

**A REPORT ON THE  
HEALTH STUDY OF RESIDENTS  
LIVING NEAR THE LIPARI LANDFILL**

**New Jersey Department of Health  
Division of Occupational & Environmental Health  
Environmental Health Service**



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Governor**

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Commissioner of Health**

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A P R O J E C T T E A C H A C T I V I T Y

Community representatives and staff from several agencies have been collaborating on the health study since it began in 1986. The study was undertaken to assess health outcomes as a function of crude distance from the Lipari Landfill. Governor Kean's FY '87 initiative, Project TEACH (Team for the Evaluation and Assessment of Community Health) was developed in part from the lessons learned in the communication of this health study. The purpose of Project TEACH is to enhance the Department of Health's efforts to respond to community health concerns.

\* \* \* \* \*

Upon compiling the data, the New Jersey State Department of Health (NJDOH) felt that a responsible public health process would entail involving the community in appointing an outside review panel which would evaluate the study methods, results, and provide comments regarding recommendations for further efforts to address community needs.

The Panel met with NJDOH study staff on January 11, 1989. A report was prepared by the Panel as a result of that meeting and the commitment made to the Lipari Health Subcommittee. Many of the specific suggestions for data presentation or interpretation made by the Panel, as well as a copy of the Review Panel's written Consensus Statement have been incorporated into the printing of the Report. A spokesperson, selected by the Panel to represent this consensus, will be present during a presentation to the community on February 2, 1989.



ACKNOWLEDGEMENTS

We would like to thank the following individuals and agencies for their valuable assistance, cooperation and technical expertise throughout the course of the study which made this report possible:

Gloucester County Health Department

NJDOH Division of Vital Statistics and  
Registration

NJDOH Division of Cancer and Epidemiology  
Cancer Registry

NJDOH Division of Community Health Services  
Special Child Health Services Program

NJDOH Center for Health Statistics

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## CITIZENS' GUIDE TO THE 1989 LIPARI HEALTH REPORT

### WHY WAS THIS STUDY DONE ?

This study was done by the New Jersey Department of Health (NJDOH) at the request of the Pitman Alcyon Lake-Lipari Landfill Community Association (PALLCA). PALLCA was set up by a group of local citizens concerned about the health effects of living near the landfill. The landfill began operating in 1958 and ended in 1971 because of neighboring residents' complaints regarding odors, respiratory problems, headaches, nausea and dying vegetation. The NJDOH met with PALLCA in February of 1986 and discussed ways in which community health concerns could be addressed. The Lipari Health Subcommittee determined that the outcomes studied would be cancer and birthweight. Birth certificates and the state cancer registry records, both of which are compiled statewide by the NJDOH, were accessible for this study.

FOR MORE DETAILS, PLEASE SEE PAGES: 1-3, 12-15

### WHAT ARE THE FINDINGS ?

The study examined the number and new cases of many types of cancer, that occurred from 1980-1984 in individuals living around the landfill.

Several types of cancer that might be related to toxic chemical exposures were examined. The rates for most of these, including respiratory cancer, in the four municipalities surrounding the landfill were found to be low when compared with statewide rates. However, there is a slight increase in the number of cases of leukemia (cancer of the blood forming organs) for those living near the landfill. Because of limited information available on individual exposures, this finding is not scientifically conclusive as to whether there is an actual relationship between leukemia and living near the landfill.

The study also examined the weight of infants at birth. Birth records from three five-year periods were examined (1961-1965, 1971-1975, 1981-1985). A lower average birthweight (about 2 1/2 ounces ) was found. A higher proportion of infants born after a usual nine month pregnancy weighed less than 5 1/2 pounds (considered to be low birthweight). This was found among infants born to families living close the landfill during the period of 1971-1975.

FOR MORE DETAILS, PLEASE SEE PAGES: 15-20, 29-30

#### WHAT DO THESE FINDINGS MEAN ?

These findings suggest that the most adverse health effects occurred during the 1970's, when exposures to hazardous substances were probably the heaviest. We know that certain hazardous chemicals were present in the landfill but it was not possible in this study to determine an individual's exposure to them. Most cancers have a long latency period (the time between exposure and the onset of symptoms). The latency period for cancer may be as short as a few years or as long as decades. The time period of the study was too short (1969-1984) from the period of probable exposure for most cancers to show up. Given this, it is possible that more cancer cases may occur later. Even though it may not become clear whether any are related to exposure, it will be necessary to closely monitor new cases of cancer over time.

Women giving birth between 1961-1965 probably had little or no exposure to hazardous materials from the site. Those giving birth between 1971-1975 may have been exposed during their pregnancies. Although those giving birth between 1981-1985 were probably not highly exposed during their pregnancies, they may have come in contact with toxic substances from the site at an earlier time.

It is very difficult to determine past individual exposure from the landfill over time. Also, other factors such as personal lifestyle, occupation, prenatal care, maternal health, and socioeconomic status are known to influence health outcomes but could not be studied from existing records.

FOR MORE DETAILS, PLEASE SEE PAGES: 21-25, 29-30, 32, 41, 45, 52, 55, 57-59

#### WHAT IS BEING DONE TO PROTECT THE COMMUNITY AGAINST POSSIBLE EXPOSURE FROM THE SITE ?

The highest exposures are thought to have occurred in the past. Since 1982, Phase I has been initiated by the United States Environmental Protection Agency (USEPA). Clean-up during this phase, has included several activities designed to decrease exposure. These activities include the installation of a chain link fence, a slurry cutoff wall and membrane cap to limit movement of contaminants from the site, gas vents, and surface water runoff controls. The second step, Phase II, plans to clean the landfill by building an on site treatment center for the purpose of removing water-transportable contaminants present within the contained area. The third step, Phase III, off site clean-up, is designed to permanently remove landfill related contaminants in the adjacent off-site areas, including Chestnut Branch Marsh and Alcyon Lake. The New Jersey Department of Environmental Protection (NJDEP) has assisted the USEPA in planning these activities.

FOR MORE DETAILS, PLEASE SEE PAGES: 3-6

**WHAT ARE THE FUTURE PLANS OF THE NEW JERSEY DEPARTMENT OF HEALTH ?**

The NJDOH will continue to collaborate with the community in defining future activities related to this study. The NJDOH will continue monitoring the incidence of cancer and low birthweight in the four communities and municipalities around the site.

FOR MORE DETAILS, PLEASE SEE PAGES: 57, 60-61

**WHO WAS INVOLVED IN THIS STUDY ?**

The Lipari Health Subcommittee consists of the following: Pitman Alcyon Lake-Lipari Landfill Community Association (PALLCA), New Jersey Department of Health (NJDOH) Environmental Health Services (EHS), United States Centers for Disease Control (CDC), Gloucester County Health Department, the Pitman Environmental Commission and the Pitman and Mantua Boards' of Health. The United States Environmental Protection Agency (USEPA), and the New Jersey Department of Environmental Protection (NJDEP), although not part of the Health Subcommittee, did provide assistance as needed.

FOR MORE DETAILS, PLEASE SEE PAGES: 1-2

**WHOM CAN I CALL IF I HAVE HEALTH QUESTIONS ?**

We suggest that you inform your personal physician during a routine office visit or checkup that you live near or around the landfill. Any health questions regarding the Lipari landfill will be addressed by the NJDOH staff at 609-633-2043.



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## EXECUTIVE SUMMARY

An epidemiological study of adverse health effects from potential chemical exposures related to Lipari Landfill in Gloucester County was conducted from 1986 to 1988 through the collaboration of the following: representatives from four adjacent communities (Glassboro, Pitman, Harrison and Mantua), local government agencies and the Environmental Health Service (EHS) of the New Jersey Department of Health (NJDOH). The health status of the community was assessed using the following existing records: birth records between 1960 and 1985 and cancer registry data from 1980 to 1984. This report provides the background and history of the site and the concerns of the four communities and presents the findings and recommendations resulting from the collaborative epidemiologic study conducted around Lipari.

The Lipari Landfill, located in Mantua and bordering Pitman, Glassboro and Harrison, is ranked number one on the United States Environmental Protection Agency's (USEPA) National Priorities List (NPL). The landfill was the source of hazardous leachate which had migrated from the landfill into two nearby streams and a lake in the vicinity of residences, schools and playgrounds. Operation of the landfill began in 1958 and ended in 1971 because of residents' complaints regarding odors, respiratory problems, headaches, nausea and dying vegetation. Although the nature and quantity of the wastes that were received at the landfill are not known due to inadequate maintenance of records, numerous chemicals have been identified at the site. The finding of bis(2-chloroethyl) ether (BCEE), a known animal carcinogen and

suspected human carcinogen, was of particular environmental and public health concern. Other carcinogens identified include benzene, methylene chloride and arsenic. Pitman residents living in a nearby housing development had the highest potential for exposure, because hazardous leachate flowed into a stream behind the development for many years prior to remediation. The primary route of potential human exposure to the contaminated leachate was inhalation of contaminated ambient air. Exposure to landfill contaminants via drinking water probably did not occur, largely because most residents use public wells which have remained unaffected.

#### LIPARI HEALTH SUBCOMMITTEE

In June of 1985 the Pitman Alcyon Lake Lipari Landfill Community Association (PALLCA) was formed by a group of active local residents and community officials who were concerned with the possible health effects from the landfill on the four surrounding communities. They organized in order to crystallize attention and gain help from county, state and federal agencies. The Lipari Health Committee, consisting of representatives from the Centers for Disease Control (CDC) and the Agency for Toxic Substance and Disease Registry (ATSDR), USEPA (Region II), New Jersey Department of Environmental Protection (NJDEP), NJDOH, Gloucester County Health Department (GCHD) and the four communities, was called into existence in January 1986 at the request of PALLCA. Subsequently, a subcommittee to evaluate specific health-related issues was requested and the Lipari Health Subcommittee was formed in February, 1986. The Health Subcommittee consists of representatives from CDC, GCHD, NJDOH EHS, the Pitman Environmental Commission, PALLCA and Pitman

and Mantua Boards of Health. The USEPA and NJDEP, although not part of the Health Subcommittee provided assistance as needed. The Lipari Health Committee was formed [1] to keep the community informed of planned activities and their results and [2] to answer questions relating to health effects of potential exposure to chemicals from the landfill. The objectives of the Lipari Health Subcommittee included [1] to provide a forum for the public to express their health concerns to local, state and federal agencies, [2] to promote a common understanding of these concerns among all interested parties and [3] to explore how health concerns could be best addressed with available techniques. The Subcommittee met on a regular basis in an effort to keep the affected community informed about study activities and findings.

The Subcommittee decided that the possibility of exposure to toxic chemicals from the landfill warranted studies to determine if any measurable health impact had occurred. The public had been especially concerned with respiratory cancer and birth defects and requested that an exposure registry be created as a first priority. EHS presented the advantages of using existing State databases, rather than building new ones, to the rest of the Subcommittee, which agreed with this approach and chose to study [1] birth certificate data for low birthweight and [2] cancer registry data for selected cancer outcomes for evidence of exposure-related health effects. The EHS agreed to conduct the study of cancer and birthweight databases with the active participation of the other groups represented by the Subcommittee.

Although there was strong community interest in birth defects and spontaneous abortions, the existing data which were collected and reviewed

were of inconsistent quality and/or not easily accessible, which precluded their inclusion in the study. Other possible health endpoints, such as neurological or other medical problems, could not be studied due to the absence of any existing databases.

#### EXPOSURE ASSESSMENT

No quantitative estimates of air contaminants are available for the years prior to 1984. Meteorologic data were consistent with expectations that homes closest to the site were likely to have the most affected ambient air, in that the wind blew predominantly north to northwesterly in the winter (toward the homes), and in a south to southwesterly direction (toward unoccupied orchards and farmland) in the summer. Since inhalation is the most probable route of exposure for residents in this area, radial distance from the landfill source was chosen as a surrogate indicator of exposure. The rationale for this was the premise that as distance increases, air contaminant concentrations decrease. The putative population at risk was encompassed by two concentric rings around the source with radii of 1.0 (Area 1) and 2.5 kilometers (Area 2), creating two exposure groups. Those residents living beyond 2.5 kilometers, but still inside township borders, comprised a third group (Area 3). Wherever internal comparisons were made in the study, a relatively "exposed" group (Area 1) was compared to a relatively "unexposed" group (Areas 2 & 3). Where New Jersey statewide rates were used for comparison, Areas 1, 2, and 3 were considered cumulatively.

## DATA COLLECTION AND ANALYSIS

### CANCER

Information on cancer was obtained from all available cancer incidence data (New Jersey Cancer Registry data, 1980-1984) allowing for a latency period of approximately 15 years. Although this is sufficient for short latency cancers such as leukemia, many cancers (e.g. lung cancer) are thought to take longer than 15 years to develop after initiation of exposure. Nevertheless, cancer incidence in the study areas relative to expectations of cancers (based on cancer rates found in the entire state) were calculated for the high and low exposure groups in the form of Standardized Incidence Ratios (SIRs). Total cancer and nine site-specific cancers were evaluated for each study area designation; three of these were statistically significantly lower than expected and none were significantly elevated based on comparison with the average New Jersey rates.

In the area closest to the dump site, the highest but not statistically significant SIR was for leukemia [SIR = 1.97, 95% Confidence Interval (CI) = 0.72, 4.29]. This ratio was based on six observed cases, compared to 3.044 expected. Leukemia, a cancer with the shortest time between exposure and effect of all the cancer types examined, might be expected to have the highest rate if exposure-related carcinogenesis were occurring. The absence of important information on factors that may affect the rate (including migration patterns of residents, utilization of health care services out-of-state, and lifestyle risk factors) weakens the evaluation of these results. This, in addition to the short fifteen-year lag between onset of exposure and evaluation of cancer outcomes, makes it necessary to continue

surveillance and to conduct follow-up studies should any new cancer patterns become apparent.

#### LOW BIRTHWEIGHT

Low birthweight is a known risk factor for health problems in early life, and low birthweight is also believed by environmental health scientists to be a plausible indicator of health effects which may be due to exposure to toxic substances. Birth certificates were collected for children born during the first five years of the 1960's, 1970's and 1980's respectively. Due to few births among non-whites, only white births could be analyzed. The birth certificates did not provide information on several very important predictors of birthweight such as smoking, height and weight of the mother, maternal illnesses, parental occupation, socioeconomic status and drinking habits. Therefore, these factors could not be included in the study, and a spurious result may have occurred due to the impact of one or more of these unmeasured factors. In all analyses, statistical adjustments were made for the following factors available on the birth certificate: sex and gestational age of the child, maternal age, education, parity and prenatal care, previous stillborns and complications during pregnancy.

During the period 1961-65, when exposure from the site was probably minimal, if at all, there was no statistically significant ( $p > 0.10$ ) difference between the two areas (Area 1 versus Areas 2 & 3 combined) on average birthweight. Differences between the two areas in proportions of low birthweight were not consistent across the sexes.

However, in the 1970's, when exposure may have been heaviest, average birthweights for both births over 27 weeks of gestation and for term births (38 - 42 weeks gestation) in Area 1 were lower (about 66 and 74 grams or 2.3 and 2.5 ounces respectively) than average birthweights in Areas 2 & 3. These results were statistically significant ( $p < 0.05$  in the multiple regressions) and are consistent with the hypothesis that exposure to contaminants from the site is associated with lower average birthweight. In addition, a higher proportion of low birthweight babies occurred both among births over 27 weeks of gestation and among term births in Area 1 than in Areas 2 & 3 (OR = 1.6, 90% CI = 1.0, 2.6; OR = 2.0, 90% CI = 1.1, 4.0 respectively), consistent with the hypothesis that exposure to contaminants from the site is associated with low birthweight when other potential risk factors are controlled.

In the 1980's, conflicting results occurred among male and female term babies. For example, among term births, males in Area 1 had an average 47 grams (1.7 ounces) lower birthweight than in Areas 2 & 3, but females in Area 1 had an average 76 grams (2.7 ounces) higher birthweight than in Areas 2 & 3. Area 1 compared to Areas 2 & 3 had higher proportions of low birthweight among males born over 27 weeks gestation (OR = 2.2, 90% CI = 1.1, 4.2) and among term births (OR = 1.7, 90% CI = 0.7, 3.8) but not among females born over 27 weeks of gestation (OR = 0.5, 90% CI = 0.2, 1.5). These results are difficult to interpret. It is not clear why an exposure to environmental contaminants would affect birthweight among males differently than among females. If exposure to the landfill is related to adverse health outcomes, we would expect to find a smaller difference between the two areas (regardless of sex) during this period than during the 1970's, since remedial

measures were taken in the middle of the 1981-85 period. Most likely, the finding of birthweight differences between the two areas during the 1981-85 period is a spurious one.

The analysis of birthweight overall indicates that, during the period when exposure to site contaminants was probably the heaviest, babies born to residents living close to the site (Area 1) had a lower average birthweight than babies born to residents living further from the site (Areas 2 & 3). This finding may be explained by factors which could not be studied in this investigation.

#### CONCLUSIONS AND RECOMMENDATIONS

No adverse health outcomes could be conclusively linked to exposure to contaminants from the Lipari Landfill in this study. The largely negative cancer findings cannot be relied upon to indicate an absence of hazard because of the insufficient period of observation for most cancers except leukemia, the possibility of underreporting and the crude proxy exposure areas used. Further, the inability to collect information on smoking, alcohol use, and other such factors which can greatly influence birthweight prevent any strong conclusions about the relationship of the landfill to birthweight in its vicinity. However, the findings on leukemia and low birthweight suggest the possibility that exposure-related adverse outcomes may have occurred among residents near the landfill during the period 1971-75 when exposures were probably heaviest.

Considering the toxicity of many of the contaminants involved, the NJDOH recommends that all further remediations, presently proposed by the USEPA, be carried out in a timely manner and with careful monitoring to limit further exposure. The NJDOH is committed to continuing surveillance of cancer, low birthweight and birth defects around the Lipari site and plans to initiate further discussions with the local community and the Subcommittee in order to define exactly what activities should be pursued.

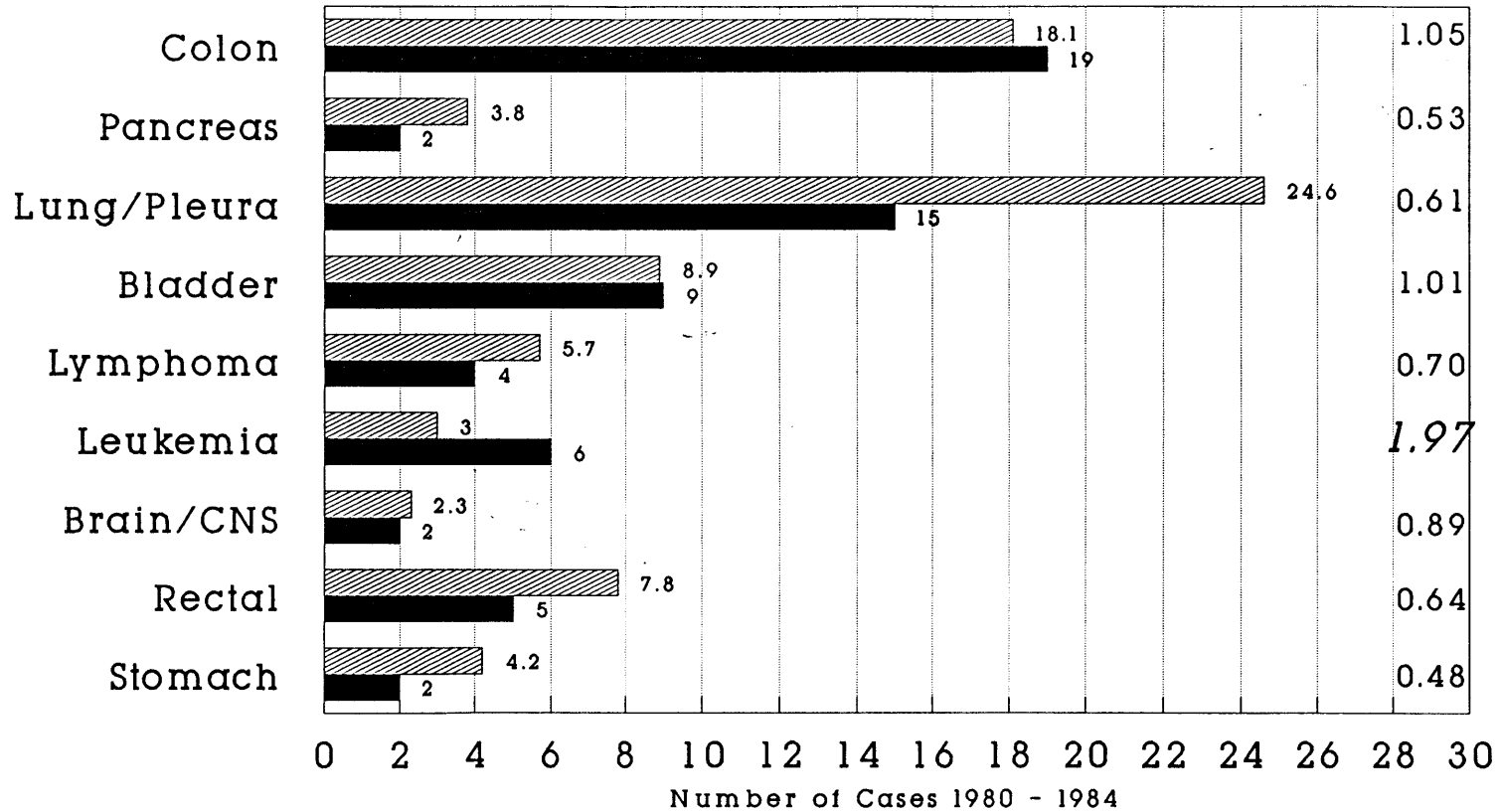
Given the suggestive but non-conclusive nature of the results of this study, medical counseling on related issues will be available to area residents, but no special diagnostic or other clinical procedures are indicated.



*Health Study of Residents Living Near the Lipari Landfill*

**Cancer Cases Observed & Expected in Area 1 and Standardized Incidence Ratios**

S I R  
(Ratio of  
Observed  
to Expected \*)



 Expected Based on N.J. Rates     
  Observed (Area 1)

\* all ratios are not significant ( $p < 0.05$ )

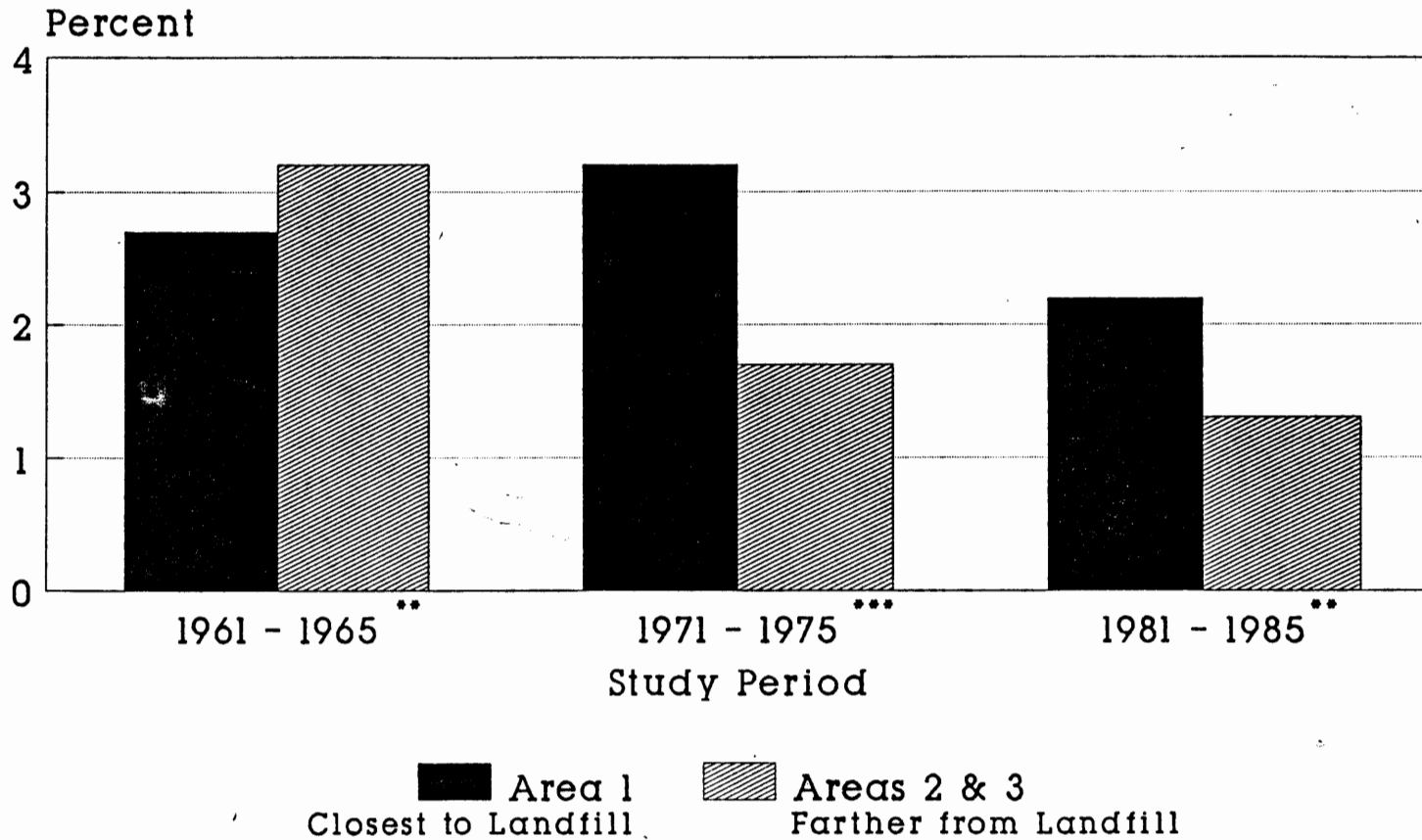
xiv

Figure 2



*Health Study of Residents Living Near the Lipari Landfill*

Percentage of Low Birthweight \*  
in each study period



\* Low Birthweight is < 2500 g (5.5 lb)  
\*\* Not Statistically Significant  
\*\*\* Stat. Significant, 0.10 > p > 0.05

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Figure 3



Location of Gloucester County and Lipari Landfill in  
New Jersey

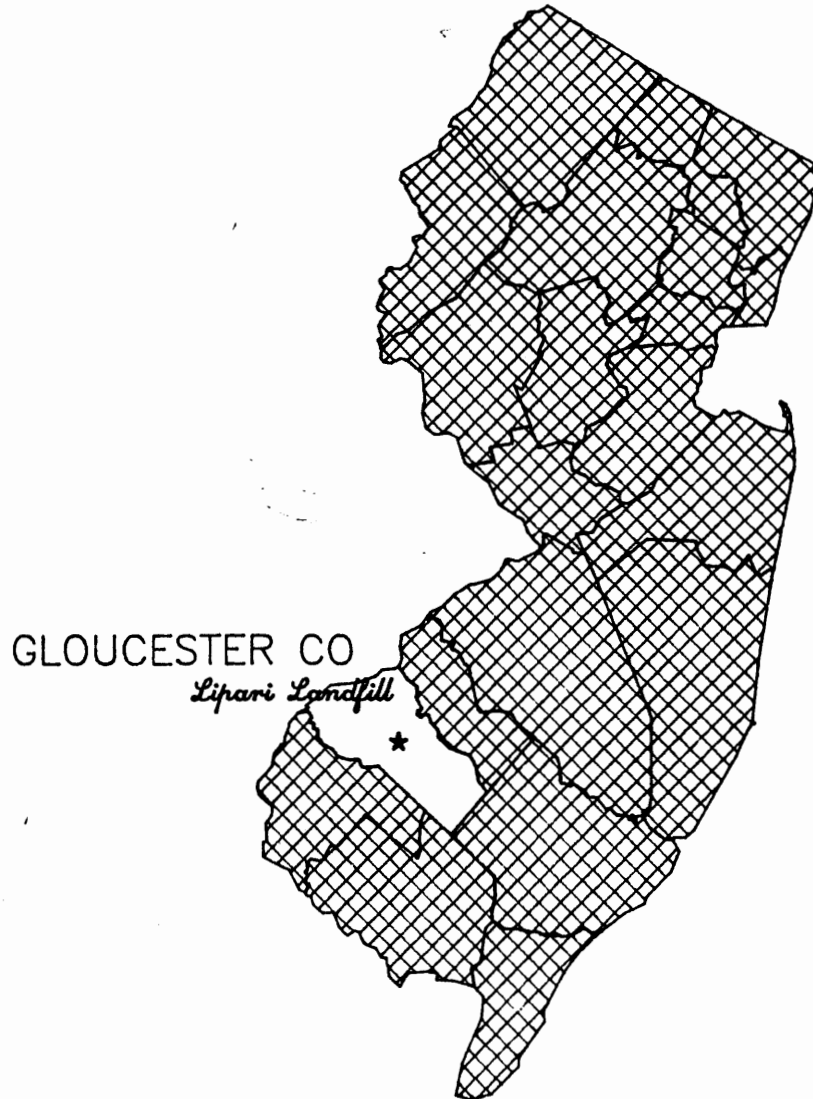
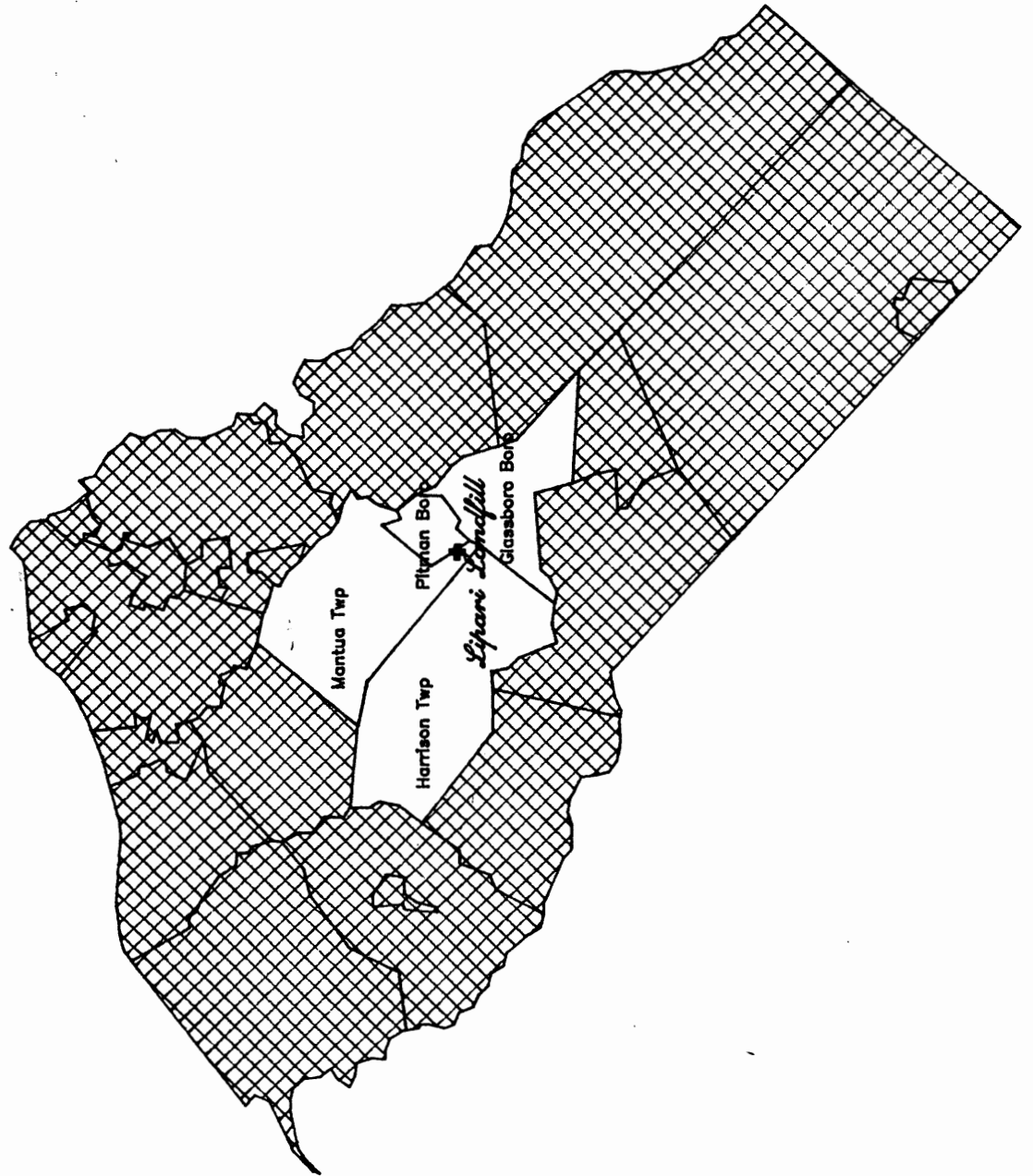




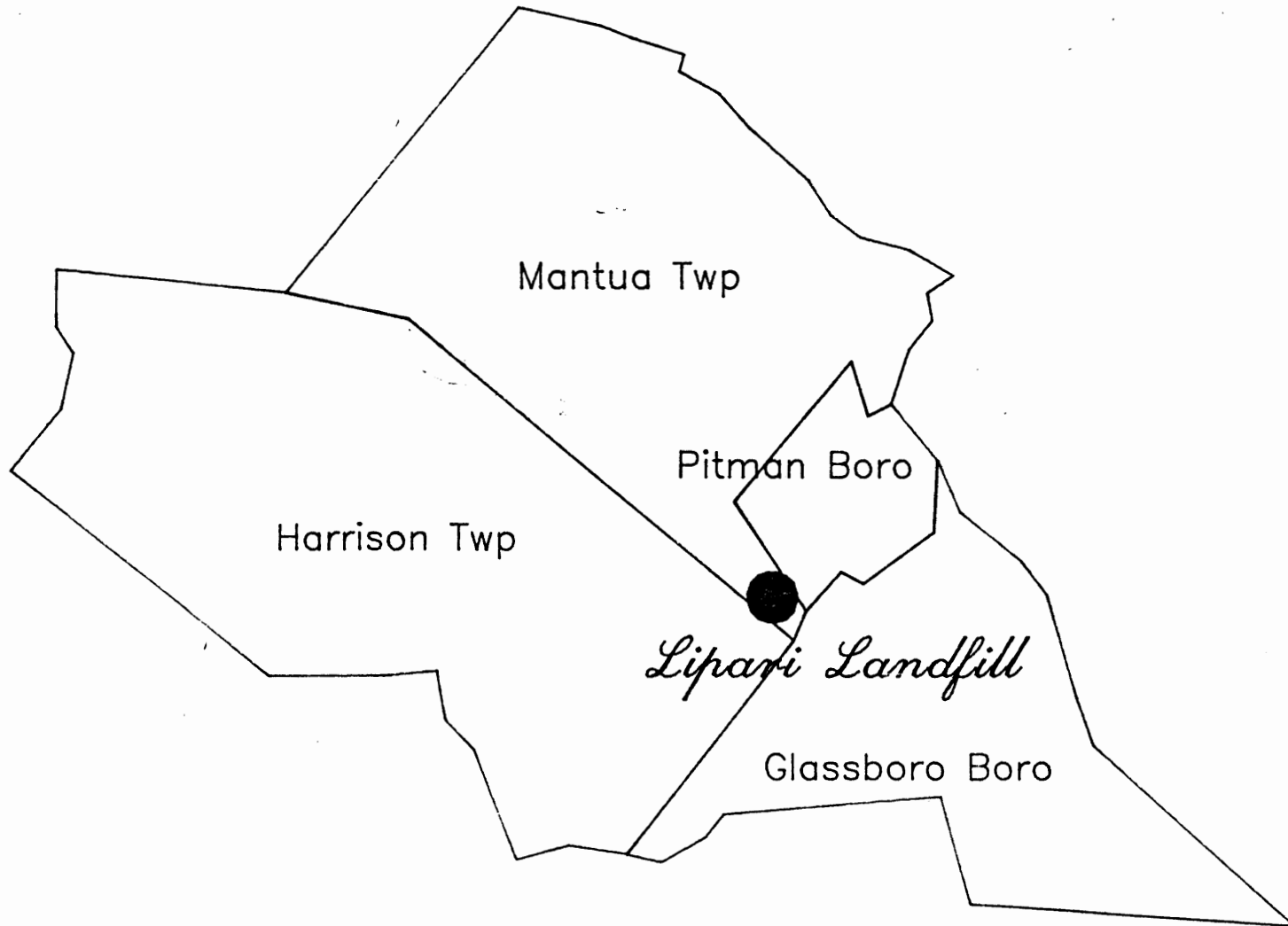
Figure 5

**Location of Lipari Landfill  
in Gloucester County**





## The Four Municipalities Surrounding Lipari Landfill





## I. INTRODUCTION

### A. HISTORY OF HEALTH CONCERNS

The Lipari Landfill, located in Mantua Township, Gloucester County, New Jersey, is ranked number one on the U.S. Environmental Protection Agency (USEPA) National Priorities List (NPL). The 15-acre site lies adjacent to the mixed agricultural and residential townships of Pitman, Harrison, Glassboro and Mantua. Both Pitman and Glassboro (population 9,691 and 14,644 respectively, 1980 U.S. Census) are located within one mile of the site. In the town of Pitman, there are houses located within several hundred feet of the site. According to the on-site Remedial Investigation/Feasibility Study (RI/FS) by the USEPA (1985), a major hazard identified with the landfill is the presence of bis (2-chloroethyl) ether (BCEE), a suspected human carcinogen and a known animal carcinogen. Other chemicals of environmental as well as public health concern include benzene, toluene, methylene chloride, 1,2-dichloroethane, phenol, chromium, nickel, mercury, lead, selenium, arsenic and silver.

Because of the concerns of the residents living close to the landfill, a citizens' group was established in June, 1985, the Pitman Alcyon Lake Lipari Landfill Community Association (PALLCA). In January of 1986, PALLCA requested that an interagency committee be established to examine health issues associated with the site. The Lipari Health Committee was then formed and included representatives from the Centers for Disease Control (CDC) and Agency for Toxic Substance and Disease Registry (ATSDR), USEPA, New Jersey Department of Health (NJDOH), New Jersey Department of Environmental Protection (NJDEP), Gloucester County Health Department (GCHD), and the four communities. In February of 1986, PALLCA requested that a Health Subcommittee be established to evaluate specific

health issues that may be associated with the landfill. The Lipari Health Subcommittee was thus formed consisting of representatives from the CDC, NJDOH, PALLCA, GCHD, the Pitman Environmental Commission and the Pitman and Mantua Boards of Health. The NJDEP and USEPA, although not members of the Lipari Health Subcommittee, would provide assistance as needed. Members of the Subcommittee soon requested that an exposure registry be instituted and a study be performed to determine if there was a discernible health impact to residents who live in the vicinity of the landfill. The Subcommittee felt that respiratory cancer and birth defects were critical outcomes to document and explore. Representatives of the Environmental Health Service (EHS) of the NJDOH urged the rest of the Subcommittee to consider, instead, the advantages of using existing state databases rather than creating and maintaining a new exposure registry. The EHS recommended that a cross-sectional historical study be performed to assess the status of the communities' health by evaluating available records from the state cancer registry, birth certificates, and birth defect registry. Spontaneous abortion data from area hospitals and local school absenteeism records were also suggested by the membership as supplemental data and was to be collected by the GCHD and community volunteers.

The Subcommittee agreed with this approach and chose to analyze birth certificate data (low birthweight) and cancer registry data (selected cancer outcomes) for evidence of exposure-related health effects. Although there was strong community interest in studying birth defects, spontaneous abortions, and school absenteeism, the lack of easily accessible data and the inconsistent quality of existing data precluded the study of these health endpoints. The NJDOH EHS agreed to carry out the studies of cancer and low birthweight with the active participation of the other groups represented by the Health Subcommittee.

## B. SITE DESCRIPTION AT THE BEGINNING OF THE HEALTH STUDY

### 1. Description of Site, Dumping History and Remediations

The site of the landfill was purchased in 1958 by Nick Lipari for use as a sand and gravel pit. According to the USEPA (1985), trenches were excavated to a depth of 6 to 15 feet for removal of the sand and gravel and were then backfilled with municipal refuse and household wastes, liquid and semisolid chemical wastes, and other industrial wastes. Liquid wastes were emptied from containers and dumped into the landfill from 1958 to 1969, and solid wastes were disposed of until May 1971. It has been estimated by the USEPA (1985) that the heaviest period of dumping occurred between 1967-1969. Therefore, the late 1960's onward is likely to be the time of greatest residential exposure to the landfill effluents. Although no detailed records of the quantity or type of waste disposed at the site were kept, wastes reported by the USEPA to have been disposed included solvents, paint thinners, formaldehyde, paints, phenol and amine wastes, resins, and dust collector residues (USEPA, 1985).

In May 1971, local residents expressed numerous complaints about odors emanating from the Lipari Landfill and Alcyon Lake which were investigated by NJDEP Solid Waste Management and Department of Water Resources that same month. NJDEP personnel observed leachate seeping from the eastern and northeastern sides of the landfill and ultimately discharging into the Chestnut Branch stream, which empties into Alcyon Lake about 1500 feet downstream of the site. The landfill was closed by the NJDEP in June 1971. Remedial action at the site was not initiated until July 1982 when a chain link fence was installed to discourage nearby residents from entering the contaminated marsh area along Chestnut Branch. In 1983, a slurry wall was installed which completely

encircled the site and a 40-mil thick synthetic cap of high-density polyethylene was placed over the site to exclude infiltration of precipitation and surface waters. Prior to fencing of the site and the construction of the slurry wall and cap, the site was frequented by children of the area and used as a motorcross bike trail. Naturalists, joggers and hikers were also reported to have used the site from time to time. With the encapsulation of the landfill exposure from onsite contamination was presumably reduced.

## 2. Recent Status of Contamination and Exposure

The USEPA began investigating the possible migration of contaminants on-site and off-site in 1984. The exposure information collected during this investigation provided a qualitative, but not a quantitative, picture of the exposures faced in earlier years. Information pertinent to prior exposure was obtained from the 1985 On-Site Feasibility Study and the Off-Site Remedial Investigation and is summarized in the Appendix.

The chemicals of most concern identified in USEPA's 1984 on-site study were benzene, bis(2-chloroethyl) ether (BCEE), chloroform, 1,2-dichloroethane, ethylbenzene, 4-methyl-2-pentanone, toluene, total xylenes and a number of metals (arsenic, chromium, lead, mercury, nickel, and zinc).

BCEE and benzene are of particular concern because of their probable routes of exposure (inhalation) and possible carcinogenicities. Other possible routes of exposure include contact with nearby contaminated soils, swimming in the lake or eating fish from the lake. The off-site soil investigations concluded that the marsh west of Chestnut Branch was contaminated with organics including benzene and BCEE. The other sites investigated were not notably contaminated.

The surface water and sediment investigations revealed BCEE in downgradient surface waters and sediments in Rabbit Run. Benzene, arsenic and mercury were also found in some surface water and sediments.

Air quality investigations conducted by the USEPA in 1986 (NUS Corporation, April 1988) indicated that there were measurable volatile organics (including benzene, BCEE, 1,2-dichloroethane) in the vicinity of the marsh and that some of these contaminants probably migrate in air to nearby neighborhoods.

The potential exposure routes included inhalation of ambient air, particularly benzene and BCEE, which are carcinogenic. Other possible routes of human exposure were through swimming or wading in the lake and eating fish which were caught there. In addition, some contaminants in the landfill could be absorbed through the skin of any person who came into direct contact with soil at the site. USEPA used a reasonable maximum exposure scenario coupled with carcinogenic potency estimates for the landfill effluents to obtain an estimated lifetime excess cancer risk of 6 per 10,000 exposed for inhalation of ambient air near the Howard Avenue Security Fence (USEPA, 1987).<sup>1</sup>

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(1) It should be noted that the average detection limit and average quantitation limit of BCEE was 4 ppb and 12 ppb, respectively. Assuming a 70 kg adult inhales 20 cu m/day, the calculated cancer risk from inhaling BCEE at these limits are 7 per thousand and 22 per thousand, respectively. On September 26, 1987, 1-3 ppb of BCEE was detected on Howard Avenue and selected backyard locations. The calculated cancer risk from inhaling this concentration for 70 years is in the range of 2 to 6 per thousand. It is noteworthy that BCEE was even detected, given the detection limits that were used, that it was detected in the Fall, and that the total cancer risk would be greater if one totaled the risk from individual chemicals.

Exposure to contaminants from the landfill most likely does not occur through drinking water. The majority of Pitman residents receive drinking water from three municipal wells drawn at a depth of 525 feet in the aquifer. Several homeowners do have private wells, however (USEPA, 1987). There was no evidence of contamination of public wells when municipal wells in a two mile radius from the landfill in all four townships studied were examined for contaminants (USEPA, June 1987). Most private wells in the vicinity of the landfill are either very deep or are upgradient of the landfill.

### 3. Chronic Toxicity of Compounds Identified

#### a. Birth Outcomes

There are very few studies available on the relationship between most of the compounds found at the Lipari Landfill (benzene, BCEE, chloroform, 1,2-dichloroethane, ethylbenzene, 4-methyl-1,2-pentanone, toluene, xylenes, arsenic, chromium, lead, mercury, nickel and zinc) and low birthweight in pregnancy outcomes. Positive results (low birthweight caused by exposure) have been reported for cadmium in animals (Rudolph and Swan, 1986; Ali, Murthy and Chandra, 1986), lead in humans (Heinrichs, 1983), benzene in animals (Davis and Pope, 1986) and xylene in animals (Mirkova, Zaikov, Antov, Mikhailova and Khinova, 1983). Negative results were reported in studies of the relationship between low birthweight and cadmium in humans (Huel, Everson and Menger, 1984), lead in rats (Winneke, Lilienthal and Werner, 1983) and xylene in rats (Rosen, Crofton and Chernoff, 1986). Although there is a lack of toxicity data for many of the compounds found at the Lipari Landfill, the evidence from the metal and the benzene studies provides a reasonable justification for investigating low birthweight.

**b. Cancer**

When the carcinogenicity of the same compounds is investigated, more definitive profiles emerge. Animal and human studies used to develop NJDOH Right To Know program (RTK) fact sheets show that arsenic, benzene and chromium are known human carcinogens while nickel and chloroform are probable human carcinogens. BCEE and 1,2-dichloroethane may be cancer-causing agents in humans because they cause cancer in animals. Toluene may cause mutations in living cells but requires further study to determine if it poses a cancer hazard. Mercury has been tested and has not been shown to cause cancer in animals. Neither xylene nor zinc have been tested for their ability to cause cancer in animals. Although levels of exposure may be very low in settings of environmental contamination, it is still reasonable to examine cancer rates in light of this knowledge.

Taken collectively, the anatomical sites of cancer caused by the known and probable carcinogens are skin, lung, blood (leukemias), throat, nasal cavities and sinuses in humans; and liver, kidney and thyroid in animals. For those compounds considered possible carcinogens the anatomical sites are stomach, lung, breast and liver. Most of these cancer sites were included in this study, but one site of special interest, the liver, could not be evaluated because of its rarity of occurrence.

## C. SUMMARY OF DEMOGRAPHIC CHARACTERISTICS

### 1. Gloucester County

Gloucester County experienced significant population growth for the decades 1950-60, 1960-70 and 1970-80, (47.0%, 28.1% and 15.8% respectively) which were higher than the overall state growth rate for New Jersey of 25.6%, 18.5% and 2.4% for the same time periods. According to the 1970 and 1980 U.S. Census data, the largest proportion of the population increases for the county were attributed to in-migration from other states, (especially from Philadelphia, Pa.) as opposed to in-migration from other New Jersey counties (Table 1 & 2).

The racial composition of the county has remained relatively constant with the white and non-white populations reported as 90.8% and 9.2% for 1960; 91.2% and 8.8% for 1970 and 90.2% and 9.7% for 1980. The median age of Gloucester County residents in 1980 was 29.1 years (the youngest in the state) and the proportion of children under 5 years of age was 7.6% (the second highest of the 21 New Jersey counties). The proportion of the over-65 age group in the county was one of the lowest in the state, 4.9%, with only four of the remaining New Jersey counties having a smaller per cent of senior citizens in 1980. The median family income for the county in 1980 was reported to be \$21,882.00 and the proportion of families below the poverty level was reported as 6.4% (Gloucester County County Planning Department, 1984).

### 2. Demographics of the Four Communities

#### a. Glassboro Borough

Glassboro is a mixed academic/commerical community which is the home of Glassboro State College, an instiution of higher education for the South Jersey

Community. The 1980 population of 14,574 reflects increases for the decades of 1960-70 and 1970-80 of 26.2% and 12.6% respectively. It is a community of 9.37 square miles with a population of 1,555 per square mile, a median age of 23.1 years, a median family income of \$19,767.00 per year, 4.9% of families below the poverty level and a racial composition of 82.3% white and 17.7% non-white as reported in the 1980 U.S. Census (Table 3).

**b. Harrison Township**

Harrison experienced population increases from 1960-70 and 1970-80 of 10.4% and 34.7% respectively. It is a rural, agricultural community of 19.08 square miles and it is rapidly undergoing development. Harrison had a population of 3,544, a median age of 29.8 years, with a population of 188 persons per square mile, and a racial composition of 94.2% white and 5.8% non-white according to the 1980 U.S. Census (Table 3). The median family income was \$19,367.00 a year with 7.8% of the families below the poverty level.

**c. Mantua Township**

Mantua is a mixture of an old farming community and rapidly developing suburbia with many single family homes being built along the major county roads. The total area is 15.96 square miles. The 1970 U.S. Census showed a 20.7% increase in the population for the 1960-70 decade, but the 1980 U.S. Census showed a 4.7% decrease in population to 9,193 persons for the 1970-80 period. The median age was 31.3 years, the population per square mile was 576 people, the median family income was \$22,566.00 per year, the percent of families below the poverty level was 3.7% and the racial composition was characterized as 98.1% white and 1.9% non-white by the 1980 U.S. Census (Table 3).

**d. Pitman Borough**

Pitman is characterized as the oldest and most stable of the four communities studied. It is highly urbanized and developed; the present municipal boundaries have had no changes since the 1920's. The majority of houses are single family homes built in the 1940's and early 1950's with the exception of a housing development near the Lipari Landfill (Timber Streams) which was built in the late 1960's and early 1970's. Pitman has an area of 2.26 square miles. Pitman's population increase for the decade 1960-70 by 18.7%, but decreased by 5.0% to 9,744 persons for the 1970-80 period. The median age was 29.0 years in 1970 and increased to 34.4 years in 1980. The population per square mile was 4,312 persons, the median family income was \$22,051.00 per year, 4.9% of the families were below the poverty level and the racial composition was 98.7% white and 1.2% non-white according to the 1980 U.S. Census (Table 3).

**D. AVAILABLE MEDICAL FACILITIES**

There are three area hospitals located in close proximity to the four municipalities surrounding the Lipari Landfill: Kennedy Memorial Hospital-University Medical Center located in Washington Township; Underwood-Memorial Hospital located in the city of Woodbury; and Elmer Community Hospital in Salem County, N.J. All three hospitals are located within 10-20 miles of the four communities and are 135 bed, 375 bed and 87 bed facilities respectively.

The four communities under study also lie within 20-35 miles of a large city, Philadelphia, Pa., where major teaching/university hospitals are located; i.e, Children's Hospital of Pennsylvania (CHOP), Hospital of the University of

Pennsylvania (HUP), Temple, Hahnemann and Jefferson University Hospitals, etc. The residents of the four communities have used these numerous medical facilities for primary diagnosis, confirmatory diagnosis, delivery of their children and medical treatment.

#### E. BIRTH TRENDS

The increase in the number of births in the 1960's reflects the nation's peak post-World War II "baby boom years", which were followed by a decrease in the number of births during the 1970's, the "baby bust years". The number of births for the state of New Jersey, Gloucester County and the four municipalities studied follow this national pattern (Tables 1,2 and 3) (Gloucester County Planning Department, 1984). A slight increase in the number of births has been recorded nationally, in New Jersey, Gloucester County and the four municipalities for some years of the 1980's and will be documented with the 1990 U.S. Census.

## II. METHODS

The hypotheses underlying the study were: [1] exposure to contaminants in the landfill would be associated with more low birthweight and more cancers and, [2] these effects would be seen most markedly in the immediate vicinity of the landfill, (i.e. Area 1) and decreasing with distance from the site.

### A. CLASSIFICATION OF EXPOSURE

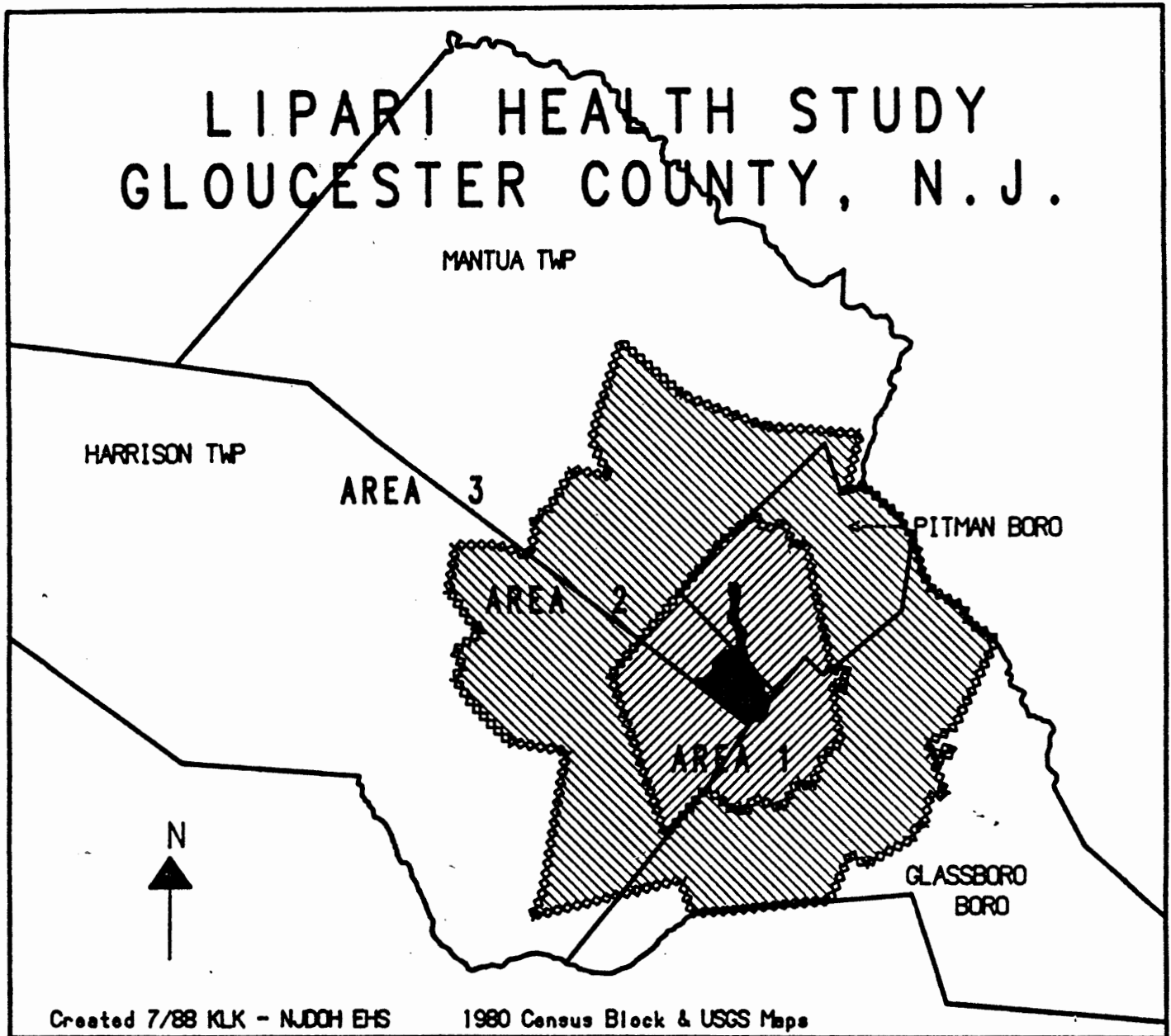
To perform an optimal environmental health evaluation, information on individual exposures is necessary to show specific links between exposure and disease. However, this level of information is rarely available. Therefore, surrogate measures of exposure based on geographical areas are typically substituted for measured personal exposures. Meteorologic information on wind velocity and direction was consistent with expectations that homes closest to the site were likely to have the most affected ambient air. (More recent monitoring has borne out this expectation.) Use of wind-rose modeling was explored, but the resources necessary to utilize such models were not available. Little environmental monitoring data existed prior to 1986 to adequately characterize exposures to residents living in the vicinity of the Lipari site since the dumping began in 1958 until USEPA performed trace atmospheric gas analyzer (TAGA) monitoring in 1985. In order to look at potentially higher and lower exposed populations and since inhalation is the most probable route of exposure for residents in this area, residential distance from the landfill perimeter at the time of diagnosis of the particular health outcome or at time of birth of interest was chosen as the best available surrogate measure for exposure. (Further discussion of this issue appears in the Discussion section, under Dose-Response.)

Four municipalities were chosen to be in the study area: Pitman, Mantua, Harrison, and Glassboro. The Lipari Health Subcommittee discussed exposure classification methods and decided to use a concentric ring approach (Lyons, et al. 1981; NJDOH, 1986), based on the consensus that in the absence of actual exposure data, no other method was any more effective in approximating exposure. The Subcommittee chose 1.0 and 2.5 km distance demarcations to represent potential exposure and to include sufficient population numbers to adequately carry out the health study. Two irregular polygons, which approximate concentric rings with radii of 1.0 and 2.5 kilometers, were extended from the perimeter of the landfill and Alcyon Lake forming two residential exposure sectors (Figure 1 - Map) referred to as Area 1 and Area 2 in the remainder of the report. The area beyond 2.5 km and ending at the municipal boundaries will be referred to as Area 3.

Population estimates were generated by aggregating census blocks into the concentric ring exposure sectors. Census blocks that were intersected by one of the concentric rings were assigned to the exposure sector that contained more than fifty percent of the census block area. Each sector was assigned population denominators based on the 1980 U.S. Census information for the aggregate blocks. Three age groups were available from the 1980 Census block data, less than 18 years, 18 through 64 years and 65 years and older. Cases with the health outcome of interest were summed over the census blocks in the appropriate exposure sectors.



Figure 1. Areas Used as Surrogates for Exposure



1.0 Km



2.5 Km



Lipari Landfill,  
Alcyon Lake and  
marsh areas.



Census block  
boundaries



Municipal  
boundaries

Scale 1:24,000



## B. CANCER INCIDENCE

### 1. Collection of Data through the State Registry

The NJDOH maintains a population-based cancer incidence registry which covers the entire State population. The State Cancer Registry collects reports of all cancer cases diagnosed among New Jersey residents since October 1, 1978. Reports are received from New Jersey hospitals (primarily), physicians, dentists and independent clinical laboratories. In addition, reporting agreements have been established with neighboring states so that New Jersey residents diagnosed with cancer in health care facilities outside of New Jersey can be identified.

The information for each newly diagnosed case available from the Cancer Registry is limited. The basic source document is an abstract of information from the patient medical record. The collected information includes demographic data on each patient and medical data on each cancer. Variables used to analyze the level of cancer in the area include: name, address at time of diagnosis, county and municipality codes, date of diagnosis, primary site, histology type, age at diagnosis, date of birth, race, sex, and accession number. It was decided that the first full year of data collection by the cancer registry, 1979, be eliminated from the study because of the probability of underreporting due to start-up issues.

### 2. Selection of Cases

In order to evaluate cancer incidence in the vicinity of the Lipari Landfill, cancer incidence information for the period of 1980 through 1984 was obtained from the Cancer Registry for the four municipalities. This time period provides about 15 years between the beginning of waste dumping (1966-67, as

estimated by USEPA) and the initiation of shorter latency cancers such as leukemia and lymphoma. Although the landfill had been in existence since 1958, the post-1966 period is likely to be the time of greatest residential exposure to landfill effluents. The bulk of liquid wastes ever dumped at this site, 2.3 million gallons from one chemical company alone, was disposed of between 1967 and 1969 (NJDEP, 1979; USEPA, 1985). At the time that this study was initiated, post-1984 cancer data were not available.

Cases were assigned to an exposure sector after intense efforts to locate addresses on municipal maps and U.S. Census block maps. Addresses which could not be found on maps were given to local health officials for further identification of address location along with assistance from U.S. Postal service staff.

### 3. Data Analysis

Cancer analysis was completed for all cancers combined and for nine site-specific cancers. All races and both sexes were combined in the analysis since U.S. Census information does not separate the population by race or sex for the census blocks used to define the exposure sectors. The types of cancers included in the analysis are: colon, pancreatic, lung, bladder, lymphoma, leukemia, brain and nervous system, rectal, and stomach. These cancer types were selected for review since state age-specific rates were available and published by the Cancer Registry. Primary liver cancer and cancers of the nose, throat, and nasal sinuses were not evaluated because of the unavailability of state rates. Only two cases of liver cancer were found in the entire study area. Analysis of total cancer by municipality and sex was also completed.

Analysis of the cancer incidence for the exposure sectors was completed using Standardized Incidence Ratios (SIRs) (Mausner and Bahn, 1985). The SIR is calculated by dividing the observed number of cases by the expected number. The expected number was mathmatically derived by multiplying the state age-specific cancer rates and the exposure sector populations. The expected number of cases was determined on the assumption that average annual incidence rates for the whole State of New Jersey for 1980-1982 (NJDOH, 1980; NJDOH, 1981-1982) would prevail in the population surveyed. The sector populations were determined from the 1980 U.S. Census block data. In order to protect the confidentiality of the population, only three age groupings were available at the census block level and these were used in the analysis: less than 18 years, 18 through 64 years, and 65 years and over.

If the observed number of cases equals the expected number of cases, the SIR will equal one (1.0). When the SIR is less than one, we conclude that fewer cases were observed than expected. Should the SIR be greater than one, then more cases than expected were observed. Statistical significance was evaluated using a 95 percent confidence interval (CI) (Breslow and Day, 1985). The 95% CI is used to evaluate whether the SIR is different than 1.0 due to chance alone. If the confidence interval includes 1.0, then the SIR is not considered to be significantly different from 1.0, but instead might be different than 1.0 due to random variation of the sample.

To supplement the results obtained by comparison to State rates, direct comparisons were made for some cancer categories between exposure classification groups (Area 1 versus Areas 2 & 3) using Poisson regression, a multivariate statistical technique for observing the effect on an outcome of many factors

simultaneously (Clayton, 1983).

In order to avoid biasing the results by excluding cancer cases that were unlocatable on municipality maps and, therefore, not given an exposure classification, a sensitivity analysis was completed. All cancer and lung cancer SIRs were recalculated for each exposure area using the additional cases of unknown location. The total unlocatable cases were added to each exposure area to give a worst case scenario for the particular exposure category.

## C. LOW BIRTHWEIGHT

### 1. Selection of Livebirths

A subject (livebirth) was included in the study if:

- 1) we could identify from the street address on the birth certificate that the mother lived in Area 1 or in Areas 2 or 3 at the time of birth of the subject (excluded were those with missing addresses, addresses indicating only post office box number or rural delivery (RD) number, incomplete addresses and those with addresses that could not be located in the 1980 U.S. Census Bureau Dual Image Map Encoding (DIME) files, on the U.S. Census block maps or by the local health officer in consultation with the local post office and field investigation);
- 2) the subject was a "singleton" livebirth (no twins, triplets, etc.);
- 3) the birth occurred in one of three 5-year periods: 1961-1965, 1971-1975, 1981-1985 (Table 4);
- 4) information was available on sex and birthweight of the child, and mother's race.

Births that failed to meet the above criteria were excluded from the study. Instate and Pennsylvania (PA) births to New Jersey (NJ) residents were included if the above criteria were met. The NJDOH birth certificate requests the address of the mother at the time of birth without differentiating between mailing address and the actual residential address. During the process of assigning exposure codes to each birth discrepancies between the actual municipal location of the mother's address and the reported address arose. The mail delivery zones were not identical with the municipal boundaries of the four communities. For example, addresses reported as being located in

Glassboro, were actually located in Washington or Elk Townships. To further compound the problem, postal delivery zones, especially rural routes and rural delivery zones, were changed several times during 1961-1985 period. A total of 2032 births could not be used in the birthweight analysis, 1166 because the actual residential location was outside of the four municipalities under study and 866 were not analyzed due to incomplete or missing address information (Tables 4 & 5). If a mother had more than one liveborn child during a particular 5-year period, the subsequent livebirth(s) were included and identified as such in the analysis.

The three five-year periods were chosen to represent periods when exposure to toxic wastes at the site was likely to be non-existent or minimal (1961-65), to be the heaviest (1971-75), and to be reduced because some remediation work was performed (1981-85). Although it would have been preferable to obtain data for the intervening years (1966-70, 1976-80), there was insufficient staff to accomplish this task.

Birthweight distributions usually differ by race as well as by sex. In order to adjust for the effect of race on birthweight, there must be sufficient numbers of white and non-white births. However, in Area 1 there were few non-white births during the study period. Because it was impossible to adjust adequately for race, the study was restricted to white births only.

## 2. Information on Potential Risk Factors

Information on potential risk factors for low birthweight was obtained from the birth certificate. Variables evaluated included sex and gestational age in weeks (stated on certificate for 1961-65 and calculated from date of

last normal menses and date of birth for 1971-75 and 1981-85), mother's race, age and education of the mother, parity, previous stillborns (born dead after 20 weeks gestation for NJ certificates and after 16 weeks for PA certificates), month prenatal care began, total number of prenatal visits, complications of pregnancy, and age and education of the father. These variables were not always available for all time periods and for both states (NJ and PA). Race of the mother was not recorded on NJ birth certificates during 1962-63 resulting in a loss of potential study subjects. No information on prenatal visits or on parental education was included in the NJ birth certificates for 1961-65 or on the PA birth certificates for 1961-65 and 1971-75. Therefore prenatal visits and parental education could not be evaluated for the 1961-65 period. No information on complications of pregnancy was included on NJ birth certificates for 1961-65 so this factor could not be evaluated for that period. APGAR scores and previous miscarriage were not evaluated since they were only available on NJ and PA birth certificates for the period 1981-85.

The exposure variable was the place of residence of the mother at the time of the child's birth as indicated on the birth certificate. Occupational information on the parents was not evaluated since it was not recorded on NJ birth certificates for 1971-1975 or 1981-85. (Only the father's occupation is available for 1961-65 on NJ certificates, and 1961-65 and 1971-75 on PA certificates).

Information on other risk factors for low birthweight was not available on the birth certificate and could not be evaluated. These factors include maternal health, cigarette and alcohol consumption during pregnancy, and parental socioeconomic status. For this reason, we cannot rule out the

possibility that a spurious result might have occurred in our study due to the unrepresentative distribution in exposed subjects of these unmeasured risk factors.

### 3. Data Analysis

First, the distributions, means, and standard deviations of birthweights and major confounding factors were generated and compared for Area 1 versus Areas 2 & 3. For the purpose of birthweight analysis, areas 2 and 3 were combined at all times, using an assumption that those areas did not involve exposure to hazardous substances from the landfill. The birth outcome variables analyzed were: the weight of the liveborn in grams, proportion of low birthweight babies, birthweight distribution and gestational age. We next compared those liveborns who weighed less than 2500 grams ("low birthweight") with liveborns who weighed 2500 grams (about 5½ pounds) or more. (An infant weighing less than 2500 grams (g) at birth is at increased risk of dying within the first four weeks of life ( National Academy of Science, NAS, 1985). Birthweights were also grouped into six categories (< 1500 g, 1500-1999 g, 2000-2499 g, 2500-2999 g, 3000-4699 g, and > 4699 g) to get a better sense of birthweight distribution differences between Area 1 and Areas 2 & 3. Length of gestation in weeks was based on the gestational age field on the birth certificate for 1961-65, and calculated from stated date of last normal menses and date of birth (from the birth certificate) for 1971-75 and 1981-85. We know of no reason that these different ways of analyzing gestational age should bias the results.

Separate analyses were performed for the three 5-year periods. We did not combine the data from the three decades since changes occurred in the birth

certificate during this time. In addition, we lacked information on the many changes that occurred over these decades (e.g., socio-economic, lifestyle; Women Infants and Children (WIC) and other programs, improvements in health care and treatment, etc.).

Mother's residence at the time she gave birth was the "exposure" variable. A "crude" analysis was performed comparing the birth outcomes for the two areas. Then, analyses were performed measuring the effect of the exposure variable on average birthweight and on low birthweight proportion after the effects of other potential risk factors were taken into account. These other factors included the age, parity, education and number of previous stillborns of the mother, age at gestation and sex of the liveborn, complications of pregnancy and prenatal care (NAS, 1973). (Paternal age and education were not included in the analysis since they were highly correlated with maternal age and education and had a weaker association with birthweight than maternal age and education). We used the National Academy of Science's standard for prenatal care, which is a formula for prenatal visit schedule. Not every birth certificate had complete information for gestational age, maternal education, prenatal care, parity, previous stillbirths and complications during pregnancy. Therefore, dichotomous variables were created corresponding to each of these risk factors and were coded with a zero if the subject had complete information on the risk factor and a one if not. Whenever a risk factor variable was included in an analysis, its corresponding variable for missing information was also included (Cohen J & Cohen P; 1983).

Risk factors were coded in the following manner:

Area of residence (exposure):	0 = Areas 2 & 3; 1 = Area 1.
Sex of child:	0 = male; 1 = female.
Gestational age:	weeks.
Maternal age:	
- continuous variable:	years
- dichotomous variables:	"under 19 yrs" and "over 35 yrs", each coded 0 = no, 1 = yes.
Maternal education:	
- continuous variable:	years
- dichotomous variable:	"less than 12 yrs", coded as 0 = no, 1 = yes.
Parity:	0 = not first live-born; 1 = first live-born.
Prenatal care:	0 = greater than or equal to the minimum standard set by the NAS for number of visits and month of pregnancy care began; 1 = below the standard.
Previous stillbirths:	0 = no previous stillbirths; 1 = one or more previous stillbirths.
Complications during pregnancy:	0 = no; 1 = yes.

Descriptive analyses of average maternal age, gestational age, parity, maternal education, and prenatal care are given separately for each time stratum studied. Comparisons of birthweight distribution and proportion of low birthweight between the two areas are presented. Statistical significance was indicated by p-values or confidence intervals (Breslow and Day, 1980).

Multiple regression and analysis of variance (ANOVA) methods (Snedecor and Cochran, 1980) were used to analyze differences in average birthweight between the two areas. Regression diagnostics were performed to identify study subjects who strongly influenced the analysis because they had extreme values for one (or more) of the risk factors and or for birthweight. (The statistic used was Cook's Distance (Cook and Weisberg, 1982)). These subjects were then removed and an additional regression analysis performed to evaluate any changes in the

size of the difference between the two areas in average birthweight. In every situation, removal of subjects with extreme values had little or no impact on the size of the difference. Logistic regression (Breslow and Day, 1980) was used to analyze differences in the proportion of low birthweight babies between the two areas. In all regression analyses, a hierarchical backward elimination method (Greenberg and Kleinbaum, 1985) was used to assess interaction and to eliminate variables. Significance tests were standard t-tests based on the coefficients and their standard errors (Breslow and Day, 1980).

Differences of birthweight distribution and average gestational age between the two areas were evaluated by the chi-square test and the t-test (Snedecor and Cochran, 1980) respectively. All p-values mentioned in the text and tables are two-tailed (equivalent to a one tailed test of  $p < 0.10$ ). We consider  $0.10 > p > 0.05$  to be of "borderline" statistical significance and  $p \leq 0.05$  as statistically significant. Separate analyses were performed on births of greater than 27 weeks gestation and on births with gestational ages between 38 and 42 weeks ("term" births). The 27-week gestational age cutoff was used, as is typically done in epidemiology, to eliminate data for early births with unreliable birth certificate data and thus to protect the validity of the analysis conducted. Analysis of term births provides an indication of whether there are delays in the growth and development of fetuses.

Since a mother may contribute more than one singleton birth during a five year period, we performed additional analyses, as above, using only the earliest child born to the mother during a five-year period.

## D. BIRTH DEFECTS, SPONTANEOUS ABORTION DATA AND SCHOOL ABSENTEEISM

### 1. BIRTH DEFECT DATA

Congenital birth defect data for the years 1961-1965, 1971-1975 and 1981-1984 were available from a birth defects registry maintained by the NJDOH Special Child Health Services Program (SCHSP) and 1985 data were available from the new, population-based NJ Birth Defect Registry, also administered by SCHSP. One hundred and four cases from the four affected townships were identified. The 1985 birth defect data had been coded using the ninth revision of the International Classification of Diseases (ICD) (US Department of Health and Human Services, USDHHS, 1980), birth defect data prior to 1985 were coded using an internal SCHSP system. Of the 49 cases assigned the SCHSP codes, 47 of these case files were successfully transcribed into ICD codes by the SCHSP. Approximately 77% (80/104) of the cases were successfully located on a U.S. Geological Survey (USGS) map with 9.6% (10/104) cases born outside of the four township boundaries, 8.7% (9/104) that could not be located and 4.8% (5/104) born outside of New Jersey.

Although New Jersey has had a legal requirement for reporting birth defects since 1928, the registration system had been part of the State's Crippled Children Program and was not population-based. Legislation signed in 1983 revised the authority of the Department of Health to collect information on children with birth defects. Rules officially adopted on March 4, 1985, require the confidential reporting of all occurrences of birth defects among live births by physicians, dentists, certified nurse mid-wives, clinical laboratories and maternity hospitals in New Jersey.

There was an apparent gross under-reporting of birth defect data prior to 1985. For example, in 1984, 1970 infants with at least one anomaly were reported in contrast to 2628 cases identified in 1985. The improvement in ascertainment occurred in 18 of the 20 major diagnostic categories. In addition, prior to 1985, the existing database was purged of cases which had either died, no longer needed services, or were no longer eligible for services. The files of the SCHSP were reentered into the database for our purposes; however, the percent successfully recovered cannot be estimated although it is probably very low.

Because of the limited database available for the period of interest (from the 1960's to the mid-1980's) and the consequent potential for loss or under-ascertainment of cases occurring before 1985, birth defect data were not analyzed. At the time of the NJDOH decision, the rest of the Lipari Health Subcommittee was apprised of the database restrictions that prevented effective study of birth defects and was informed that the NJDOH would not pursue analysis of these limited data.

## 2. SPONTANEOUS ABORTION DATA

Similarly, an attempt to explore the rates of spontaneous abortion in the affected municipalities did not come to fruition. Early community concerns had focused on this outcome and the NJDOH EHS had agreed to examine available hospital records. Data on spontaneous abortions from hospital medical records were to be collected by the Gloucester County Department of Health with the help of community volunteers and medical expertise from the NJDOH EHS.

Major problems with undertaking a hospital-based study of spontaneous abortions include:

- difficulty in gaining access to hospital records;
- variation in the quality of record keeping over time and among hospitals;
- lack of information on which hospital facilities are utilized for prenatal services or emergencies by residents of the four towns;
- spontaneous abortions are frequently treated in clinics and doctor's offices, with records less accessible and of lesser quality than hospital records; and
- the large potential for under-reporting of spontaneous abortions to any primary health care source.

Medical records were screened from two area hospitals for a maximum of ten years (Kennedy Memorial Hospital, 1971-1975 and 1981-1985; Underwood- Memorial Hospital, 1973-1975 and 1981-1985). A total of 226 spontaneous abortions were identified from these hospitals for the four municipalities in the Lipari study area. Verification of diagnosis was completed on a small subset (n = 11) of the cases by review of the individual hospital records. It was determined that review of all potential cases was not feasible due to the lack of information contained in the medical record on individual risk factors and due to the amount of time required to review each record. Because of these weaknesses and the lack of knowledge of the study population's use of hospitals, if any, for spontaneous abortions, it was determined that it would be impossible to enumerate the actual number of spontaneous abortions that had occurred near Lipari Landfill for any time period. These difficulties were reported to the entire Lipari Health Subcommittee when the decision not to analyze the data was made.

### 3. SCHOOL ABSENTEEISM

At the request of the Lipari Health Subcommittee, the Gloucester County Health Officer selected schools both in and beyond the 1.0 and 2.5 km boundaries of the study areas to perform a comparison of student absenteeism rates for three recent years. Research of the Gloucester County School Superintendent's records indicated a 5-7% yearly absenteeism rate for all schools in and outside of the 1.0 and 2.5 km areas which was similar to the Gloucester County yearly rates of 5-8%. As no unusual absenteeism rates were found, the Health Subcommittee agreed not to pursue this part of the study.

### E. QUALITY ASSURANCE OF DATA

#### 1. Cancer

Quality assurance of cancer incidence information is a routine activity of the Cancer Registry. Annual audits are conducted by Registry staff to confirm the completeness of reporting by all health care facilities. Listings of the cancer cases which have been reported to the Registry are checked against all primary data sources in the hospital to verify that all incident cases have been identified and that data are accurate. The Registry estimates that Statewide underreporting (based on mortality records) of incident cases runs about 7 to 8 percent annually. Regional analysis of underreporting is not available.

EHS took a random sample of 10% of the cancer cases to compare against the actual abstracts from the Cancer Registry to ensure that residential address was transcribed correctly. No errors were found between the Registry abstracts and the data listing which were supplied to the EHS.

## 2. Birthweight

Quality assurance was performed throughout the process of data compilation and data entry. Every third birth record was proofread against the original certificate for the birthweight database and each cancer record was proofread against the original cancer registry printout. In addition, a final quality assurance check was made for the birthweight database after the data were analyzed, as follows. Every 20th birth record (5%) was proofread against the original birth certificate for the 15 years of the Lipari birthweight database. Fifteen variables (when available) were checked for errors: street address, child's name, date of birth, sex, race of mother, age of mother, education of mother, gestational age, plurality, birthweight in pounds and ounces, date of last normal menses, number of prenatal visits, other live births now living, other live births now dead, terminations before 20 weeks and terminations after 20 weeks.

No clerical errors were found in birthweight, date of birth, and street address for any of the three five-year time periods. For 1961-65 the error rates for mother's age, plurality and sex of child were 0.64%, 0.64% and 2.56% respectively. For 1971-75 the error rate for number of prenatal visits was 0.80%. For 1981-85 error rates for age of mother, date of last normal menses and race of mother were 0.83%, 0.83%, and 3.31% respectively, where in the latter case, race was mistakenly recorded as missing data. In all other cases there were no errors detected. The error rate was very low and no concentration of errors was found in any one time period.

The validity of recorded data compared to actual dates, weights and addresses was not evaluated in this study. Other studies have identified rounding of birthweights by hospital clerical staff, and inaccurate reporting of addresses by mothers, but we did not have the means to check these problems in this study. There is no a priori reason to believe that the results were biased by rounding or digit preference.

### III. RESULTS

#### A. CANCER

A total of 633 cancer cases were identified in the four municipalities over the five-year study period, 1980 through 1984. A description of these cases is presented by exposure area in Table 6.

Of the total cases, 580 or 91.6 percent of all cancer cases in the study area were given an exposure area sector code. Only 53 (8.4%) cases were not locatable on municipality maps and, therefore, could not be classified by an exposure area.

Of the 633 total cancer cases diagnosed between 1980 and 1984, 127 (20.1%) lived in Area 1, 219 (34.6%) lived in Area 2, and 234 (37.0%) lived in Area 3 at the time of their diagnosis.

Table 7 presents total cancer incidence by area, sex, and year of diagnosis. Distribution of total cancer incident cases by municipality and sex is presented in Table 8. Standardized Incidence Ratios were calculated for total cancer for each municipality by sex (Table 9). None of the SIRs were elevated for any town, compared to NJ average annual incidence rates for 1981-82, but four SIRs were significantly low.

Standardized Incidence Ratios were calculated by exposure area for all cancers combined and for nine site specific cancers. Certain sites such as liver, nasal and throat cancer could not be analyzed because of absence of state rates. Table 10 presents the results of the SIR analysis by primary cancer type

and area. None of the SIRs were significantly elevated over the State average. Three of the SIRs were significantly lower than the State average. The low SIRs occurred for all cancer types combined for each of the three exposure areas.

The highest SIR, though not statistically significant, was for leukemia in the area closest to the landfill. This SIR was 1.97 (95% CI = 0.72 - 4.29,  $p > 0.05$ ), or six cases observed compared to an expected 3.04 cases, nearly double the expected number.

To supplement the results obtained by comparison to State rates, direct comparisons were made for two cancer categories (all cancer sites and leukemia) between exposure classification groups (Area 1 and Areas 2 & 3) using Poisson regression. In this treatment there was no statistically significant difference found for either cancer category, but the relative risk for all cancer sites was 0.9, very close to 1.0, while the risk for leukemia was still elevated at 2.3, but was not statistically significant (95% CI = 0.82 - 6.55,  $p > 0.05$ ).

The exact residential location of 53 cases (8.4%) attributable to the four municipalities could not be determined from the data abstracted by the Registry. The two main reasons for unlocatable residences were street addresses that did not exist in any of the four municipalities or consisted of a post office box (POB) number only. Because of the uncertainty of where these people actually lived in relation to Lipari Landfill, none of these cases were assigned to an exposure sector. However, to account for the potential bias of excluding the 53 cases of unknown residential location at the time of diagnosis, a sensitivity analysis was completed. SIRs were recalculated for the three exposure sectors for all cancer types combined, lung cancer, and brain/nervous system by adding

in all the unlocatable cases to the known cases for each exposure sector. Table 11 presents the new SIRs. With the addition of the unlocatable cases, all nine SIR's were elevated over 1.0, but only one category was statistically elevated, brain and nervous system cancer (SIR = 2.50; 95% CI = 1.14 - 4.74). The highest SIR was for brain and nervous system cancer in Area 1 (SIR = 2.67; 95% CI = 0.97 - 5.80). Leukemia was not included in the sensitivity analysis since none of the leukemia cases were unlocatable.

## B. LOW BIRTHWEIGHT

### 1. 1961-65

There were 2801 singleton births with birth certificate information on birthweight and mother's residence in Areas 1, 2 or 3 at time of birth (Table 12). Of those birth certificates with information on race of mother, 8% (187) listed race as non-white. Nine non-white births occurred in Area 1 during this period. Although race is an important risk factor for low birthweight, there were too few non-white births in Area 1 to adjust for race in the analysis. As a result, only white births were analyzed. Among white births, 53% (1144/2148) were male.

Some characteristics of the study population births with gestational age greater than 27 weeks (n = 2135) are listed in Table 13. Area 1 mothers were on the average slightly younger than mothers in Areas 2 & 3. Average gestational age was the same for the two groups. Information on education of the mother and prenatal care received was not available from the birth certificate during this period.

Table 13 shows that average birthweight was similar in the two areas.

Birthweight distributions and proportions of low birthweights were compared between the two areas. There was a statistically significant difference in birthweight distribution. However, the difference in proportion of low birthweights was not statistically significant (odds ratio = 0.95). (Similar results were found when all births were included regardless of gestational age; i.e., including those with no information on gestational age.)

Analysis of average birthweight by multiple regression and analysis of variance (ANOVA) was performed on 2135 births in which an interaction term for area of residence and sex of child was included. The difference in average birthweight between the two areas (Area 1 versus Areas 2 & 3) was not statistically significant (Table 14). A logistic regression, including an interaction term for area of residence and sex of child, was used to analyze the difference in the proportions of low birthweights between the two areas. The interaction term was not statistically significant nor was the term for area of residence. This result indicates that there was no statistically significant difference in proportion of low birthweight babies between the two areas. (Similar results were obtained when all births were included regardless of gestational information.)

The results of the analyses of "term" births (38-42 weeks, n = 1980) are presented in Tables 15-16. The mean birthweight for Area 1 was very close to that of Areas 2 & 3 combined. Among males, there is a higher proportion of low birthweights in Area 1 than in Areas 2 & 3. On the other hand, among females there is a lower proportion of low birthweight babies in Area 1 compared to Areas 2 & 3. When other variables (e.g. sex of the child, primiparity and maternal age) were not taken into account, there was no increase in low

birthweight for Area 1 ( $OR = 0.84, p > = 0.10$ ). However, when these other variables were accounted for in the logistic regression analysis, the interaction term was of "borderline" statistical significance ( $0.05 < p < 0.10$ ), indicating that the difference in the proportion of low birthweights between the two areas is not consistent across the sexes.

Finally, we analyzed the earliest child born of each mother during the period, excluding any later births a mother may have had during the period. The only difference from the above results was that the coefficient for the interaction term in the logistic regression for area of residence and sex of the child was cut in half and was not statistically significant.

In summary, during this period there was little difference in mean birthweight between the two areas. Although differences in the proportions of low birthweights existed between the two areas, they were not consistent across the sexes. In Area 1, males had a higher proportion and females had a lower proportion of low birthweights than Areas 2 & 3. When additional (i.e. later) births of each mother were excluded, these differences disappeared. The lack of consistency across sexes, and the fact that these differences disappeared when additional births were excluded, lead us to conclude that these differences are not real ones.

## 2. 1971-75

There were 2151 singleton births with birth certificate information on birthweight and on mother's residence in Areas 1, 2 or 3 at time of birth (Table 17). Of those birth certificates with information on mother's race, 11% (234) listed race as non-white. Thirteen non-white births occurred in Area 1 during

this period. Although race is an important risk factor for low birthweight, there were too few non-white births in Area 1 to adjust for race in the analysis. As a result, only white births were analyzed.

Some characteristics of the study population births with gestational age greater than 27 weeks ( $n = 1815$ ) are listed in Table 18. Area 1 mothers were on average slightly younger and of lower parity than mothers in Areas 2 & 3. Average levels of education achieved, prenatal care sought and gestational age were similar in the two groups. Average birthweight in Area 1 was 80 grams less than in Areas 2 & 3. Birthweight distributions and proportions of low birthweights were compared between the two areas (Table 19). There was a "borderline" statistically significant difference in birthweight distribution between the two areas. This was due to the higher proportion of low birthweights in Area 1 ( $OR = 1.60$ ,  $90\% CI = 1.08, 2.37$ ,  $p < 0.05$ ). (Similar results were obtained when all births were included regardless of gestational age, i.e., including those with no information on gestational age).

Analysis of average birthweight by multiple regression and analysis of variance (ANOVA) was performed in which an interaction term for area of residence and sex of child was included. Table 20 shows the results of this analysis for births over 27 weeks of gestation. The interaction term was not statistically significant ( $p > 0.10$ ), indicating that the difference in average birthweight between the two areas was consistent across the sexes for these births. Among births over 27 weeks of gestation, Area 1 had an average birthweight 66.1 grams lower than Areas 2 & 3 ( $p < 0.05$ ). Logistic regression, including an interaction term for area of residence and sex of child, was used to analyze the difference in the proportions of low birthweights between the two

areas. The interaction term for area of residence and sex of the child was not statistically significant, indicating that the difference in proportion of low birthweights between the two areas was consistent across the sexes. Area 1 had a "borderline" statistically significant higher proportion of low birthweight than Areas 2 & 3 (OR = 1.63, 90% CI = 1.03, 2.57). (Similar results were obtained when all births were included regardless of gestational age; i.e., including those with no information on gestational age).

Births with gestational ages between 38-42 weeks (term) (n = 1420) were analyzed separately. The results are presented in Tables 21-22. Area 1 had a statistically significant lower average birthweight for term births (74 g), than Areas 2 & 3 (Table 22). Area 1 also had a higher rate of low birthweight among term births than Areas 2 & 3, as indicated in the simple results (OR = 1.97, 90% CI = 1.04, 3.75) and in the logistic regression analysis (OR = 2.07, 90% CI = 1.07, 3.99).

Finally, we analyzed the earliest child born of each mother during the period, excluding any later births a mother may have had during the period. The differences in average birthweight were similar (about 70 grams) to those found in the above analyses. However, the differences in birth distribution and proportion of low birthweights were not statistically significant. These results are due to the smaller sample size (n = 1597 for all births after 27 weeks and n = 1248 for term births), since the odds ratios obtained (1.61 for births after 27 weeks and 1.86 for term births) were similar to those obtained in the previous analyses when the additional births were not removed.

In summary, average birthweight for births over 27 weeks of gestation and for term births in Area 1 was statistically significantly lower than in Areas 2 & 3. In addition, Area 1 had higher proportions of low birthweights among births over 27 weeks of gestation and among term births than Areas 2 & 3. When only the earliest birth of each mother was analyzed, the difference in proportion was not statistically significant, but Area 1 continued to have a statistically significant lower average birthweight than Areas 2 & 3.

### 3. 1981-85

There were 1986 singleton births with birth certificate information on birthweight and on mother's residence in Areas 1, 2 or 3 at time of birth (Table 23). Of those birth certificates with information on mother's race, 13% (261) listed race as non-white. Five non-white births occurred in Area 1 during this period. Although race is an important risk factor for low birthweight, there were too few non-white births in Area 1 to adjust for race in the analysis. As a result, only white births were analyzed.

Some characteristics of the study population births with gestational age greater than 27 weeks (n = 1666) are listed in Table 24. Average maternal age, parity, level of education achieved, level of prenatal care sought and gestational age were similar in the two groups. Average birthweight in Area 1 was 24 grams higher than Areas 2 & 3. Birthweight distributions and proportions of low birthweights were compared between the two areas (Table 25). No statistically significant differences were found. (Similar results were obtained when all births were included regardless of gestational age, i.e., including those with no information on gestational age).

Analysis of average birthweight by multiple regression and analysis of variance (ANOVA) was performed in which an interaction term for area of residence and sex of child was included (Table 26). The interaction term was statistically significant indicating that the difference in average birthweight was not consistent across the sexes. In Area 1, males had a lower average birthweight (mean difference = -43 g) while females had a higher average birthweight (mean difference = +89 g) compared to Areas 2 & 3. Logistic regression, including an interaction term for area of residence and sex of the child, was used to analyze the difference in the proportions of low birthweights between the two areas. The interaction term was statistically significant indicating that in Area 1 males have a higher proportion of low birthweights while females have a lower proportion of low birthweights compared to Areas 2 & 3. (Similar results were obtained when all births were included regardless of gestational age; i.e., including those with no information on gestational age).

Births with gestational ages within 38-42 weeks, i.e., term (n = 1306) were analyzed separately. The results are presented in Tables 27-28. Area 1 had an elevated proportion of low birthweight babies, but the excess was not significant in the simple analysis (OR = 1.71,  $p > 0.10$ ) or in the logistic analysis (OR = 1.65, 90% CI = 0.72, 3.75).

Finally, we analyzed the earliest child born of each mother during the period, excluding any later births a mother may have had during the period. The differences in average birthweight were similar to those found in the above analyses. However, no statistically significant difference was found between the two areas in the proportions of low birthweights. The coefficient for the

interaction term, area of residence and sex of the infant, was sharply reduced and was not statistically significant. This indicates that the smaller sample size only partly explains the lack of statistical significance for the interaction term.

In summary, differences between Area 1 and Areas 2 & 3 combined on average birthweight and proportion of low birthweights were not consistent across the sexes. Males in Area 1 had a lower average birthweight and a higher proportion of low birthweights than Areas 2 & 3. The reverse was true for females. The lack of consistency across sexes and the fact that the difference between the two areas in proportions of low birthweights disappeared when additional births were excluded lead us to conclude that these differences are probably not real ones.

#### IV. DISCUSSION AND INTERPRETATION

It is necessary to consider the results in the context of how epidemiologists determine whether statistical relationships between exposures and health outcomes are due to chance or not. Therefore, the results of the study are discussed considering the rationale for the study design, the strengths and weaknesses of the study, and some guidelines for interpreting associations as either representing cause and effect or as not suggesting causal relationships. It is important to remember that many phenomena which are statistically associated with each other do not actually have any cause-effect relationship between them and vice versa.

##### A. DESIGN AND FINDINGS

###### 1. Objective Health Data

One of the greatest strengths of this study is the use of health data (cancer diagnoses and birth weight) which are objective and verifiable by medical observation and records. Such data are assembled routinely for the entire population of New Jersey and were not collected just for this study. Further, by selecting endpoints which are common events with accessible data, we have been able to gain much more statistical power than if such observations were rarely made in a population.

###### 2. Power

An important consideration that must be addressed when testing hypotheses is the power of the statistical test. Power is the relative frequency with

which a true difference of specified size between populations would be detected by the proposed test. In essence, it is the probability that our data will lead us to correctly reject the null hypothesis (the hypothesis of no difference between populations).

Power calculations were done for low birthweight during the design of this study. It was determined that, with a probability (power) of 70% to 80%, a 5-year period for each of the three study decades would be sufficient to detect a 5% difference in the proportion of low birthweights between exposure groups. Power was not calculated prior to initiating the cancer study.

There was sufficient power to detect a 74g difference among the populations with respect to the birthweight analysis. However, there was only an 80% chance that our data would detect a three-fold increase in leukemia incidence in Area 1 (using the conventional criteria for ruling out false positives).

### 3. Significance Tests and the Use of "p-values" to Evaluate Hypotheses

In epidemiology and medicine, potential associations are measured by the statistics used on the data. The actual estimate or measurement of the association (such as mean difference, relative risk or odds-ratio) is useful to gauge the strength of an association (such as association of an exposure with a disease). The significance level, or p-value, provides evidence for or against the hypothesis that chance could produce the observed measure of association. However, the assessment of the possible relationships between exposure and health outcome depends not only on the statistical tests but also on a critical evaluation of the study design, possible biases in the results (in order to look at the real underlying relationships if possible), and any scientific knowledge

about the nature of the relationship between the exposure and the disease. The synthesis of these considerations, are generally more important than how extreme the value of the significance test might be. In this report, we have considered both previous scientific knowledge and statistics.

The significance test is a rule for deciding the strength of the discrepancy between a result found in a study sample and what is predicted by the null hypothesis (Armitage, 1971). In this study, the null hypothesis is that those living near the Lipari site (Area 1) have the same outcome or disease rate as the comparison group (Areas 2 & 3 or the New Jersey state average rates). The p-value is the hypothetical probability that the result obtained in the study, and more extreme results, would occur if the null hypothesis was true. The notion of p-value is hypothetical since it is based on the idea that we could take an unlimited number of samples of the same size and situation (i.e., if we had an unlimited number of situations just like Lipari), we would expect to see results as extreme or more extreme as the one we found a certain percent of the time, given that the null hypothesis is true. This percent is represented by the p-value.

The notion of the confidence interval is also based on the hypothetical situation where unlimited numbers of samples can be taken. However, we interpret the confidence interval differently than the p-value. For example, a 95% confidence interval means that given the degree of variability in the data and the result we obtained, we can construct an interval of values wide enough to have a 95% probability of containing the "true result" we are trying to estimate. If a result has a p-value of less than 0.05, this also means that the 95% confidence interval does not include the value representing no difference (e.g., an SIR of 1.0), and thus provides evidence that the finding was probably

not due to chance alone.

Statistical significance testing and the construction of confidence intervals are useful as part of a decision-making process in which we evaluate the strength of the evidence is strong enough against the null hypothesis. If the strength of the evidence is strong enough against the null hypothesis, we may reject it in favor of an alternative hypothesis that living near the Lipari site is associated with an adverse health outcome. A statistically significant result provides some degree of evidence against the null hypothesis. However, issues such as the magnitude of the difference in disease rate between the exposed and unexposed groups, whether the result may be due to bias or whether the alternative hypothesis is biologically plausible, must also be considered before the null hypothesis is rejected in favor of an alternative hypothesis. It is also important to recognize that a result which is not statistically significant does not provide evidence that the null hypothesis is true, i.e., that the Lipari site has no impact on health. It merely means that the evidence against the null hypothesis is not strong.

#### 4. Confounders

Statistical associations or lack of associations may be due to chance or, very often, may be due either to biases inherent in a study design or to factors not accounted for which can themselves influence both exposure observations and effect observations but were not included in the study. A good textbook example of this last point is the suspected connection between excessive alcohol consumption and oral cancer. Smoking is also related to this disease, but it is known that there are more smokers in the drinking population than in the non-drinking population, and that smoking by itself increases the incidence of

oral cancer (Rothman, 1986). Thus, smoking will distort the estimate of the effect of drinking on oral cancer unless it is accounted for. This distortion is called a confounding effect.

## 5. Cancer

Cancer incidence in the population living in the area surrounding the Lipari Landfill was evaluated relative to average state incidence rates. On a more regional basis, cancer incidence for the four municipalities combined is statistically below the expected numbers. Leukemia was elevated near the landfill (without statistical significance) both when comparing it to statewide rates and when comparing the sector closest to the landfill, Area 1, with the other sectors, Areas 2 & 3.

It is difficult to interpret the cancer findings. Underreporting may play a role in the low observed numbers found in the study area for most of the cancers. Although New Jersey has an agreement with Pennsylvania to recover information on New Jerseyans diagnosed in the Philadelphia area, the study has no way of gauging the completeness of out-of-state reporting. The impact that underreporting might have on each exposure sector is unknown. Furthermore, average NJ incidence rates used to calculate expected numbers tend to be higher than national cancer incidence rates (Surveillance Epidemiology End Results, SEER, 1984). Use of the state rates could therefore overestimate the number of expected cases for the study area. (This problem has been partly reduced in the case of leukemia and other cancers by modeling incidence rates (of area 1 vs area 2 & 3) rather than standardized rates.

Another concern with the cancer data is a possible inadequate length of

time within the study design for the latency of most cancers other than leukemia. Latency is the delay between exposure to a disease-causing agent and the diagnosis of the disease. In the case of carcinogenesis, the latency period may be as short as a few years or may be several decades.

Another potential problem with the cancer data is that the cases were given a sector code based on residence at the time of diagnosis. The study design does not permit evaluation of the length of time lived at that location prior to cancer onset, even though, due to cancer latency periods, such data would have been preferable. In-migration of cancer cases could give a false picture of the level of cancer in an area. On the other hand, out-migration or loss of those cases who moved away from the study area just prior to cancer diagnosis could reduce the ability of the study to detect a difference if one exists. It is assumed that in- and out-migration balance each other in terms of overall cancer incidence, but there may be an effect in either direction at the level of specific cancer types.

Information on other risk factors such as occupational exposures or personal life style habits are unknown for the study population. The potential risk factors that cannot be accounted for in the study design may vary significantly from one exposure sector to another or for the state as a whole.

#### **6. Low Birthweight**

Low birthweight is a significant determinant of infant mortality and morbidity (NAS, 1973). After taking into account information on risk factors available on the birth certificate, a lower average birthweight and a higher proportion of low birthweight were found in Area 1 compared to Areas 2 & 3

during the period 1971-75, when Area 1 residents were most likely to be exposed to toxic waste from the site. Conflicting results were obtained for each sex for the period 1981-85 which we could interpret only speculatively.

The findings for the period 1981-85 are difficult to interpret. Statistically significant differences between the two areas were found for average birthweight and for the proportion of low birthweights. However, exactly opposite trends occurred between the sexes. This is an unexpected and puzzling result. It is unclear how the sexes could react so differently to the same exposure. We believe that the findings are due to chance, and therefore, that there is no evidence of an association between the site and birthweight during the period 1981-85.

It must be remembered in the interpretation of this study's results that information on other risk factors for low birthweight was not available on the birth certificate and could not be taken into account. These factors include maternal health, occupation, cigarette and alcohol consumption during pregnancy, and parental socioeconomic status. For this reason, we cannot rule out the possibility that a spurious result might have occurred in our study due to the impact of one (or more) of these unmeasured risk factors.

#### **B. EXPOSURE MISCLASSIFICATION**

The most serious weakness of this study is probable exposure misclassification. The critical piece of information required to assure a meaningful evaluation of this data is actual personal exposure to chemicals

emanating from the landfill over time; that is, who was exposed and who was not exposed and what was the magnitude of the exposures that did occur. Since personal exposure information does not exist, residential distance from the landfill was used as a surrogate measure for potential past exposure. This was accomplished by aggregating the population into exposure sectors using concentric rings of 1.0 and 2.5 km from the landfill perimeter. Although this method may have been the best way to estimate past potential exposures at the time the study was designed, it is also likely that some unexposed residents were classified into the exposed area (1.0 km sector).

In the low birthweight and the cancer (leukemia) studies, Area 1 was considered the "exposed" area while Areas 2 & 3 were grouped together and considered the "unexposed area". This is an extremely crude categorization of exposure and many (if not most) of Area 1 residents were probably not exposed to hazards from the site. On the other hand, some residents in Areas 2 & 3 might have been exposed if they ventured onto the site for recreational purposes or into neighboring parks where metals and pesticides were detected. This "misclassification" of exposure would tend to reduce any real disease-exposure association (i.e., bias the study towards finding no effects when one truly exists). So, a finding of no effect (or no disease-Area 1 association) cannot be interpreted as meaning that there is no health effect. A finding of no association merely means that the study is inconclusive, given the almost certain exposure misclassification.

This potential random error in determining who is exposed and unexposed cannot be rectified due to the lack of data on air, soil and water contamination during the 1970's and early 1980's. Since then, data have become available on

off-site contamination, but these data were obtained after some remedial work was performed and do not reflect earlier contamination levels. In addition, these data are inadequate for exposure assessment purposes because of the problems inherent in air sampling and because there is no information on the residents' activities that may bring them in contact with contamination from the site.

In summary, if there was any effect of the Lipari Landfill on disease endpoints or health problems, this potential for exposure misclassification would tend to underestimate these effects.

#### C. OUTCOME MISCLASSIFICATION

It is also possible that erroneous data involving the health endpoints analyzed in this study could lead to an error in the study results. The most likely error here is missing cancer incidence data, which would tend to reduce the incidence rates for the area studied. Therefore, underreporting may play a role in the low observed numbers found in the study area. The largest potential source for this error is missing data for cancer cases diagnosed in Philadelphia but who were not reported to the NJ Cancer Registry. Although New Jersey has an agreement with Pennsylvania to recover information on New Jerseyans diagnosed with cancer in the Philadelphia area, this study has no way of gauging the quality of out-of-state compliance with reporting requirements. However, since there is no reason to expect underreporting to be more frequent in one exposure sector of the study than the other, direct comparison of the sectors should control for this problem.

Average NJ incidence rates tend to be higher than national cancer incidence rates (SEER, 1984). Use of the state rates could therefore overestimate the number of expected cases for the study area, thus decreasing the ratio of observed to expected cancers and possibly obscuring any association between the landfill and excess cancer. However, as was noted above, this problem has been addressed in the case of leukemia by comparing the incidence of the assigned exposure sectors to calculate relative risks.

Because of the long latency period in cancer etiology, in- and out-migration from the study area could lead to serious limitations in cancer studies of this type. The cases were given a sector code (Area 1, 2 or 3), based on their stated residence at the time of the diagnosis. The study design does not permit evaluation of the length of time lived at this location prior to the onset of cancer. In-migration of cancer cases could give a false picture of the level of cancer in an area. On the other hand, out-migration or loss of those cases who moved away from the study area just prior to the cancer diagnosis could reduce the ability of the study to detect a difference if one truly exists. It is hoped that in- and out-migration balance each other in terms of overall cancer incidence, but there may be an effect in either direction at the level of specific cancer type.

#### D. APPROACHES TO EVALUATING ENVIRONMENTAL EPIDEMIOLOGY STUDIES

##### 1. Strength of Association Between Health Outcomes and Exposure Sectors

In interpreting epidemiological data, a greater difference in rates of disease or dimension of biological characteristics between high exposure and low exposure groups, is interpreted as indicating a higher likelihood that an observed association is due to a causal relationship rather than to a non-causal one. For example, relative risks greater than 2.0 are often interpreted as indicating a strong relationship, and above 5.0, one that is extremely strong.

For overall cancer data in this study the association was weak in that the observed-to-expected ratios were decreased. For leukemia incidence closest to the landfill, the observed-to-expected ratio was almost 2.0, but the limited number of cases of this cancer type resulted in a lack of statistical significance for that ratio.

For the birthweight data, the mean difference in 1971-75 birthweights between Area 1 and Areas 2 & 3 combined was 74 grams (2.5 ounces). The proportion of births below 2500 g (classified as low birth weight) was statistically significant for the high exposure sector, Area 1. (See Table 29 for a clinical perspective on the 74 g difference.) The difference in average birthweight found in the period 1971-75 is smaller than the range of birthweight reduction found in studies of cigarette smoking during pregnancy (150 grams to 250 grams) but similar to findings from studies of moderate alcohol consumption (Kline, 1987) and caffeine consumption (Martin, 1987). Marijuana use 2-3 times per week during pregnancy has been found to reduce average birthweight by 127 grams (Kline, 1987) (Table 29). Under conditions of mild-to-moderate malnutri-

tion and depending on initial nutritional status, increasing the caloric intake in the maternal diet during pregnancy can raise birthweight by 40 to 80 grams (Susser, 1984). In a study of a community potentially exposed to arsenic from a nearby copper smelter in northern Sweden, investigators found a statistically significant decline in average birthweight of 68 grams (Nordstrom, 1978) (Table 29). From a medical standpoint 74 g can be important in the future health status for newborns of low birthweight (less than 2500 g at birth), but 74 g is not generally considered medically significant for newborns who are of normal birthweight, i.e. over 2500 g.

## 2. Consistency With Other Findings

When an association in a specific study is found between exposures and effects, inference that the association may be causal tend to be supported if consistent or similar findings have been previously reported in other populations.

### a. Cancer

Recent studies in Woburn, Massachusetts examined childhood leukemia incidence with respect to known well water contaminations and found evidence that some excess cases might be related to exposure (Lagakos, 1986). The investigators had precise information about temporal and geographic variation that was not obtainable for the Lipari study. In general, the Woburn leukemia results were consistent with the Lipari results, but it must be remembered that the leukemia findings in Lipari were not statistically significant and included many adult onset leukemias which may not be comparable to the childhood disease. An accompanying critique of the Woburn study pointed out that there have been two recent negative studies of adult leukemia where contaminated drinking water

was examined (Whittemore, 1986). In Love Canal, the leukemia rates of females were elevated, but not significantly, in the census tract including the canal area. Because of the many differences in exposure settings and study designs, it is difficult to compare the consistency of the leukemia results in Lipari with other related study results.

#### **b. Low Birthweight**

There have been several other occasions in which proximity to a problem source has been statistically associated with a tendency to decreased birth weight. In a study of a community potentially exposed to arsenic from a nearby copper smelter in northern Sweden, investigators found a statistically significant decline in average birthweight of 68 grams (Nordstrom, 1978). At Love Canal, an elevated rate of low birthweight among residents potentially exposed was also found. However, investigations of communities living near a Lowell, Massachusetts, toxic waste site (Ozonoff, 1983) and a toxic waste landfill in Ontario, Canada (Hertzman, 1987) did not find declines in average birthweight or an elevated prevalence of low birthweight infants. The latter two studies had far fewer numbers (on the order of 300 exposed and 300 controls) than did the Lipari studies, and thus had less power to detect small significant differences than did the present study.

### **3. Temporality, or Order of Occurrence, of Exposure and Effect**

The study design used here focuses on two health indices, one for which data was available only after exposure had occurred (cancer incidence) and the other (birthweight) which could be analyzed for time periods before, soon after, and long after the exposures were believed to be most intense.

**a. Cancer**

For cancer, the latency period, the lag between exposure and clinical symptoms, is a serious issue in interpreting the findings. A valid study must incorporate sufficient time between potential exposure and effect. The latency period for cancer may be as short as a few years or as long as decades.

The Lipari Landfill began operation in 1958 with significantly increased activity and probable human exposure occurring after 1966. The study period for cancer, 1980 through 1984, permitted a more adequate time interval for cancers with shorter latencies, such as leukemia and lymphoma, than for other cancers. No more than 18 years (1967-1984) exist since the earliest likely exposure to the end of the study. Therefore, longer latency cancers, such as lung cancer, would not have been diagnosed before the end of the study period.

Leukemia, a generally shorter latency cancer (17 to 20 years), was elevated, though not statistically significant, for the 1.0 km sector. A total of six cases were observed compared to an expected 3.04 for the "higher exposed" sector. The difference in the observed and expected numbers could be an indication that a landfill effect occurred. Because of the long latency period of many cancer types, there may not have been sufficient elapsed time to rule out other possible cancer effects. Further, risk factors such as occupational or other exposures unrelated to the landfill could not be examined.

**b. Low Birthweight**

Environmental influences during gestation have the potential to affect birthweight; in contrast to cancer, effects may potentially be seen within a nine-month period after exposure begins. Women giving birth during 1961-65

probably had little or no exposures to hazardous substances from the site. Those giving birth during 1971-75 may have been exposed during their pregnancies. Those giving birth from 1981-85 were probably not highly exposed during their pregnancies, although they may have come into contact with toxic substances from the site at an earlier time.

#### 4. Biological Coherence, Plausability, and Analogy

When there is a logical basis for relating an exposure and hypothetical effect (based on biological knowledge and other analogous findings), there is more support for such an association to be interpreted as representing causation rather than confounding or coincidence. As discussed earlier, one reason that these two health indices were selected was prior suggestions in human and animal research that some specific chemicals which were found at Lipari could have caused cancer, especially leukemia, or could have decreased birthweight if residents came into contact with these substances. Further, the mechanisms by which some chemicals can induce cancer have been intensely researched, and it is clear that many substances found at Lipari are potential carcinogens or could have deleterious effects on gestational development. For example, benzene, a known leukemogen, was found in air, soil and leachate samples. Since numerous contaminants were detected in off-site soil and air samples, it is impossible to implicate any one contaminant as a likely cause of the elevated prevalence of low birthweight infants in Area 1. Moreover, any exposure that occurred was probably to a mixture of contaminants. Little is known about the effects of exposures to chemical mixtures. Nevertheless, some of the volatiles and metals found off-site, such as lead, benzene and xylene, have been reported to cause low birthweight in animals and/or humans.

## 5. Dose-Response Issues

Whenever an exposure-effect relationship increases in strength with increasing dose, causality is supported. It is not possible in this study to examine whether higher degrees of exposure to the mixture of chemicals from the Lipari Landfill were associated with greater likelihood of cancer or with lower birthweight. To do so would have required the ability to classify residents with great confidence into at least three exposure groups, and as previously discussed, exposure classification is the weakest aspect of this study. The issue of dose-response or gradient of exposure can not be effectively addressed in this study.

During the period 1961-65, when exposure from the site was probably minimal if at all, there were no remarkable differences between the two areas on average birthweight, and the differences between the two areas on the proportion of low birthweights were not consistent across the sexes. However, after taking into account information on risk factors available on the birth certificate, Area 1 had a statistically significant higher proportion of low birthweights compared to Areas 2 & 3 in the period 1971-75.

Since it is believed that the heaviest exposures probably occurred during this period and that at least some residents in Area 1 were exposed (particularly air exposure), our findings appear to be consistent with the hypothesis that such exposures had a negative impact on birthweight in Area 1 from 1971-75. For the years 1981-85, the opposite trends for the two sexes may be due to chance and suggests no association of birthweight and Area 1 versus Areas 2 & 3.

In summation, the occurrence of low birthweight trends immediately following the highest potential exposures suggests, but inconclusively, a causal relationship between lower birthweight and residence close to the Lipari Landfill.

## V. CONCLUSIONS AND RECOMMENDATIONS

A study of birthweight in periods of the 1960's, 1970's and 1980's, and the occurrence of cancer cases from 1980-1984 in the vicinity of the Lipari Landfill was performed because of local concerns about possible effects on the communities due to the presence of the landfill. Because of a lack of historically accurate exposure information (see Appendix) a crude approach to categorizing areas of potentially greater exposure was used. This consisted of designating census tracts within 1.0 km of the perimeter of the site as areas of greater exposure than areas within 2.5 km of the site. Additionally, some of the study population resided in areas of the four towns outside of 2.5 km.

In the area within 1.0 km of the site, there was a lower average birthweight and greater proportion of low birthweight babies (regardless of sex) born in the period 1971-1975 than in areas more than 1.0 km from the site. This was not the case in the period 1961-1965, when chemical exposures from the site were probably non-existent. In the 1981-1985 period there was still a reduction in birthweight among male babies within 1.0 km, but an unexplainable higher birthweight among females.

In view of all the considerations in the Discussion section, we believe that the findings of the study are consistent with the hypothesis that exposure from the Lipari site in the early 1970's had an impact on birthweight. Because of the impossibility of determining actual exposures during this past period, further study of this possible effect cannot be made. Whether such an impact continued into the 1980's cannot be answered since the data appear to offer conflicting trends between the sexes.

Cancer incidence overall, by standard analysis of adjusted rates, was statistically low in the four towns. Within the 1.0 km area, there was no statistical elevation in total cancer or in any of nine specific cancers analyzed. In fact, total cancer incidence was statistically low. However, there was an elevation in leukemia in the area within 1.0 km (six cases versus three expected), but this elevation was within the range of chance variation because of the small number of cases involved. We cannot draw from these data any inference about a relationship between formerly living near the landfill and increased risk of leukemia.

It should be noted, however, that most of the limitations of the study would tend to obscure rather than exaggerate an association between health outcomes and exposure to chemicals from the landfill.

#### RECOMMENDATIONS

The NJDOH continues to urge prompt remediation of the landfill, as it did even before this study commenced. The presence of hazardous substances to which residents are potentially exposed is, in itself, sufficient rationale for cleanup, and is bolstered by the suggestive results of this study. The cleanup should in no way be delayed due to lack of a conclusive link of adverse health effects to the landfill in this study.

The EHS will follow up shortly on some of the additional analyses and data presentations suggested by the Peer Review Panel (See Addendum). In addition, continuing involvement by the NJDOH with this community is recommended and planned, including both services and further surveillance.

The precise form of those activities will be developed through continued participation by the Department and the Health Subcommittee and other interactions with the community. The Department will designate specific medical staff to counsel residents who have questions about birthweight, cancer or other health issues related to this study. We would like an opportunity to discuss with the community what, if any, other feasible services would be most useful. At present, no special clinical diagnostic or treatment procedures, other than general good preventive health practices, are appropriate for area residents.

Continued surveillance of birthweight and cancer are recommended for this community. The NJDOH would like to develop, in cooperation with the Subcommittee and the community, the specific forms such continued surveillance will take, recognizing that limitations which affected the current study (such as availability of exposure and health data, availability of personnel and other resources, and latency periods for cancer) would need to be addressed for future efforts. It is important that any further data collection include, so far as possible, information on other risk factors such as smoking, alcohol consumption, and occupation.

Lastly, the NJDOH appreciates the deep concern which members of this community express about their health and environment, and encourages these and all New Jersey citizens to continue to learn about preventive environmental and health practices of all types.



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**TABLES**



TABLE 1  
 SUMMARY OF POPULATION CHARACTERISTICS\*  
 NEW JERSEY

US CENSUS YEAR	1950	1960	1970	1980
TOTAL POPULATION	4,835,000	6,070,780	7,192,805	7,364,823
PERCENT INCREASE	--	25.6	18.5	2.4
PERCENT WHITE POPULATION	--	91.3	88.6	83.1
MEDIAN FAMILY INCOME	--	\$6,786	\$11,403	\$19,621
MEDIAN AGE (YEARS)	32.9	32.4	30.1	32.2
RESIDENT BIRTHS	97,743	132,594	120,168	96,438
BIRTH RATE (PER 1,000 POPULATION)	20.2	21.8	16.7	13.1
PERCENT CHANGE	--	+35.7	-9.4	-19.7

\* From 1960, 1970 and 1980 U.S. Census Bureau and State of New Jersey Department of Labor, June 1984.

TABLE 2  
 SUMMARY OF POPULATION CHARACTERISTICS\*  
 GLOUCESTER COUNTY, N.J.

US CENSUS YEAR	1950	1960	1970	1980
TOTAL POPULATION	91,727	134,840	172,681	199,917
PERCENT INCREASE	--	47.0	28.1	15.8
WHITE POPULATION	--	122,391	157,542	180,281
PERCENT OF TOTAL	--	90.8	91.2	90.2
NON-WHITE POPULATION	--	12,449	15,139	19,343
PERCENT OF TOTAL	--	9.2	8.8	9.7
MEDIAN AGE (YEARS)	30.8	29.0	27.1	29.1
MEDIAN FAMILY INCOME		\$6,341	\$10,620	\$21,882
RESIDENT BIRTHS	--	3,315	3,111	2,994
BIRTH RATE (PER 1,000 POPULATION)	--	24.4	18.0	15.0

\* From 1960, 1970 and 1980 U.S. Census Bureau and State of New Jersey Department of Labor, June 1984.

TABLE 3  
\*  
SUMMARY OF POPULATION CHARACTERISTICS  
THE FOUR MUNICIPALITIES STUDIED

	GLASSBORO BOROUGH	HARRISON TOWNSHIP	MANTUA TOWNSHIP	PITMAN BOROUGH
TOTAL POPULATION				
1960	10,253	2,410	7,991	8,644
1970	12,938	2,661	9,643	10,257
1980	14,574	3,544	9,193	9,744
PERCENT CHANGE				
1960-70	+26.2	+10.4	+20.7	+18.7
1970-80	+12.6	+34.7	-4.7	-5.0
PERCENT WHITE POPULATION				
1960	83.2	88.9	99.1	99.3
1970	85.4	91.8	99.3	99.2
1980	82.3	94.2	98.1	98.7
PERCENT NON-WHITE POPULATION				
1960	17.7	11.1	0.9	0.7
1970	14.3	8.2	0.8	0.8
1980	16.1	5.8	1.2	1.3
MEDIAN AGE (YEARS)				
1970	23.8	29.7	25.2	29.0
1980	23.1	29.8	31.3	34.4
MEDIAN FAMILY INCOME				
1970	\$10,950	\$9,984	\$10,669	\$11,448
1980	\$19,767	\$19,367	\$22,566	\$22,051
PERCENT CHANGE MEDIAN FAMILY INCOME				
1960-70	+3.7	+28.4	+27.7	+10.8
1970-80	-6.5	+7.6	+9.6	-0.2
RESIDENT BIRTHS				
1960	261	70	253	175
1970	256	59	188	182
1980	214	74	219	147

\* From 1960, 1970 and 1980 U.S. Census Bureau; Gloucester County Office of Education, November, 1976; Gloucester County Planning Department, November, 1976.

TABLE 4

RESIDENT BIRTHS BY FIVE YEAR TIME PERIOD AND MUNICIPALITY  
 BIRTHWEIGHT DATABASE \*

TIME PERIOD	GLASSBORO BOROUGH	HARRISON TOWNSHIP	MANTUA TOWNSHIP	PITMAN BOROUGH	SUBTOTAL
1961-65	1233	318	1119	921	3591
1971-75	1051	255	789	717	2812
1981-85	969	287	998	605	2859
SUBTOTAL	<u>3253</u>	<u>860</u>	<u>2906</u>	<u>2243</u>	<u>9262</u>
UNABLE TO LOCATE 12 BIRTH CERTIFICATES: REMOVED FROM DATABASE					-12
TOTAL FOR THE 15 YEAR TIME PERIOD					9250

\* Data from NJDOH Vital Statistics and Registration and NJDOH Center for Health Statistics.

TABLE 5

NUMBER OF BIRTH CERTIFICATES BY REASON-NOT-ANALYZED  
BIRTHWEIGHT DATABASE

	GLASSBORO	HARRISON	MANTUA	PITMAN	SUBTOTAL
1961-65					
* INCOMPLETE ADDRESSES	87	87	153	21	348
OUTSIDE 4 TWP**	109	76	148	1	334
1971-75					
* INCOMPLETE ADDRESSES	50	78	93	11	232
OUTSIDE 4 TWP**	71	71	159	4	305
1981-85					
* INCOMPLETE ADDRESSES	49	93	136	8	286
OUTSIDE 4 TWP**	83	39	403	2	527
SUBTOTAL	449	444	1092	47	2032
DUPLICATE BIRTH RECORDS, SEALED RECORDS AND TWINS, TRIPLETS ETC. REMOVED FOR 15 YEAR PERIOD					203
MISSING DATA FIELDS ON BIRTH CERTIFICATES (All variables and includes 49 resident out of state births not retrieved.)					585
BIRTH RECORDS OF NON-WHITE BIRTHS					680
TOTAL OF BIRTH RECORDS UNABLE TO INCLUDE IN ANALYSIS					<u>3500</u>
TOTAL NUMBER OF BIRTH RECORDS ANALYZED (WHITES ONLY)					5750

\* Incomplete or unlocatable addresses: lacking residence information or post office box or rural delivery route reported on birth certificate as address of mother.

\*\* Actual residential address located outside of the four townships. Mail delivery address is not identical with actual residential address.

TABLE 6  
 CANCER INCIDENCE BY TYPE AND AREA  
 (1980 - 1984)  
 LIPARI LANDFILL

Cancer Type	All Areas	AREA			UNKNOWN LOCATIONS
		1	2	3	
Buccal cavity	11	2	5	2	2
Esophagus	7	1	3	2	1
Stomach	11	2	2	7	0
Colon	81	19	25	34	3
Rectal	31	5	13	12	1
Pancreas	11	2	5	2	2
Liver	2	0	2	0	0
Gallbladder	4	1	1	1	1
Other digestive	1	0	1	0	0
Larynx	11	2	4	4	1
Lung and pleura	94	15	28	38	13
Other respiratory	2	1	0	1	0
Bones and joints	2	0	0	1	1
Soft tissue	4	0	4	0	0
Skin *	56	9	18	23	6
Breast	76	16	22	30	8
Cervix uteri	28	4	8	13	3
Corpus uteri	24	4	9	11	0
Ovary	20	2	7	11	0
Other female genital	2	0	1	0	1
Prostate	41	6	19	12	4
Other male genital	5	1	3	1	0
Bladder	29	9	10	8	2
Kidney	10	2	5	3	0
Brain/central nervous system	14	2	5	3	4
Endocrine system	5	2	2	1	0
Hodgkin's disease	5	3	2	0	0
Non-Hodgkin's lymphoma	9	1	4	4	0
Multiple myeloma	1	1	0	0	0
Leukemia	15	6	4	5	0
Miscellaneous reticuloendothelial	4	1	2	1	0
Unknown primary	17	8	5	4	0
Totals	633	127	219	234	53
Percent of Total		20.06%	34.60%	36.97%	8.37%

\* Skin cancer does not include Basal cell. Basal cell skin cancer was not reportable to the Registry during the study period.

TABLE 7

TOTAL CANCER INCIDENCE  
 BY YEAR, SEX, AND AREA  
 (1980 - 1984)  
 LIPARI LANDFILL

Year and Sex	ALL AREAS	AREA			UNKNOWN AREAS
		1	2	3	
1980					
Male:	69	14	21	31	3
Female:	63	16	21	22	4
1981					
Male:	54	12	19	15	8
Female:	68	16	25	22	5
1982					
Male:	42	7	19	13	3
Female:	82	15	26	36	5
1983					
Male:	75	9	33	26	7
Female:	68	15	26	19	8
1984					
Male:	60	13	19	24	4
Female:	52	10	10	26	6
All Years					
Male:	300	55	111	109	25
Female:	333	72	108	125	28

TABLE 8

THE NUMBER OF ALL INCIDENT CANCER CASES  
BY MUNICIPALITY AND SEX  
(1980 - 1984)  
LIPARI LANDFILL

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Municipality	Male	Female	Total
Glassboro	94	94	188
Harrison	22	19	41
Mantua	60	76	136
Pitman	99	116	215

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TABLE 9

STANDARDIZED INCIDENCE RATIOS (SIR) FOR TOTAL CANCER INCIDENCE  
 BY MUNICIPALITY AND SEX  
 (1980 - 1984)  
 LIPARI LANDFILL

Municipality	Expected	Observed	SIR	95% Confidence Interval (CI)
Glassboro				
Male:	112.817	94	0.833	0.673 - 1.020
Female:	120.755	94	0.778 *	0.629 - 0.953
Harrison				
Male:	37.294	22	0.590 *	0.370 - 0.893
Female:	33.582	19	0.566 *	0.340 - 0.884
Mantua				
Male:	83.204	60	0.721 *	0.550 - 0.928
Female:	79.534	76	0.956	0.753 - 1.196
Pitman				
Male:	100.413	99	0.986	0.801 - 1.200
Female:	124.805	116	0.929	0.768 - 1.115

\* Statistically low,  $p < 0.05$ .

\*\* The NJ Cancer Incidence Rates for 1981-82 are the basis for these rates. The municipal-specific rates were determined directly from the NJ Cancer Registry and 1982 census estimates prepared from 1980 U.S. Census data by the NJDOH, Center for Health Statistics.

TABLE 10  
 STANDARDIZED INCIDENCE RATIOS (SIR)  
 BY PRIMARY CANCER TYPE AND AREA  
 (1980 - 1984)  
 LIPARI LANDFILL

Primary Type	Area	Expected	Observed	SIR	95% Confidence Interval (CI)
ALL TYPES:	1	168.700	127	0.753 *	0.628 - 0.896
	2	262.486	219	0.834 *	0.727 - 0.952
	3	278.769	234	0.839 *	0.735 - 0.954
COLON:	1	18.181	19	1.045	0.629 - 1.632
	2	27.174	25	0.920	0.595 - 1.358
	3	29.841	34	1.139	0.789 - 1.592
PANGREAS:	1	3.797	2	0.527	0.059 - 1.902
	2	5.759	5	0.868	0.280 - 2.026
	3	6.222	2	0.321	0.036 - 1.161
LUNG/PLEURA:	1	24.640	15	0.609	0.341 - 1.004
	2	38.454	28	0.728	0.484 - 1.052
	3	40.300	38	0.943	0.667 - 1.294
BLADDER:	1	8.890	9	1.012	0.463 - 1.922
	2	13.474	10	0.742	0.355 - 1.365
	3	14.589	8	0.548	0.236 - 1.081
LYMPHOMA:	1	5.695	4	0.702	0.189 - 1.798
	2	9.057	6	0.662	0.242 - 1.442
	3	9.882	4	0.405	0.109 - 1.036
LEUKEMIA:	1	3.044	6	1.971	0.720 - 4.291
	2	4.676	4	0.856	0.230 - 2.190
	3	5.619	5	0.890	0.287 - 2.077
BRAIN/CNS:	1	2.251	2	0.888	0.100 - 3.207
	2	3.607	5	1.386	0.447 - 3.235
	3	4.160	3	0.721	0.145 - 2.107
RECTAL:	1	7.815	5	0.640	0.206 - 1.493
	2	11.860	13	1.096	0.583 - 1.875
	3	12.807	12	0.937	0.484 - 1.637
STOMACH:	1	4.211	2	0.475	0.053 - 1.715
	2	6.355	2	0.315	0.035 - 1.136
	3	6.909	7	1.013	0.406 - 2.088

\* Statistically low,  $p < 0.05$ .

TABLE 11

SENSITIVITY ANALYSIS OF THE STANDARDIZED INCIDENCE RATIOS (SIR)  
 FOR ALL CANCER TYPES AND FOR LUNG CANCER  
 (INCLUDING CASES OF UNKNOWN LOCATION)  
 (1980 - 1984)  
 LIPARI LANDFILL

Area	Cancer Type	Expected	Observed	SIR	95% Confidence Interval (CI)
1	ALL TYPES:	168.700	180	1.067	0.917 - 1.235
	LUNG:	24.640	28	1.136	0.755 - 1.642
	BRAIN/NS	2.251	6	2.665	0.973 - 5.801
2	ALL TYPES:	262.486	272	1.036	0.917 - 1.167
	LUNG:	38.454	41	1.066	0.765 - 1.446
	BRAIN/NS:	3.607	9	2.495**	1.139 - 4.737
3	ALL TYPES:	278.769	287	1.030	0.914 - 1.156
	LUNG:	40.300	51	1.266	0.942 - 1.664
	BRAIN/NS:	4.16	7	1.683	0.674 - 3.467

\*\* Statistically high, p <0.05

NOTE - Unlocatable cases include: all sites = 53  
 lungs = 13  
 brain/ns =4

TABLE 12

ALL BIRTHS TO RESIDENT WOMEN, 1961-65

	<u>TOTAL</u>	<u>AREA 1</u>	<u>AREAS 2 &amp; 3</u>
A. ELIGIBLE BIRTHS <sup>1</sup> :			
BIRTHS	2801	583	2218
B. RACE:			
NON-WHITES	187	9	178
WHITES	2148	478	1670
MISSING DATA <sup>2</sup>	466	96	370
C. SEX (WHITES ONLY):			
MALES	1144	253	891
FEMALES	1004	225	779

<sup>1</sup> Singleton births with information on residence, birthweight and with maternal residence in Areas 1, 2 or 3 at the time of birth.

<sup>2</sup> During part of the time period 1962-63 information on race was not requested on the NJDOH Vital Statistic birth certificate.

TABLE 13

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1961-65

(NUMBER = 2135)<sup>1</sup>

A. POTENTIAL RISK FACTORS:	<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
	MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
MATERNAL AGE (YEARS)	26.2	(5.6)	477	26.9	(5.8)	1658
GESTATIONAL AGE (WEEKS)	40.1	(2.4)	477	40.1	(2.6)	1658

B. AVERAGE BIRTHWEIGHT: (GRAMS)	<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
	MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
	3384.5	(554.5)	477	3383.5	(554.3)	1658

C. BIRTHWEIGHT DISTRIBUTION: (GRAMS)	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
501-1499	4	(0.8)	1	(0.1)
1500-1999	3	(0.6)	16	(1.0)
2000-2499	18	(3.8)	74	(4.5)
2500-2999	85	(17.8)	278	(16.7)
3000-4699	364	(76.3)	1268	(76.5)
4700 +	3	(0.6)	21	(1.3)

p < 0.05 \*

D. LOW BIRTHWEIGHT (<2500 GRAMS):

	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
YES	25	(5.2)	91	(5.6)
NO	452		1567	

Odds Ratio = 0.95      90%CI = 0.65, 1.39      p > 0.10 \*\*

<sup>1</sup> 13 births had missing gestational age or gestational age < 28 weeks.

\* Chi-square test, 5 degrees of freedom.

\*\* Chi-square test, 1 degree of freedom.

TABLE 14

GESTATIONAL AGE > 27 WEEKS, 1961-65

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 2135)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	14.30	(25.6)	>.10
sex of child	-147.00	(21.0)	<.05
gestational age	164.00	(7.0)	<.05
maternal age	4.90	(2.1)	<.05
primiparity	-	-	>.10
previous stillborn	-	-	>.10
area of residence and sex of child (interaction term)	-	-	>.10

R<sup>2</sup> = 0.22

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 2135)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	-0.04	(0.28)	>.10
sex of child	-	-	>.10
gestational age	-0.78	(0.06)	<.05
maternal age	-	-	>.10
primiparity	0.59	(0.28)	<.05
previous stillborns	-	-	>.10
area of residence and sex of child (interaction term)	-	-	>.010

Odds Ratio = 0.97

90% CI = 0.61, 1.52

\* Coefficients are given for variables other than area of residence only if the p-values are greater than 0.05 and less than 0.10.

TABLE 15

\*  
TERM BIRTHS, 1961-65

(NUMBER = 1980)

A. AVERAGE BIRTHWEIGHT (GRAMS)

<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
MEAN	(STANDARD DEVIATION)	NUMBER	MEAN	(STANDARD DEVIATION)	NUMBER
3426.9	(488.5)	447	3427.4	(506.8)	1533

B. LOW BIRTHWEIGHT (<2500 GRAMS)

	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
YES	12	(2.7%)	49	(3.2%)
NO	435		1484	

ODDS RATIO = 0.84

90%CI = 0.49, 1.44

p > 0.10\*\*

\* Term is defined as 38-42 weeks gestational age.  
 \*\* Chi-square test, 1 degree of freedom.

TABLE 16

<sup>\*</sup>  
TERM BIRTHS, 1961-65

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 1980)

VARIABLE	COEFFICIENT <sup>**</sup>	STANDARD ERROR	P-VALUE
area of residence	18.30	(26.1)	>.10
sex of child	-152.00	(22.0)	<.05
gestational age	137.00	(14.0)	<.05
maternal age	5.60	(2.1)	<.05
primiparity	-	-	>.10
previous stillborn	-	-	>.10
area of residence and sex of child (interaction term)	-	-	>.10

$R^2 = 0.07$

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 1980)

VARIABLE	COEFFICIENT <sup>**</sup>	STANDARD ERROR	P-VALUE
area of residence	0.26	(0.41)	-
sex of child	0.40	(0.30)	-
gestational age	-0.83	(0.15)	<.05
maternal age	-	-	>.10
primiparity	0.79	(0.32)	<.05
previous stillborns	-	-	>.10
area of residence and sex of child (interaction term)	-1.28	(0.74)	>0.05 & <0.10

Odds Ratio = 1.30 for males and 0.36 for females.

90% CI = 0.66, 2.55 for males and 0.13, 0.97 for females.

\* Term is defined as 38-42 weeks gestational age.

\*\* Coefficients are given for variables other than area of residence only if their p-values are less than 0.10.

TABLE 17

ALL BIRTHS TO RESIDENT WOMEN, 1971-75

	<u>TOTAL</u>	<u>AREA 1</u>	<u>AREAS 2 &amp; 3</u>
A. ELIGIBLE BIRTHS <sup>1</sup> :			
BIRTHS	2151	485	1666
B. RACE:			
NON-WHITES	234	13	221
WHITES	1910	472	1438
MISSING DATA <sup>2</sup>	7	0	7
C. SEX (WHITES ONLY):			
MALES	1025	261	764
FEMALES	885	211	674

<sup>1</sup> Singleton births with information on residence, birthweight and with maternal residence in Areas 1, 2 or 3 at the time of birth.

<sup>2</sup> Race was not reported on the NJDOH Vital Statistic birth certificate.

TABLE 18

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1971-75

(NUMBER = 1815)<sup>1</sup>

A. POTENTIAL RISK FACTORS:

	<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
	MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
MATERNAL AGE (YEARS)	24.6	(4.7)	446	25.5	(5.2)	1369
PARITY (NUMBER OF PREGNANCIES)	2.0	(1.2)	446	2.2	(1.5)	1363
% POOR PRENATAL CARE <sup>2</sup>	52.0%		427	51.9%		1285
MATERNAL EDUCATION (YEARS)	12.4	(2.1)	425	12.4	(2.1)	1269
GESTATIONAL AGE (WEEKS)	40.1	(2.5)	446	40.2	(2.5)	1369

B. AVERAGE BIRTHWEIGHT: (GRAMS)

	<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
	MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
	3360.4	(568.4)	446	3440.1	(544.6)	1369

<sup>1</sup> 95 births had missing gestational age or gestational age < 28 weeks.

<sup>2</sup> The method is described in Institute of Medicine, National Academy of Sciences' study on infant death, 1973. The variables are month prenatal care began and number of prenatal visits as reported on the birth certificate.

TABLE 19

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1971-75

(NUMBER = 1815)<sup>1</sup>

(GRAMS)	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
501-1499	2	(0.5)	7	(0.5)
1500-1999	11	(2.5)	10	(0.7)
2000-2499	15	(3.4)	38	(2.8)
2500-2999	68	(15.3)	188	(13.7)
3000-4699	346	(77.6)	1109	(81.0)
4700 +	4	(0.9)	17	(1.2)

0.10 > p > 0.05 \*

B. LOW BIRTHWEIGHT (<2500 GRAMS):

	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
YES	28	(6.3)	55	(4.0)
NO	418		1314	

ODDS RATIO = 1.60      90% CI = 1.08, 2.37      p < 0.05 \*\*

<sup>1</sup> 95 births had missing gestational age or gestational age < 28 weeks.

\* Chi-square test, 5 degrees of freedom.

\*\* Chi-square test, 1 degree of freedom.

TABLE 20

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1971-75

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 1815)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	-66.10	(26.7)	<.05
sex of child	-178.00	(23.0)	<.05
gestational age	92.00	(5.0)	<.05
maternal age	5.00	(2.7)	<.10
primiparity	-92.00	(27.0)	<.05
maternal education	11.60	(5.9)	<.10
prenatal care	-59.00	(24.0)	<.05
complications during pregnancy	-237.00	(61.0)	<.05
previous stillborn	-	-	>.10
area of residence and sex of child (interaction term)	-	-	>.10

R<sup>2</sup> = 0.22

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 1815)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	0.49	(0.28)	<.10
gestational age	-0.53	(0.05)	<.05
complications during pregnancy	1.60	(0.40)	<.05
area of residence and sex of child (interaction term)	-	-	>.10

Odds Ratio for area of residence = 1.63. 90% CI = 1.02, 2.61

\* Variables with p-values greater than 0.10 that were included in the analysis were: sex of child, maternal age, maternal education, prenatal care, primiparity and previous stillborns.

TABLE 21

\*  
TERM BIRTHS, 1971-75

(NUMBER = 1420)

A. AVERAGE BIRTHWEIGHT (GRAMS)

MEAN	<u>AREA 1</u>		MEAN	<u>AREAS 2 &amp; 3</u>	
	(STANDARD DEVIATION)	NUMBER		(STANDARD DEVIATION)	NUMBER
3402.6	(477.6)	341	3469.8	(484.7)	1079

B. LOW BIRTHWEIGHT (<2500 GRAMS)

	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
	YES	11	(3.2%)	18
NO	330		1061	

ODDS RATIO = 1.97      90% CI = 1.04, 3.75      0.10 > p > 0.05\*\*

\* Term births defined as 38-42 weeks gestational age.

\*\* Chi-square test, 1 degree of freedom.

TABLE 22

\*  
TERM BIRTHS, 1971-75

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 1420)

VARIABLE	COEFFICIENT**	STANDARD ERROR	P-VALUE
area of residence	-74.00	(28.0)	<.05
sex of child	-198.00	(24.0)	<.05
gestational age	110.00	(10.0)	<.05
maternal age	7.20	(2.9)	<.05
primiparity	-83.00	(29.0)	<.05
maternal education	14.50	(6.3)	<.05
prenatal care	42.00	(24.0)	<.10
area of residence and sex of child (interaction term)	-	-	>.10

Variables included in the analysis with p-values > 0.10 were: complications during pregnancy and previous stillborns.

$R^2 = 0.14$

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 1420)

VARIABLE	COEFFICIENT**	STANDARD ERROR	P-VALUE
area of residence	0.73	(0.40)	<.10
sex of child	0.70	(0.39)	<.10
gestational age	-0.82	(0.18)	<.05
complications during pregnancy	1.40	(0.70)	<.05
area of residence and sex of child (interaction term)	-	-	>.10

Variables included in the analysis with p-values > 0.10 were: maternal age, maternal education, primiparity, prenatal care and previous stillborns.

Odds Ratio for area of residence = 2.07                      90 % CI = 1.07, 3.99.

\* Term births defined as 38-42 weeks of gestational age.

\*\* Coefficients are for variables other than area of residence only if their p-values are less than 0.10.

TABLE 23

ALL BIRTHS TO RESIDENT WOMEN, 1981-85

	<u>TOTAL</u>	<u>AREA 1</u>	<u>AREAS 2 &amp; 3</u>
A. ELIGIBLE BIRTHS <sup>1</sup> :			
BIRTHS	1981	421	1560
B. RACE:			
NON-WHITES	259	4	255
WHITES	1692	407	1285
MISSING DATA <sup>2</sup>	30	10	20
C. SEX (WHITES ONLY):			
MALES	871	219	652
FEMALES	821	188	633

<sup>1</sup> Singleton births with information on residence, birthweight and with maternal residence in Areas 1, 2 or 3 at time of birth.

<sup>2</sup> Race was not reported on the NJDOH Vital Statistic birth certificate.

TABLE 24

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1981-85

(NUMBER = 1666)<sup>1</sup>

A. POTENTIAL RISK FACTORS:		<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>		
VARIABLE	MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
MATERNAL AGE (YEARS)	26.3	(5.3)	402	26.1	(4.9)	1264
PARITY (NUMBER OF PREGNANCIES)	1.9	(1.0)	399	1.9	(1.1)	1255
% POOR PRENATAL CARE <sup>2</sup>		24.2%	396		27.5%	1249
MATERNAL EDUCATION (YEARS)	12.9	(2.2)	401	13.0	(2.1)	1260
GESTATIONAL AGE (WEEKS)	39.8	(1.6)	402	39.8	(1.5)	1264

B. AVERAGE BIRTHWEIGHT:  
(GRAMS)

<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
3482.6	(540.1)	402	3458.6	(575.1)	1264

<sup>1</sup> 26 births had missing gestational age or gestational age < 28 weeks.

<sup>2</sup> The method is described in Institute of Medicine, National Academy of Sciences' study on infant death, 1973. The variables are month prenatal care began and the number of prenatal visits as reported on the birth certificate.

TABLE 25

WHITE BIRTHS, GESTATIONAL AGE > 27 WEEKS, 1981-85

(NUMBER = 1666)<sup>1</sup>

(GRAMS)	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
501-1499	2	(0.5)	13	(1.0)
1500-1999	1	(0.3)	7	(0.6)
2000-2499	15	(3.7)	32	(2.5)
2500-2999	42	(10.5)	161	(12.7)
3000-4699	336	(83.6)	1031	(81.6)
4700 +	6	(1.5)	20	(1.6)

p > 0.10 \*

B. LOW BIRTHWEIGHT (<2500 GRAMS):

	OBSERVED	(PERCENT)	OBSERVED	(PERCENT)
YES	18	(4.5)	52	(4.1)
NO	384		1212	

ODDS RATIO = 1.09

90% CI = 0.69, 1.73

p > 0.10 \*\*

\* Chi-square test, 5 degrees of freedom.

\*\* Chi-square test, 1 degree of freedom.

TABLE 26

GESTATIONAL AGE > 27 WEEKS, 1981-85

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 1666)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	-43.00	(40.0)	-
sex of child	-177.00	(29.0)	-
gestational age	81.00	(5.0)	<.05
maternal age	-	-	>.10
primiparity	-120.00	(28.0)	<.05
maternal education	18.20	(6.9)	<.05
prenatal care	-73.00	(29.0)	<.05
complications during pregnancy	-144.00	(47.0)	<.05
previous stillborn	-	-	>.10
area of residence and sex of child (interaction term)	132.00	(59.0)	<.05

$R^2 = 0.19$

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 1666)

VARIABLE	COEFFICIENT*	STANDARD ERROR	P-VALUE
area of residence	0.77	(0.40)	-
sex of child	0.38	(0.35)	-
gestational age	-0.50	(0.05)	<.05
primiparity	0.88	(0.30)	<.05
complications during pregnancy	1.38	(0.34)	<.05
area of residence and sex of child (interaction term)	-1.44	(0.77)	>.05 & <0.10

Odds Ratio = 2.16 for males and 0.51 for females.

90% CI = 1.12, 4.17 for males and 0.18, 1.50 for females.

\* Variables included in the analysis with p-values greater than 0.10 were: maternal age, maternal education, prenatal care, and previous stillborns.

TABLE 27

<sup>\*</sup>  
TERM BIRTHS, 1981-85  
 (NUMBER = 1306)

A. AVERAGE BIRTHWEIGHT (GRAMS)

<u>AREA 1</u>			<u>AREAS 2 &amp; 3</u>		
MEAN	STANDARD DEVIATION	NUMBER	MEAN	STANDARD DEVIATION	NUMBER
3540.2	(508.1)	315	3525.9	(482.4)	919

B. LOW BIRTHWEIGHT (<2500 GRAMS)

	<u>AREA 1</u>		<u>AREAS 2 &amp; 3</u>	
YES	7	(2.2%)	13	(1.3%)
NO	308		978	

ODDS RATIO = 1.71      90% CI = 0.79, 3.73      p > 0.10<sup>\*\*</sup>

\* Term is defined as 38-42 weeks gestational age.

\*\* Chi-square test, 1 degree of freedom.

TABLE 28

\*  
TERM BIRTHS, 1981-85

A. MULTIPLE REGRESSION ON BIRTHWEIGHT (NUMBER = 1306)

VARIABLE	COEFFICIENT**	STANDARD ERROR	P-VALUE
area of residence	-47.00	(41.0)	-
sex of child	-164.00	(29.0)	-
gestational age	112.00	(11.0)	<.05
maternal age	-	-	>.10
primiparity	-93.00	(28.0)	<.05
maternal education	15.00	(7.0)	<.05
area of residence and sex of child (interaction term)	123.00	(60.0)	<.05

Variables included in the analysis with p-values > 0.10 were: maternal age, prenatal care, complications of pregnancy and previous stillborns.

$R^2 = 0.11$

B. LOGISTIC REGRESSION ON LOW BIRTHWEIGHT (NUMBER = 1306)

VARIABLE	COEFFICIENT**	STANDARD ERROR	P-VALUE
area of residence	0.50	(0.50)	>.10
gestational age	-0.42	(0.19)	<.05
primiparity	1.19	(0.51)	<.05
complications during pregnancy	1.45	(0.54)	<.05
area of residence and sex of child (interaction term)	-	-	>.10

Variables included in the analysis with p-values > 0.10 were: sex of child, maternal age, maternal education, prenatal care and previous stillborns.

Odds Ratio for area of residence = 1.65                      90% CI = 0.72, 3.75

\* Term births defined as 38-42 weeks of gestational age.

\*\* Coefficients are for variables other than area of residence only if their p-values are less than 0.10.

TABLE 29

COMPARISON OF EFFECTS OF LIFESTYLE ON LOW BIRTHWEIGHT TO  
PRESENT FINDINGS (1)

Lifestyle Factor During Pregnancy	Reduction In Birthweight
Cigarette smoking	150 to 250 g (5.3-8.8 oz.)
Marijuana use 2-3 times/week	127 g (4.5 oz.)
Caffeine consumption of over 300mg/day	105 g (3.7 oz.)
Lipari findings for term births 1971-75	74 g (2.5 oz.)
Community exposure to nearby copper smelter in Sweeden	68 g (2.4 oz.)
Caffeine consumption of 151 to 300 mg/day	31 g (1.1 oz.)
Caffeine consumption of 1 to 150 mg/day	6 g (0.2 oz.)
Medically supervised lifestyle change	Increase in Birthweight
Supplementation of maternal diet due to mild to moderate malnutrition during pregnancy	40 to 80 g (1.4-2.8 oz.)

(1) see Kline, 1987; Martin, 1987; Nordstrom, 1978; Susser, 1984.



**APPENDIX**



APPENDIX

LIPARI LANDFILL, MANTUA TOWNSHIP, GLOUCESTER COUNTY

Final Draft Reports: USEPA On-site FS, 8/85, Off-site RI, 6/87, CDM

The site is approximately 15 acres in a mixed agricultural and residential area located in Mantua Township, NJ, adjacent to the towns of Pitman, Glassboro and Harrison. In close proximity to the northeast of the site is a housing development containing single family homes. Surrounding much of the site are the Zee Orchards.

The off-site area encompasses the towns of Pitman and Glassboro as well as Harrison and Mantua Township and also includes the drainage basin ENE of the site. Chestnut Branch, the main drainage system, has its headwaters above the site and flows 1500 feet downstream past the NE section of the site. Rabbit Run, a small tributary of the Chestnut Branch, derives its headwater flow from a small spring located adjacent to the site and flows along the full length of the site's NW edge before it discharges to Chestnut Branch at a point north of the site. The two streams drain about a 3 square mile area, converge and flow north into the man-made, 18.5-acre Alcyon Lake. The lake has an average depth of 3.4 feet with a maximum of 6.4 feet and about 4,800 feet of shoreline. (The outflow of the lake eventually empties into Mantua Creek). The lake is also fed by Girl Scout Branch which originates NW of the site. Lost Lake Run originates NE of the site within the residential area and discharges into Chestnut Branch east of the site. Three public parks, Alcyon, Betty and Hollywood Dell are also included in the off-site area.

The urbanized communities of Pitman (1980 pop. 9,744) and Glassboro (1980 pop. 14,644) are both located within one mile of the site and, within Pitman, there are houses located across Chestnut Branch within several hundred feet of the site. Apple and peach orchards form the remaining borders of the site. Orchards are the predominant land use to the NW and SW of the site.

The site was purchased by Mr. Lipari in 1958 for use as a sand and gravel pit and immediately began accepting solid and liquid wastes. Approximately 6 acres in the western portion of the site were used for waste disposal. Municipal and household wastes, liquid and semi-solid chemical wastes, and other industrial wastes were dumped. Liquid wastes were emptied from containers and back dumped into the landfill from 1958 to December 1969 and solid wastes were disposed of until May 1971 when the site was closed by the state. At least one explosion and two fires occurred during the period 1958 to December 1969. It is estimated that the site accepted 2.9 million gallons of liquid waste and 12,000 cubic yards of solid wastes. The liquid wastes probably are uncontained. Phenol or amine wastes and residues, solvents, paints, paint thinners, and resins and ester presscakes were dumped. In 1970, NJDOH observed leachate seeping out from the site and discharging into Chestnut Branch. Seeps visible along the E and NE slopes, were brown and viscous in appearance and had a pungent irritating odor noticeable to area residents, particularly those residing along Howard Avenue. The site was closed in June of 1971 with the impetus of an affidavit signed by local residents that complained of intolerable odors, headaches, nausea and residents' inability to breathe.

After closure of the site, no investigation occurred until 1979. In January 1979, the New Jersey Solid Waste Authority (NJSWA) sampled Chestnut

Branch marsh and found bis (2-chloroethyl)ether (BCEE) (120 ppm), methyl isobutyl ketone (MIBK) (83 ppm), acetone (51 ppm), phenol (28 ppm), toluene (16 ppm), methyl ethyl ketone (MEK) (9 ppm), methylene chloride (6 ppm) and other organics. No action was taken as a result of these findings. Air monitoring samples were taken September 1979 by New Jersey Institute of Technology under NJDEP supervision - 3 samples at or near the seeps in the marsh, one bordering a private residential property and one near the Carew Avenue bridge which transverses Chestnut Branch. The NJDEP concluded that the contaminants identified were well within the usual ranges when analyzed by gas chromatograph (GC). However, the sample near the bridge was also analyzed by gas chromatograph/mass spectrophotometer (GC/MS) and BCEE was detected in an unquantifiable concentration. In September 1979 the NJDEP sampled leachate from the marsh and found similar contaminants including BCEE. Fish samples taken from Alcyon Lake in December 1979 had BCEE levels between 50 and 116 ppb. The lake was subsequently closed to fishing. In July 1984; the NJDEP tested air samples for total volatile organic chemicals (TVOCs) in the basements of 19 homes along Howard Avenue in response to citizen concerns of indoor air pollution caused by the flooding of basements with contaminated water. No unusual odors or readings were detected although one sample was slightly elevated possibly due to open cans of paint and paint thinner in the basement.

The Borough of Pitman and the Gloucester County Planning Department performed sampling in 1978-79 of the leachate seeps, Chestnut Branch, Alcyon Lake, groundwater and a municipal potable supply well. The pesticides dieldrin and endrin were present at less than 1 ppb in Alcyon Lake and Lost Lake Run. All parameters analyzed in the municipal well were determined to be within applicable maximum contaminant levels (MCLs). Captan was detected in runoff from the Zee Orchards. The Gloucester County study concluded that poor water

quality (i.e. contamination by oil and grease, suspended solids, metals and light hydrocarbons) was due to urban and agricultural runoff as well as the landfill. The studies implicated the landfill for the contamination of groundwaters, surface waters and sediments in Chestnut Branch and Alcyon Lake.

The USEPA performed testing on samples of leachate in the marsh area in 1979 and detected BCEE at levels from 38 to 76 ppm as well as other organic compounds. Further leachate samples found BCEE at 210 ppm, toluene (22 ppm), benzene (1 ppm), ethyl benzene (1.1 ppm), phenol (5.9 ppm), and metals. BCEE was detected in all surface water samples taken from Chestnut Branch, Rabbit Run and Alcyon Lake. No contamination was found in potable wells. Warning signs were placed along Chestnut Branch and Alcyon Lake in 1980. In 1981-82, the EPA sampled Chestnut Branch, Lost Lake Run, Rabbit Run, soil below the leachate seeps, Alcyon Lake and 8 private wells in Pitman. None of the private potable wells were found to have "significant amounts of priority pollutants." Downstream surface water samples detected BCCE, naphthalene and phenol. One leachate seep area soil sample detected benzene, toluene and other organics.

In 1982-83, fencing was installed around the 16 acre main landfill site and along Chestnut Branch between the houses on Howard Avenue and east of Chestnut Branch. In 1983, the 16 acre main landfill site was encapsulated and surrounded by a slurry wall to reduce the flow of leachate and contaminated groundwater from the site.

Pitman consists of predominantly medium density housing (greater than 7 dwellings per acre 1980 census). The residential community of Pitman, particularly that at Howard, Lake, Lakeside, and Lakeview Avenues, is within a

few hundred feet east of the site and/or Alcyon Lake. Located 1/2 mile from the site are 2 elementary schools. Three public parks border Alcyon Lake; all have picnic areas and playground equipment. Hollywood Dell Park has a baseball field and Betty Park is the most utilized. Before its fencing, the marsh area was used by joggers, hikers, etc. Although the marsh is now fenced and posted, the area is still accessible due to lack of security. Bacterial contamination led the County to close Alcyon Lake to swimming in 1958. Fishing was banned in 1979. Compliance with the swimming and fishing bans can not be verified. Local residents receive drinking water from a public supply. Citizens are also concerned about flooding of the parks and the illegal dumping near the Carew Avenue Bridge.

#### SURFACE SOIL CONTAMINATION

Sampling occurred in 1985-1986.

##### 1. Chestnut Branch Marsh

All 14 indicator chemicals (benzene, BCEE, chloroform, ethyl benzene, 1,2-dichloroethane, 4-methyl-2-pentanone, toluene, xylene, arsenic, lead, chromium, mercury, nickel and zinc) as well as carbon disulfide, were present in the marsh west of Chestnut Branch during sampling in June 1985 and May 1986. No pesticides or PCBs were detected. However, during the confirmatory sampling, the organochlorine pesticides, 1,1-dichloro-2,2-bis (p-chlorophenyl)ethane (DDD), dichlorodiphenyldichloroethylene (DDE), 1,1,1-trichloro-2,2-bis (p-chlorophenyl)ethane (DDT), alpha-hexachlorocyclohexane (a-BHC), b-BHC, heptachlor and dieldrin were detected. The authors conclude: "The pesticides observed in soil samples west of Chestnut Branch are probably due to other sources such as agricultural runoff and other nonpoint sources, since pesticides have not

been exclusively associated with the Lipari Landfill leachate." Poly aromatic hydrocarbons (PAH's) were also detected but are not attributed to the site by the authors since they were also detected in upgradient "background" samples and were not detected in the most recent leachate analysis from the site.

The only organic indicator chemical detected in the marsh east of Chestnut Branch was 4-methyl-2-pentanone (40 ppb). PAH's such as anthracene, benzo(a)anthracene (BAP), chrysene, indeno(1,2,3)pyrene, etc. were detected as were several pesticides including DDT (110 ppb), and chlordane (4.9 ppm). The authors claim that PAH's and pesticides "... cannot be said to solely be attributable to the Lipari Landfill." Several areas of burned waste piles were evident. Lead (476 ppm), chromium (400 ppm) and other indicator inorganics were detected.

## 2. Parks Surrounding Alcyon Lake

Toluene was the only organic indicator chemical present in a surface soil sample. Present in soil samples at Betty Park taken at depths of 1 1/2 to 2 feet were nonindicator organics such as trichloroethylene (TCE) (43 ppb) and trichloroethane (TCA) (190 ppb). In surface soil samples at Betty Park DDT (181 ppb) and its metabolites were detected. DDT and its metabolites were detected at Hollywood Dell Park and Alcyon Park. Chlordane (119 ppb) was detected at Betty Park and Hollywood Dell Park. Arsenic was detected at Hollywood Dell Park (64 ppm) and Alcyon Park (61 ppm), both above the limits set by the Environmental Clean-up and Responsibility Act (ECRA). Elevated levels of other indicator inorganics were detected except mercury. "ATSDR concluded that the measured levels of pesticides found in [the parks] do not represent a public health threat (ATSDR 1986)." ATSDR also

concluded that levels of inorganics in the parks were "unremarkable" and are not at "background levels". Camp Dresser & McGee's (CDM's) risk assessment identified a risk to arsenic and lead in the parks.

#### SURFACE WATER AND SEDIMENT CONTAMINATION

Sampling occurred in 1985-1986.

The highest concentration of BCEE occurred in Rabbit Run (87 ppb) and detectable levels were still present below the Alcyon Lake spillway (17 ppb). The indicator chemicals which exceeded the human health criteria for fish consumption from Alcyon Lake were BCEE and mercury. The arsenic criterion was exceeded in Rabbit Run. "The indicator chemicals BCEE, chloroform, arsenic and mercury were elevated above background in the sediment samples. BCEE and mercury were elevated in Alcyon Lake sediments. Lead was elevated in Chestnut Branch just below the Lipari Landfill." Compared to "background" upstream sediment samples, "chromium, lead, zinc and mercury were elevated, and all inorganics except for chromium were also elevated in surface waters. "None of the metals exceed the standards for an FW-2 NT<sup>1</sup> water (Alcyon Lake)."

#### POTABLE SUPPLIES

Public water supply wells within a 2-mile radius of the site, and nine private wells in Pitman, were tested in 1985-86. Existing or proposed Safe Drinking Water Act (SWDA) standards were not exceeded in any of the

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(1) Water classification: fresh water-2, non-trout.

public water samples. No organics were detected in any of the private wells sampled and the inorganic results were below the primary drinking water standards. "For some chemicals (benzene, 1,1-DCE, PCBs, toluene, beryllium and mercury), the Federal standards are lower than the analytical limit of detection."

#### AIR CONTAMINATION

Odors emanating from the site have been attributed to leachate seepage from the ENE area of the site into Chestnut Branch marsh. Air studies in 1979 concluded that, except for BCEE, air contaminants identified in the marsh area "were well within the usual range of those monitored by [DEP's] air quality program." Relatively high levels of toluene and p-xylene were also found in the marsh. BCEE was detected at the Carew Avenue Bridge near the Howard Avenue residential area. In 1984, NJDEP conducted air monitoring of 19 basements to determine the presence of VOCs due to groundwater seepage but found no unusual odors or readings. In 1985, CDM found VOCs in the ppm range in the marsh west of Chestnut Branch. In the fall of 1983, backyard air samples detected methylene chloride at high levels (225 ppm) but the blank sample also had levels of the same order of magnitude. TCE (0.1 ppm) and toluene (0.7 ppm) were also detected.

For the offsite remedial investigation, air samples were taken in the marsh leachate seepage area, Alcyon Lake and spillway, Rabbit Run and Chestnut Branch. "Analyses for Alcyon Lake and spillway, Rabbit Run and Chestnut Branch were limited because of the data loss resulting from blank contamination and sample misplacement during the analytical phase." Air

dispersion models were used to estimate exposure at selected receptor locations. Based on modeling and sample data from the area in the marsh with the maximum amount of emissions, CDM concluded that at the fence area, the risk for benzene was 0.0002, PCE 0.000003 and BCEE 0.00066. The maximum long-term concentrations for these contaminants at the fence area were estimated to be  $2 \text{ g/m}^3$ . (Short-term max. conc. = approx.  $20 \text{ g/m}^3$ ). The authors conclude that "VOCs do not exceed the TLV guidelines for short-term (acute) public health risk." However CDM found a potentially chronic health risk for 3 contaminants. Only one valid sample was obtained for the Alcyon Lake area. Benzene (below minimum detection level), toluene (580 ppb), ethyl benzene (85 ppb), xylene isomers (380 ppb), PCE (160 ppb), and TCA 180 ppb) were detected above the surface waters at the lake. USEPA's Field Investigation Team (FIT) performed air monitoring in the summer of 1986 and found that "no significant contaminants have impacted the downwind residential areas."

#### RISK ASSESSMENT CONCLUSIONS

Greater than .000001 cancer risks were estimated for exposure to the maximum concentration of arsenic detected in a soil sample in the marsh west of Chestnut Branch and for exposure of a child to arsenic concentrations in the parks' soils. Exposure of children to lead in the parks' soils "presents intakes greater than the reference dose for the reasonable maximum scenario, but not for the average scenario." For receptor points near the Howard Avenue security fence, modeling indicated that "public health risks could result from lifetime inhalation of volatilized compounds, based on the conditions and assumptions of the modeling and the conservation exposure scenario."

CHRONOLOGICAL SUMMARY OF DISPOSAL AND REMEDIATION HISTORY AT LIPARI

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<u>DATE</u>	<u>ACTIVITY</u>
1958	Sand, gravel, and landfill operations begin.
1963	NJDOH periodically inspects site.
1967/1969	Over 2 million gallons liquid waste disposed in the Lipari Landfill.
1968/1969	Site receives acceptable ratings from NJDOH.
1969	Two landfill fires caused by mishandling waste.
December 1969	Liquid waste disposal ends.
1970	NJDOH inspectors first observe and report leachate seeps along bluff overlooking Chestnut Branch.
June 1971	Solid waste disposal ends.
July 1971	NJDEP notifies Nick Lipari of his responsibility to clean-up site.
1972	NJDEP files suit against Lipari and requests a clean-up of the site.
1972	Lipari implements remedial actions. Lipari constructs drainage ditches, regrades, and spreads lime with little effect.
1973	Lipari spreads lime and fills low area with little effect.
1974	Lipari ordered by court to clean-up site.
1974	Lipari implements additional remedial actions.
1979	Sand and gravel operations end.

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CHRONOLOGICAL SUMMARY OF DISPOSAL AND REMEDIATION HISTORY AT LIPARI

(continued)

<u>DATE</u>	<u>ACTIVITY</u>
July 1982	EPA issues Record of Decision I.
1982	Fence installed around landfill site.
August 1983	Second fence installed along Chestnut Branch.
August 1983	Work begins on remedial actions, including slurry cutoff wall, surface cap, gas vents, and surface water runoff controls.
December 1983	Slurry wall completed, surface cap installation begins.
December 1983	Cold weather stops work on cap with only 70 percent of cap completed.
March 1984	Works resumes on cap.
May 1984	Water table rises to top of cutoff wall, affecting surface cap.
September 1984	Temporary ground water dewatering and treatment system installed.
October 1984	Pump-down completed.
November 1984	Onsite work completed.
September 1985	EPA issues Record of Decision II.



**ADDENDUM**



A D D E N D U M

I. A draft of this report was reviewed in January, 1989 by a Peer Review Panel. The Panel consisted of five members and three alternates who were chosen by a consensus of the Subcommittee in the fall of 1988. The Panel was asked to critique the methodology, interpretation of results, written presentation of the study and recommendations. The panel met with NJDOH study staff on January 11, 1989. The following report was prepared by the Panel as a result of that meeting. Many of the specific suggestions for data presentation or interpretation have already been incorporated during the two weeks between January 11th and the printing of the Report.



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January 19, 1989

The Lipari Health Study Peer Review Panel met on January 11, 1989, to discuss the "Health Study of Residents Living Near the Lipari Landfill" prepared by the New Jersey Department of Health.

The panel arrived at a consensus view that the study was not able to detect an increase in risk for most of the cancers studied, because insufficient time has elapsed from exposure. Thus the study does not contribute to our understanding of possible cancer risks from the landfill.

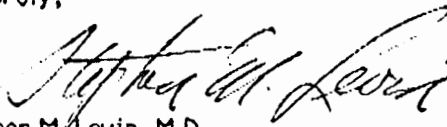
The exception to this general rule is leukemia, where the data are consistent with a biologically plausible effect. Inherent limitations in both the data and the study design, however, make it extremely difficult to conclude at this time that any excess leukemia has resulted from exposure to agents present in the landfill. Limitations in information about exposure and insufficient data on the distribution of other factors that might affect the leukemia rate, in both the exposed and unexposed areas, are the most serious problems. It is not obvious at the moment that these limitations can be overcome.

The panel further wished to emphasize that the study is necessarily silent about other outcomes that might or might not be pertinent. The health outcomes that were studied were chosen, in large part, because they could be studied by looking at vital event data. Hence the study presents a limited inquiry into the health effects issue. Because answers to important public health questions may not be possible, even with unlimited resources, the panel wished to go on record as saying that remedial activity should proceed with all deliberate speed, independent of findings of this or any future study, so as to prevent any further existing or potential exposure from this site.

The study suggested a possible adverse effect of exposure to toxins from the landfill upon average birth weight for one of the three time periods studied (1971 - 75). The decrease in average birth weight among the exposed was small (2.5 oz [74 gm]), however, and may have been due to the effect of other variables that are known to contribute to low birthweight (such as smoking and alcohol consumption), about which no information is available. We cannot conclude, therefore, that the observed effect is attributable to exposure to agents in the landfill.

Given the limitations inherent in the available data and the relatively short time period of exposure which could be analyzed, the study reflected a serious effort to address the community's concerns. The report could be strengthened, however, by providing a better description of the population exposed (eg. age, sex, occupation, migration and socioeconomic characteristics), the geoclimatic characteristics (soil and wind) and the medical facilities available to diagnose cases. Also, the standard used for comparison should include rates from regions closest to that of the study site rather than New Jersey as a whole.

Sincerely,



Stephen M. Levin, M.D.  
Spokesperson for the Peer Review  
Committee

II. The panel, collectively or individually, also made some specific written and oral suggestions regarding further analysis, data presentation, or discussion. These suggestions appear below with comments of the Environmental Health Service staff.

1. *County cancer rates rather than NJ rates could be used as a standard for calculating "expected" numbers of leukemia and other cancers for SIR analyses.*

County rates are not readily available at this time, but could possibly be generated. The DOH plans to perform these further analysis.

2. *Specific distance from the landfill would be a better surrogate for exposure than the more categorical variable of living within or outside Area 1.*

Those distances were not part of the original database. Calculating them would be extremely difficult and labor intensive, and the gain would not be worth the resources needed.

3. *Analysis of other risk factors (cofounders) such as alcohol and tobacco use, occupation, etc. would strengthen the study conclusions.*

4. *Analyzing birthweight for the intervening years 1966-1970 and 1976-1980 is indicated in order to add to data on the time patterns of probable exposure in relation to birthweight.*

To accomplish the manual searches on hundreds of thousands of certificates would entail a level of effort close to that already expended on this study. However, the DOH intends to discuss with the rest of the Health Subcommittee the possibility of obtaining additional resources needed if carrying out this recommendation is deemed essential.

5. *It would be useful to perform sensitivity analyses for other types of cancer besides leukemia, and to break down the cases of unknown residence location by type of cancer.*

The DOH will perform these analyses for brain/nervous system cancers, lung/pleura, and all cancers combined.

6. *Intense case-finding activities for leukemia and a case-control study of leukemia should be undertaken.*

The DOH will discuss with the rest of the Subcommittee the possibilities for such activities.

7. *Cancer analysis should be conducted using the same exposure areas as those used for birthweight.*

This has already been done since the meeting of the Peer Review Panel and is included in the February 1989 report attached.

8. *For consistency, confidence intervals for the cancer analyses and birthweight analyses should be the same.*

The DOH will construct new confidence intervals on one of these data sets and report the outcome. However, we do not expect that changing the confidence intervals will have any affect on the results or inferences.

9. *It would be informative to know how the results would be affected by different assignments of census tracts among sectors (for those tracts which crossed sectors).*

This activity would involve intense use of staff time by the DOH since it must be done manually. Further, age, race, and sex data by census block is limited and is kept confidential by the census bureau when small population numbers are involved.

10. *If children played on the site, cancer incidence ratios should also be calculated for those under 18 years old.*

The DOH will ascertain whether children played on the site through discussions with the Health Subcommittee. If this activity occurred, these calculations will be performed by the DOH.

11. *Live births who were excluded from the analysis because of non-availability of their birth certificate should be presented by exposure area to examine whether there may have been selection bias.*

Only 61 birth certificates were unavailable, and these cannot be assigned to an exposure area because of lack of address.

12. *We should compare low birthweight rates in Area 1 with State rates.*

This was not done since State rates are not reflective of the southern portion of New Jersey where Lipari is located. State rates are much more reflective of low birthweight rates in the urban areas of Northern New Jersey. County rates are not readily available before the 1980's. We believe that the comparison between Area 1 versus Areas 2 and 3 is the most appropriate one, since these areas are similar demographically and sociologically.

13. *The area outside of 2.5 km should be analyzed separately for cancer and birthweight.*

Based upon the limited exposure data in existence, DOH staff do believe that it is neither appropriate to imply that Areas 2 and 3 differ appreciably in exposure potential, nor that separate analyses are necessary for birthweight. The DOH will discuss the

possibility of these analyses with the rest of the Subcommittee, however. Since January 11th, the DOH did reanalyze these areas separately for cancer.

14. *It would be useful for the data analysis to assess the residential stability of the study population via census data.*

Some new demographic tables and descriptions are already included in the report, including in-migration patterns. The DOH will explore whether further data exist and should be acquired for this purpose.

15. *The paucity of lung cancers around the site, in comparison to other common cancers (such as colorectal) suggests possible underreporting.*

The NJDOH does not know of any reason that lung cancer would be under-reported in this area, relative to other types of cancer. We will inquire with local medical institutions as to any available explanation, and will consider whether urban-rural gradients may contribute.

16. *The decrease by decade in the absolute number of live births in the study areas are of interest, and the reasons for this pattern should be explored.*

A discussion on demographic changes has been added to the report, including comparison of national trends with Gloucester County and the four towns.

17. *The identified cases of leukemia in the study area should be presented with details as to sex, age, and histology.*

This information is available and will be presented by the NJDOH shortly after February 1989. It is unlikely, however, that this information will aid in understanding the leukemia results.

18. *It has been suggested that " direct age standardization" be used instead of the "indirect" standardization of the SIR method. This would allow direct comparison of different sub-groups.*

The DOH believes that the population is so small in these sectors that age-specific cancer rates would not be stable enough to use.

19. *Poisson regression analyses should be performed for all the cancer outcomes included in the study.*

Poisson regression analyses were performed for leukemia, lung/pleura and all cancers combined. The results were similar to the results obtained in the SIR analyses presented in Table 10. We expect that the same findings would occur for the other cancer outcomes. For this reason, we decided not to perform Poisson regression analyses on the other cancer outcomes. If the Subcommittee requests reconsideration, we may perform further such calculations.

20. *Population denominators for cancer rates should be shown.*

1982 denominators are available for the age brackets described in the cancer analysis section, and will be presented at a later date.

21. *We should evaluate to what extent smoking could account for the differences in mean birthweight and proportion of low birthweight between Area 1 and Areas 2 and 3.*

Smokers have a mean birthweight of about 200 grams (7.1 oz.) less than non-smokers. Smokers also have 1.5 to 3 times higher proportion of low birthweight babies than non-smokers (NAS data). About 26% of white women of child-bearing age smoke. If we assume that this is also the proportion of smokers among white women of child-bearing age in Areas 2 and 3, then, in order for smoking to account for all of the mean difference for term births in 1971-75 found between Area 1 and Areas 2 and 3, about 65% of white women of child-bearing age in Area 1 would have to smoke. If 35% of the white women of child-bearing age in Area 1 smoked, then, after adjusting for smoking, the mean difference would be under 60 (2.1 oz.) grams and would no longer be statistically significant.

For proportion of low birthweight, smoking could account for all of the excess between Area 1 and Areas 2 and 3 in 1971-75 if all of the women of child-bearing age in Area 1 smoked. If 55% of Area 1 women of child-bearing age smoked, then, after adjusting

for smoking, the odds ratio for Area 1 versus Areas 2 and 3 would be less than 1.5 and would not be statistically significant. It is unlikely that such enormous difference in smoking rates would exist between Area 1 and Areas 2 and 3. Demographically and sociologically, the areas are similar. Therefore, we conclude that if we had information on actual smoking rates in the three areas and adjusted for smoking, differences found between Area 1 and Areas 2 and 3 during the period 1971-75 would probably not be substantially reduced and would probably remain biologically and statistically significant.

22. *We should elaborate on the method used in the birthweight analysis for dealing with missing data.*

If a birth certificate lacked information on birthweight, sex, race or street address, the birth was not included in the study, since area of residence and birthweight were the factors of primary interest. Since race is strongly associated with birthweight, we also decided to exclude subjects without this information. A table illustrating the number of births removed for various reasons has been included with the report.

For all other variables, we followed a strategy presented in Cohen and Cohen: for example, if a subject was missing information on a particular variable such as age of the mother, the mean maternal age for the study area was entered for the subject. A second, "dummy" variable was then created. All subjects with information

on maternal age would be given the value of zero for this dummy variable, while those with missing data on maternal age would be given a value of 1. The dummy variable captures the difference between those with actual values for maternal age and those without, and who were assigned the mean maternal age.

Another strategy is to eliminate subjects with missing data. This was done for those with no information on birthweight, race, sex or street address. This strategy reduces the number of subjects available for analysis and adversely affects the statistical power of the study to detect a health effect when one exists. It may also introduce a bias if those excluded have a different exposure-birthweight relationship than those included in the study (i.e., "selection bias").

23. *Certain birth defects, such as neural tube defects, should be searched among medical registries and records.*

Since the birth defect "registry" is not population-based before 1984, it cannot be used for this study. Researching all area hospital records is a very labor-intensive undertaking, and the issue can be better addressed through other study designs.





