contain ^{131}I above background (Mo79a). In addition, a composite sample of rabbit thyroids from animals taken 1.6 to 4.8 km northeast of TMI on 24 April 1979 contained 161 pCi/gm ^{131}I, whereas none was detected in spleen, liver, and bone samples (Mo79b).

A number of explanations are possible for the ^{131}I content of vole thyroids at Site III. Herbicides such as 2,4 dichlorophenoxyacetic acid increases uptake of ^{131}I in animals (So58;F162), hence high levels of ^{131}I at Site III may have been due to herbicide treatments. However, none of the sites had been treated with herbicides for at least two years. Minute amounts of ^{131}I from local hospitals are discharged routinely into sewer systems (C177) and may ultimately contaminate rivers downstream. Since our animals and their food source do not obtain water from the Susquehanna River, the contribution from this source is negligible.

The mean concentration of ^{131}I in the thyroids of voles from Site III was 1866 pCi/gm, assuming a thyroid weight of 3 mg. Elevated levels of ^{131}I have also been found in thyroids from large mammals exposed to ^{131}I during uncontrolled releases from nuclear plants and weapons testing (Ma80;Pe71; Bara66;Be60;Ha59;Co57). While comparisons of thyroid ^{131}I content between studies are difficult, it is apparent that the relatively small vole is as effective as large mammals in monitoring ^{131}I.

In order to estimate the impact of radionuclide pollution on wildlife one must consider not only the external irradiation of organisms due to the dispersal of radioactive material outside of the organism, but also the internal irradiation due to accumulation of radionuclides in specific organs. While isopleth data (Bat79) would suggest that the voles at Site III were exposed to 18 mrem (Figure 1), the actual radiological burden to the vole thyroid may have been several orders of magnitude greater. For example, the mean dose of radiation from ^{131}I to the thyroid of voles from Site III was 210 mrem using an accepted computational procedure (Gr70). This estimate is conservative for the following reasons: a) the thyroid with the highest ^{131}I content received a dose of 420 mrem; b) thyroid weight may be less than 3 mg, hence the mean concentration of ^{131}I was greater than 1866 pCi/gm; c) voles were exposed to ^{131}I from 28 March while thyroid contents were determined as of 9 April, more

than a halflife after the major venting; d) undoubtedly other isotopes of iodine were vented and not included in our assay of ^{131}I; e) mixtures of iodine isotopes have been illustrated to be more harmful than a single radionuclide for a given dose (Bo80); and f) fetal thyroid concentrate radioiodine to a much greater degree than those of adults (Pe71). Clearly, thyroid glands of herbivores should be sampled to assess the radiological impact of radioiodine pollution of an ecosystem.

To test the validity of the vole as a monitoring organism we compared our data to Department of Energy estimates of the distribution of radionuclides in the vicinity of TMI. The mean ^{131}I content of vole thyroids for Site III was 2.5 times higher than that for Site II. Sample Sites II and III were placed on the exposure isopleth grid calculated for external whole body radiation exposure to the population around TMI by the Department of Energy (Figure 1); from these isopleth data we estimated the ratio of radiation exposure to be essentially the same as that for our vole data. Estimates of the distribution of ^{131}I released from TMI (Pa80) show that the relative degree of contamination of Sites II and III also agree with our vole data. The use of isopleths as indicators of exposure to ^{131}I is limited because (1) the isopleths are based on gamma radiation emitted mainly from noble gases and are not specific for ^{131}I, (2) sampling techniques used for calculating the isopleths are not standardized, and (3) the available isopleths do not take into

account radiation released after 3 April 1979. Because the vole thyroid, radiation isopleth, and ^{131}I distribution data are in accord, the vole is useful as an assay animal.

Domestic animals have been used extensively in monitoring radionuclide pollution with an intent to assess impact on humans. However, wildlife are better indicators of environmental contamination for several reasons. First, contamination as measured in domestic animals may underestimate actual environmental contamination due to their use of stored, and therefore uncontaminated, food. This was evident in the levels of ^{131}I in thyroids of domestic sheep fed stored food compared to those of sheep allowed to graze (Pe71). Secondly, foods of wildlife may not be the same as those for domestic animals and may accumulate ^{131}I to a greater degree than domestic crops (Pe71). For many reasons the vole is ideal as a monitoring organism. Because of the limited home range, 0.04 to 0.66 ha (Am69;Va69), and sedentary habits of the vole the precise geometry of environmental contamination can be determined. Voles adequately sample local habitats because they consume a variety of vegetation equivalent to one third of their body weight each day. Moreover, the vole is used as a model herbivore (Sh76;Sh75;Sh74). The abundance of voles enables one to destructively sample thyroid glands relatively easily and at little expense. The widespread distribution of voles permits one to employ them as monitoring organisms in a great number of situations.

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Others (Ma80,Pe71) have stressed the importance of using thyroid assays in evaluating the radiological impact of ^{131}I pollution. We found the content of ^{131}I in vole thyroid glands to reflect the extent of environment contamination in the vicinity of TMI. Hence it seems prudent to assay thyroids of voles living adjacent to sites where ^{131}I is vented to the environment. Moreover, the reservoirs and fluxes of ^{131}I in the vole should be investigated in the laboratory.

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Table 1. Iodine-131 activity in the thyroids of the meadow vole (*Microtus pennsylvanicus*) on 9 April 1979 in the vicinity of the Three Mile Island Nuclear Generating Plant

Site	Sample Size	I-131 Activity (pCi/thyroid) Mean*	Standard Error
I	20	0.0_a	0.8
II	22	$2.2_{a,b}$	1.1
III	18	5.6_b	1.2

*Means with the same subscript do not differ significantly; $p < 0.01$ ($F_{cal} = 6.76$).

Figure 1. Radiation (mrem) isopleths for the period 28 March through 3 April 1979 in the vicinity of the Three Mile Island Nuclear Generating Plant (adapted from Department of Energy; Bat79). Site I is 12.9 km Northeast of the reactor and is not illustrated in this figure, Site II is 2.0 km directly East of the reactor, and Site III is 1.9 km Northeast of the reactor.


50
100
100
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25
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200
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400
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TMI
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Appendix 3

NRC'S GROSS UNDERESTIMATION OF THE RADIOACTIVE RELEASES
AND POPULATION DOSES DURING THE TMI-2 ACCIDENT

by Seo Takeshi
Kyoto University Nuclear Reactor Laboratory

POPULATION DOSE ESTIMATES

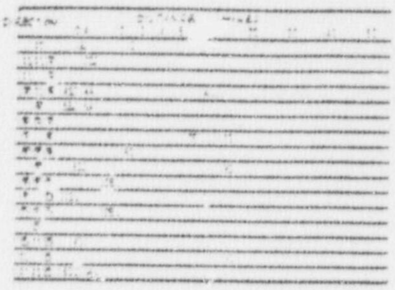

Table 1

Location of 20 TLD stations deployed by the utility, showing that there are no data at all for most of the 160 sectors (10 different distance divisions in the 16 directions). Estimates of the collective dose and quantity of released radioactivity based on this poor data cannot be accurate and should be considerably under the actual level.

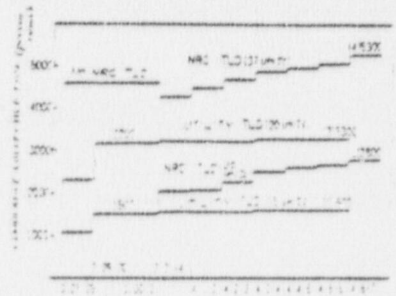

Figure 1

Estimates of the collective dose made by the ad hoc committee. The collective doses are significantly underestimated when TLDs are fewer in number. Moreover, there are no NRC data for the first 3 days of the accident. By correcting these deficiencies, the collective dose should be estimated to be at least as high as 16,000 person rems.

Here, four different sets of cumulative doses are shown:

(1) 1,300 person rems based on 15 TLDs deployed within the 8 mile-radius by the utility;

(2) 1,800 person rems based on 30 NRC TLDs in the same sectors as above;

(3) 3,300 person rems based on 20 TLDs of the utility;

(4) 5,300 person rems based on all the 37 NRC TLDs.

These differences clearly indicate that the number of dosimeters affects the dose estimation. It has been pointed out that the background radiation for the NRC dosimeters was evaluated at too low a level[4] and that the data for the first day, the period of March 31-April 1, is not reliable because of the poor maintenance of TLDs.[3] It would be reasonable to suppose that the background level was underestimated by 40 person rems per day. Consequently, the cumulative dose for the period of March 31 through April 6 is 460 person rems for the 30 NRC TLDs within the 8 mile-radius, and 770 person rems for all the 37 NRC dosimeters.

```
    1,800 person rems   NRC TLDs (30 units)
  - [illegible]         NRC TLDs on March 31st (30 units)
    ---------
      700
    - 240               (40 person rems x 6 days)
    ---------
      460 person rems

    [illegible] person rems   NRC TLDs 37 units
  - [illegible]               NRC TLDs on March 31st 37 units
    ---------
    [illegible]
    - [illegible]             ([illegible] person rems x 6 days)
    ---------
      770 person rems
```

As a result, the collective dose for the whole period based on the [illegible] NRC dosimeters is approximately [illegible] person rems, and [illegible] person rems for all the [illegible] NRC dosimeters, by adding [illegible] and [illegible] to the two different sets of doses recorded by the utility for the first three days.

Based on these figures, the dose for the first three days when NRC dosimeters were not being used should be estimated as follows. The ratio of the dose received by [illegible] TLDs of the utility for the period of March [illegible] through April [illegible] and that for March [illegible] through [illegible] is

$$\frac{[illegible] + [illegible]}{[illegible]} = [illegible].$$

And as the [illegible] utility TLDs are concerned, it is

$$\frac{[illegible] + [illegible]}{[illegible]} = [illegible].$$

The value of [illegible] is considered to be about the average. Then, if from the very beginning the [illegible] NRC TLDs had been set up, the dose of

[illegible] × [illegible] = 15,400 person rems

would be aquired for the first three days. To this figure of 15,4[illegible], the dose of [illegible] for the next [illegible] days is added and the total of 16,[illegible] person rems is consequently estimated to be the collective dose for the period of March [illegible] through April [illegible].

Although the above calculation is an estimation which ignores factors such as the possible changes in meterological conditions, there is evidence that the actual dose could probably be far greater since [illegible] dosimeters can hardly be considered sufficient in number.

NOBLE GAS RELEASE[illegible]

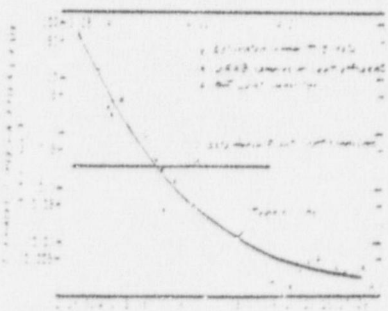

Figure [illegible]

Estimated noble gas release rate by the utility. The earliest two values based on data from the TLDs are underestimated as to be less than one fourth the actual level.

Evaluating the released amount of radioactive noble gases solely on the basis of this uncorrected TLD data, combined with the available meteorological information, cannot but result in an underestimation of the released levels. The final conclusion by NRC (NUREG-[illegible])[illegible] adopts their own preliminary estimation made in their July report of [illegible] × 10[illegible] Ci for the total amount of the noble gas releases. This July report presents a sequence of the noble gas releases (see Figure [illegible]) tabulated after a detailed calculation with a computer system. However, such detailed and precise calculations cannot correct an extensive loss of actual basic data.

As already shown, the collective dose for the period of March [illegible] through April [illegible] should, by correcting the apparent technical deficiencies, be estimated at around [illegible] person rems, while NRC provides the figure of [illegible] person rems for the same period. Here, the value of

[illegible] / [illegible] = [illegible]

[illegible] to correct the final estimation made by NRC of the amount of

noble gas releases. Thus, instead of 1.0 × 10[illegible] Ci (or 9 × 10[illegible] Ci in terms of Xe-133) should be the estimate for the amount of noble gases released.

IODINE RELEASES

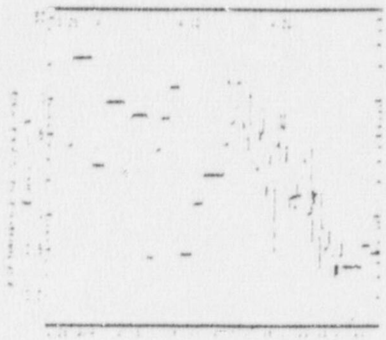

Figure 3
Radioactive iodine release rate based on TMI-2 vent monitors (charcoal cartridges)
It is clear that during the first two weeks the time intervals between cartridge changings were significantly longer than the following weeks. This indicates that for the first two weeks there should be a major underestimation in the iodine releases. The actual iodine quantity released during these two weeks may have been over several hundredfold of the level estimated by NRC.

It is clear that during the period before April 14 the average sampling intervals were seven to eight times longer than those during the period after April 14. Also it should be noted that after the sampling intervals became shorter, the declining gradient of the release rate was at a higher level by several tenfolds from that for the period between March 28 and April 14.

The Japanese Atomic Energy Commission's second report on the TMI-2 accident states that this sudden rising of the monitored iodine release is due to the filter changings done between April 12 and April 20. If this were the case, however, the effects of the replacement of filters would appear in peaks rather than in the overall increased level as shown in Figure 3. Moreover, according to the NRC staff report published in June, those cartridge changings were done not during April 12-20 but on April 20, 24-25, and May 23-24.

Therefore, it seems reasonable, instead, to explain this strange behavior of the monitored iodine releases as follows: For the first two weeks the charcoal cartridges were changed only every day or every two days because there existed a real danger that workers replacing the cartridges would be exposed to extremely high levels of radiation. This period was the most critical phase of the plant's status with an extremely high iodine concentration in the ventilation system. There also existed unusual amount of aqueous vapor. Under those conditions the absorbent capacity of the cartridges must have been rapidly minimized, resulting in the unusually low level of iodine concentration as shown in Figure 3. NRC and the utility did not make any corrections on those values recorded from the vent monitors in their estimates of the actual iodine releases. This is inexcusable.

Figure 2 and 3 indicate that on April 20 the approximate quantity of released iodine and noble gases were 1.4 μCi/sec and 4,700 μCi/sec respectively. The ratio of iodine to noble gases is

1 : 3,400.

The reason for obtaining the ratio from the time period of April 20 is that the noble gas radiation monitors in the plant ventilation exhaust which went off scale at a very early stage had been recovered by then so that direct measurement of noble gas releases were available, and that the time intervals between charcoal cartridge changings were short enough to provide relatively reliable data. Then, if we assume that the ratio of iodine to noble gases was relatively constant, we can estimate the

iodine/noble gas ratio for the period right after the beginning of the accident to be around

1 / 8,800

by taking the different half-lives of iodine and noble gases into account.

45x10^6 Ci

As the total amount of released noble gases is at least 4.5 × 10E7 Ci, the total released iodine should be estimated to be over 5,100 Ci. !

However, the above assumption of constant ratio between iodine and noble gases demands some discussion. First, iodine concentration in the effluent air depends on the temperature of the liquids. During the early stages of the accident the temperature is expected to have been considerably high so that the ratio would be much greater. For example, Table II-3-3 of NUREG 0600 provides the ratio of 1 / 700 for the time period a little before 7:00a.m. March 28.[5] Also on page II-3-20 of the same report it states that the major release of noble gases began around 7:00a.m. March 28 and that a few hours later the major iodine release started. Thus, it is very probable that after these few hours the ratio was much greater than 1 / 700 which corresponds to the quantity of 64,000Ci. It is also reported that even during routine operation these iodine filters had been used at TMI-2,[11] and there seems to be no reason to negate the value of 1 / 700.

Consequently, even the most conservative calculation would estimate the total iodine quantity released during the accident to be 5,100Ci. There remain reasons to expect that the released iodine quantity was far greater than 64,000Ci as indicated above.

Among the survey data in a task group report to the presidential commission,[3] there are some fragmentary data to challenge NRC's unconvincing estimation of released iodine. For example, (1) 1.2 × 10E-8 μCi/cc of airborne I-131 concentration recorded at 2:27p.m., March 28 in Middletown (1.6miles, north), and (2) 9.6 × 10E-6μCi/cc during 4:00-6:00p.m. at an off-site location, are hundreds or a thousand times larger than the values expected from the assumed release rate (several ten μCi/sec) on which NRC's estimation of the total iodine release (14Ci) was based.

Also, Lake Barrett reported the rate of 40 μCi/sec of iodine release at TMI-1 vent stack (6:00a.m., March 29).[13] Now, according to the July report,[6] the rate at TMI-2 vent stack was approximately a hundred times greater than TMI-1. This leads us to estimate that radioactive iodine was released into the atmosphere at the rate of 4mCi/sec from TMI-2 at that time of the accident. This value is approximately two hundred times greater than the quantity shown in Figure 3.

(Excerpts from the author's review published in Nuclear Engineering Vol.26,no.3)

1) Ad Hoc Population Dose Assessment Group (L. Battist et al.), "Population Dose and Health Impact of the Accident at the Three Mile Island Nuclear Station" (A preliminary assessment for the period March 28 through April 7, 1979), May 10 (1979).
2) ibid.
3) J. A. Auxier, C. D. Berger, C. M. Eisenhauer, T. F. Gesell, A. R. Jones and M. E. Masterson, "Report of the Task Group on Health Physics and Dosimetry to President's Commission on the Accident at Three Mile Island", Oct. 31 (1979).
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7) "Assessment of Offsite Radiation Doses from the Three Mile Island Unit 2 Accident" ibid.
8) "Second Interim Report on the Three Mile Island Nuclear Station Unit 2 Accident" ibid.
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12) J. A. Auxier et al.
13) L. H. Barrett, NRC memo unpublished, Mar. 29 (1979).
14) "Assessment of Offsite Radiation Doses from the Three Mile Island Unit-2 Accident" ibid.

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1972-73, NATO, Dept. Molecular Genetics, Univ. Ghent, Ghent, Belgium

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1969-72, Asst. Professor Microbiology, Univ. Kansas, Lawrence, KS.

1973-74, Chargé de Recherches, Dept. Molecular Biology, Univ. Geneva, Geneva, Switzerland.

1974-75, Research Associate Dept. Molecular Genetics, Univ. Heidelberg, Heidelberg, W. Germany.

1975-76, Exchange Scientist of US National Academy of Sciences, Inst of Biochemistry, ~~Prg~~ Prague, Czechoslovakia.

1976-78, Assoc. Professor of Microbiology & Surgery, Med. College of Wisconsin, Milwaukee, WI.

1979-present, Science Director, Environmental Cancer Prevention Center, Philadelphia, PA.

Publications

- 20 articles on molecular genetics
- Genetic predisposition to cancer in man (with A.G. Knudson)
- comment to venting of TMI-2 containment facility (NUREG-0662-Vol.2)
- comment to PEIS for TMI-2 cleanup (Nov, 1980)

RELATED CORRESPONDENCE

UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

BEFORE THE ATOMIC SAFETY AND LICENSING BOARD

In the Matter of	)	
	)	
METROPOLITAN EDISON COMPANY	)	Docket No. 50-289
	)	(Restart)
(Three Mile Island Nuclear	)	
Station, Unit No. 1)	)	

CERTIFICATE OF SERVICE

I hereby certify that copies of the Testimony of Bruce Molholt Regarding Aamodt Contention 4 (EP-1), which was hand delivered to Licensee's hearing office by Intervenor Norman Aamodt on February 23, 1981, were served upon the parties identified on the attached Service List by deposit in the United States mail, first class, postage prepaid, this 23rd day of February, 1981.

[signature]

Dated: February 23, 1981

UNITED STATES OF AMERICA
NUCLEAR REGULATORY COMMISSION

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Station, Unit No. 1)	)	

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