Mortality among Individuals Occupationally Exposed to Benzene

M. GERALD OTT, M.S.
JEAN C. TOWNSEND, M.S.
WILLIAM A. FISHBECK, M.D.
RALPH A. LANGNER, Ph.D.
The Dow Chemical Company
Midland, Michigan

ABSTRACT

The mortality experience of 594 individuals occupationally exposed to benzene was investigated using a retrospective cohort design. Three hundred thirty-five of the employees began working in benzene areas prior to 1950, which provided a sound data base from which to examine latency. Data derived from work histories and industrial hygiene records permitted estimation of exposure intensities and cumulative dosages for each empioya. No mortalities directly attributable to benzene exposure were observed. Several cases of leukemia m d other blood disorders were noted and discussed.

REPORTS of human deaths associated with benzene toxicity date back to the late nineteenth century.[1] At that time, in chronically exposed individuals aplastic anemia was a prominent condition. The possible relationship between chronic benzene exposure and leukemia has been the subject of a number of studies, primarily using the case history approach. Erf and Rhoads,[2] and de Bowin,[3] reported cases of myelocytic leukemia, following anemia which had been associated with earlier benzene exposure. At the University of Milan, forty-seven patients with benzene hemopathy were studied from 1942 to 1963.[4] Six of the patients were leukemic, one of whom exhibited aplastic anemia before leukemia. In 1974, type reported on a corporation survey of eight European affiliates that handled benzene.[5] Although eighteen cases of leukemia were identified in the period 1962 through 1971, the observed number of leukemia deaths was less than expected from mortality rates of the general population. As in many of the previous studies, minimal environmental measurements and exposure histories were available for correlation with medical findings.

The present study examines the long-term mortality experience of a cohort of 594 workmen exposed to benzene at the Michigan Division of Dow Chemical. Availability of employee work histones and exposure data enabled correlation of mortality with cumulative dosage and latency.

Environmental Considerations

Three production areas which use benzene on a continuing basis were selected for inclusion in the present study:

I. Chlorobenzol, date of operation—1920 to present,
II. Alkyl benzene (propylbenzene, ethylbenzene. styrene), date of operation—1935 to present,
III. Ethyl cellulose, date of operation—1936 to present.

In the first two production areas, benzene is a raw material consumed in the process whereas, in Production Area III benzene is employed as a solvent. Benzene concentrations in the work areas were estimated from industrial hygiene surveys and discussions with plant-operating personnel. Other compounds were present in the occupational environment and throughout the manufacturing complex, therefore, none of the employees in the study experienced exposure to benzene alone. Many long-term workers may have been exposed to a wide range of chemicals as a result of transfers to and from non-benzene production areas. A description of the environment in each benzene area is presented in the paragraphs that follow.

Production Area *I.* In this area benzene is chlorinated to produce monochlorobenzene and dichlorobenzene. Benzene is a starting material in the process, and is a potential hazard during the initial chlorination step. Further chlorination and separation into component parts are also carried out within the production area. The unreacted benzene is separated for recycling purposes, and thus, exposure to benzene in these latter operations is minimized. Table 1 summarizes the measurements of benzene levels in Production Area I from 1944 through 1973. As few process changes had occurred between the plant opening and 1944, it was assumed that early exposures were similar to those found in 1944. The low time-weighted-average concentrations (TWA) reflect the use of closed continuous systems. The following categorization rules were adopted for jobs in Production Area I:

Low Exposure (2 to 9 ppm TWA): Mechanics and still operators in the primary chlorination area.

Very Low Exposure (< 2 ppm TWA): Jobs in

Table 1.—Summary of Measurements of Benzene Levels in Production Area I, 1944 to 1973

Job Categories	Years of Survey	Samples Number (per job classification)	Samples Type	Range of Samples ppm Benzene	Estimated TWA ppm Benzene
Not specified	1944-48	43	Area samples	0 - 259	15*
Jobs in early chlorination steps	1964	2- 5	Breathing zone	.2-3.9	.5 - 2.8
Other jobs in production area	1964	1- 7	Breathing zone	0-2.1	.1 - 1.0
Jobs in early chlorination steps	1973	18-24	Breathing zone	.1-62	4.6 - 6.2
Other jobs in production area	1973	3-39	Breathing zone	.1-42	.1 -4.6

*Indicates an average of sample points rather than estimate of TWA.

the production area other than those in the low exposure group.

Because of limited sampling performed prior to 1960, the categorization may be conservative for exposures occurring during that earlier period. In addition to monochlorobenzene and dichlorobenzene, tri- and tetrachlorobenzene were detected in those areas of very low benzene concentrations.

Production Area II. Ethylbenzene production began in 1935 as a semi-plant operation and expanded in 1937 when styrene production was initiated. The benzene is alkylated with ethylene to produce ethylbenzene and diethylbenzene, and with propylene to produce isopropyl benzene. Dehydrogenation leads to the end products styrene, divinylbenzene, and a-methyl styrene. The processes are closed continuous systems, with unreacted benzene being recovered by distillation and recycled. Highest exposures occur in the benzene unloading areas and during sampling of process lines. The levels of benzene found in the alkyl benzene units are shown in Table 2. The following categorization rules were adapted for jobs in Production Area II (shown in abridged form):

High Exposure (25+ ppm TWA): Operating and maintenance jobs in the ethylbenzene process before 1943.

Moderate Exposure (10 to 24 ppm TWA): Operating jobs, ethylbenzene, 1943 to 1960; maintenance mechanics, ethylbenzene unit. 1943 to 1971; operating and maintenance jobs, propyl benzene unit, 1943 to 1968.

Low Exposure (2 to 9 ppm TWA): Operating and maintenance jobs, styrene unit, 1937 to present; miscellaneous jobs in propyl and ethylbenzene over varying periods since 1943.

Very Low Exposure (< 2 ppm TWA): Clerical and supervisory jobs in all areas, and several operating jobs in styrene, from beginning of the process.

Other materials considered to be of toxicologic importance were known to be present on a routine basis in Production Area II, including ethylbenzene, divinylbenzene, toluene, xylene, isopropyl benzene, styrene, diisopropyl benzene, and a-methyl styrene.

Production Area III. In the production and fabrication of ethyl cellulose resins, benzene is used as a solvent. The highest exposures occurred in the sheeting operation where operators entered closed ares to make adjustments on the casting equipment. High concentrations in the sheeting operators were found in 1953. As a result, engineering changes were adopted, including increased localized ventilation. In late 1963 and 1964, concentrations were found to be high due to process changes and the deteriorating condition of the equipment. At that time, detailed medical examinations were given to employees, a continuous monitor was installed, and improvements were made to lower exposure levels. A brief description of the medical findings in ten workmen was reported by Stewart et al.[1] A current review of the medical records of the ten workmen has shown no subsequent hematopoietic abnormality. The sheeting process was shut down in 1967 for economic reasons.

The resin-producing operation, with the loading and unloading of batch reactors, frequent sampling of the process, and existing work practices also contributed to high benzene exposures. In recent years, concentrations have been reduced below 10 ppm TWA. Table 3 summarizes the measurements of benzene levels in Production Area III. Categorization was developed for the ethyl cellulose units similar to those devised for Production Areas I and II. Additional toxic materials in the environment were ethyl chloride, ethyl ether, and ethyl alcohol. In all production areas, TWAs reflect average exposures for employees on a given job rather than exposures particular to an individual whose work practices may differ from the norm.

Method

The population of interest was identified from annual departmental census lists for the years 1938 to 1970. Employees who were assigned to job classifications involv-

Table 2.–Summary of Measurements of Benzene Production Area II, 1953 So 1972

Job Categories	Years of Survey	Samples Number (per job classification)	Samples Type (per job classification)	Range of Samples ppm Benzene	Estimated TWA ppm Benzene
Alkylating operators (one production unit)	1953	5-25	Breathing zone	0-≥100	9.0-11.0
Alkylating operators (across production units)	1965	2-36	Breathing zone	0-56	10-73
Mechanics (across production units)	1965	3-4	Breathing zone	0-250	10-10.0
Other jobs in production units	1965	2-39	Breathing zone	0-56	10-53
Alkylating operators (across production units)	1971-72	30-135	Breathing zone	0-283	3-13.0
Mechanics (across production units)	1971-72	30-94	Breathing zone	0-283	13-14.7
Other jobs in production units	1971-72	13-195	Breathing zone	G74	.6-53

Table 3.–Summary of Measurements of Benzene Levels in Production Area III, 1952-1974

Job Categories	Year of Survey	Samples Number (per job classification)	Samples Type (per job classification)	Range of Samples ppm Benzene	Estimated TWA ppm Benzene
Jobs in manufacturing area	1952	13-25	Breathing zone	0-≥100	17-35.5
Jobs in manufacturing area	1965	4-24	Breathing zone	.2-575	5-17
Jobs in manufacturing area	1973	25-38	Breathing zone	.1-321	38-40
Technician in laboratory	1961	4	Breathing zone	105-210	16
Technician in laboratory	1974	29	Breathing zone	.7-184	4.0
Fabrication area	1953	9	Breathing zone	10-937	35
Fabrication area	1965		Continuous monitoring with infra-red analysers	0-≥100	11-32

ing exposure *to* benzene m the 2 to 9 ppm *range* from Production Area I, and all employees from Production II and III, were included in the *study.* Job histories abstracted from personnel records for each employee coded according to the categorization outlined above. Due to job mobility within the units and changes in environment, most employees were exposed at more than one job assignments for one or mom months wen categorized.

In the analysis, cumulative dosage was calculated by multiplying the mean TWA value for each category of intensity by the number of months spent exposed to each level. The mean TWA value of 1 ppm was used for the very low level, 5 ppm for low level, 17 ppm for moderate level, and 30 ppm for high level.

The mortality data were analyzed by production area,

according to cumulative dosage, and interval since first exposure.

Expected deaths were *calculated* by the indirect method from U.S. white male mortality for the years 1942,1947,1952,1957,1962,1967, and 1971. *Age* and cause-specific standardized mortality ratios (SMRs) were utilized in the comparisons.

The analysis covered the years 1940 through 1973, and included employees working for the company on or after January 1, 1940. Follow-up of employees who left the company was obtained from the Social Security *Administration*. Since Social Security identification of decedents may be less than 100% (approximately 94% for a sample of known company deaths), several could have been missed. A subsequent verification of vital *status* based on personal contacts of former employees, or someone directly knowledgeable regarding their current vital status, revealed two additional deaths and 96 individuals definitely alive. Thus, for 49 individuals vital status determination was made from Social Security records alone.

Copies of death certificates were not obtained for three decedents traced through Social Security, all of whom were exposed to benzene dosages estimated to be less than 1000 ppm X months of exposure (ppm months).

In *the* present benzene cohort, there were 53 persons who were exposed *to* arsenicals, vinyl chloride, or asbestos *at* levels associated with excess malignancies.[7] These employees were excluded from analyses that dealt with dose response in relation to benzene exposure.

Results

The vital and occupational status of the total cohort (594 individuals) is presented in Table 4. Through company records we found 255 employees *still* working for the company, 87 retired, and 71 known deceased. Of the 181 former employees for whom Social Security checks were performed, 31 were identified as dead. Sixty-four of the 181 individuals had worked for less than 1 year in benzene areas; 165 had worked for less than 10 years in benzene areas.

Table 4.—Vital and Occupational Status of Benzene-Exposed Population as of January 1, 1974 by Production Area

Vital and Employment Status	All Production Areas	Production Area I	Production Area II	Production Area III
Total population	594	121	152	321
Still working	255	56	65	134
Retired	87	27	25	35
Deceased (company records)	71	21	24	26
Left company	181	17	38	126
Deceased	31	2	14	15
Verified living	101	10	19	72
Presumed living (based on social security records)	49	5	5	39
Population less exclusions*	541	108	135	298
Still working	233	54	57	122
Retired	75	23	19	33
Deceased (company records)	62	16	22	24
Left company	171	15	37	119
Deceased	29	2	13	14
Verified living	95	9	19	67
Presumed living (based on social security records)	47	4	5	38

*Does not include employees exposed to arsenicals, asbestos, and high vinyl chloride levels.

Table 5.—Duration of Exposure by Date of First Exposure for Each Production Area, and for Employees with Arsenicals, Asbestos, or High Vinyl Chloride Exposure

Production Area and Year of First Exposure	Duration of Exposure				
	Total	<1 Year	1-9 Years	10-19 Years	20+ Years
Production Area I					
Total	108	9	36	20	43
<1940	25	0	5	7	13
1940-1949	42	5	12	3	22
1950.1959	21	2	2	9	8
1960+	20	2	17	1	--
Production Area II					
Total	135	21	64	23	27
<1940	21	3	12	1	5
1940-1949	62	13	21	lo	18
1950-1959	31	3	17	7	4
1960+	21	2	14	5	--
Production Area III					
Total	298	96	139	39	24
< 1940	37	1	13	13	10
1940-1949	147	55	65	13	14
1950.1959	64	21	30	13	0
1960+	50	19	31	0	--
Population with arsenic, asbestos, or high VCL exposure					
Total	53	3	29	6	15
<1940	20	0	9	3	8
1940-1949	20	1	12	1	6
1950-1959	10	2	6	1	1
1960+	3	0	2	1	--

Duration of exposure to benzene by year of initial exposure is given in Table 5 for each production area, and separately for the 53 employees who were also exposed to arsenicals, asbestos, or vinyl chloride. Employment tended to be more stable in Production Areas I and II, than in Area III. Overall, 108 employees in the study worked for 20+ years in areas of benzene exposun; 175 had been potentially exposed to benzene in excess of 25 ppm TWA for at least 1 month.

The cause-specific mortality trends shown in Table 6 agree with results of a cohort study of over 8,000 employees at the same location." Observed deaths were considerably less than expected based on US. white male mortality. No statistically significant increases were found in any cause-of-death category in the population excluding employees with arsenicals, asbestos or high vinyl chloride exposure. Cardiovascular, accidental, and residual causes of death wen much lower in the industrial cohort than expected. Five deaths due to suicide were observed versus 3.1 expected. No association with intensity of benzene exposure or cumulative dosage was found with respect to these individuals.

Detail- review of medical and occupational records was undertaken for five decedents whose medical findings were judged to be of clinical interest with regard to benzene toxicity. Two of the employees died from causes of death categorized under anemias. One death was due to aplastic anemia and the second was due to pernicious anemia; the second diagnosis was confirmed by autopsy. This second employee first worked for the company from 1916 to 1919. Details of exposure for that period are unknown. In 1931 he returned as a laborer in chemical sewage disposal, and had potential exposures to a variety of chemicals. From 1936 to his retirement in 1951, he was exposed to benzene (Production Area III [184 months of high exposure; estimated dosage, 5520 ppm months]) This person died in 1955 at the age of 68 years. No company medical record was available for review.

An autopsy was not performed on the individual who died of aplastic anemia. The decedent's work history at the company began in 1929 with exposure to arsenicals for 5 months. He held a variety of jobs until 1934. Beginning in 1934, he was exposed to low levels of benzene for 98 months (estimated dosage, 453 ppm months) in the chlorobenzol area. From 1942 until his retirement in 1955, he worked in three different locations and his potential exposures included many other chemicals. He died in 1957 at the age of 67. No family history of cancer or blood diseases was noted in the medical record.

The remaining three case histories were of employees with leukemia. One death was due to leukemia, another to acute myelogenous leukemia, and a third to myelo-

Table 6.—Observed and Expected Deaths by Cause Including and Excluding Employees with Arsenicals, Asbestos, or High Vinyl Chloride Exposure, 1940-1973

Cause of Death Category	Total Population Obs/Exp	Total Population SMR	Population Less Exclusions Obs/Exp	Population Less Exclusions SMR
All causes	102/1283	80	911114.1	80
Total malignancies	30/22.8	132	24120.3	108
Buccal cavity & pharynx	1/.8	*	11.7	•
Digestive organs & peritoneum	916.9	130	916.1	131
Respiratory system	10/7.2	125	716.3	111
Genital organs	1/1.4	•	011.3	•
Urinary organs	2/1.5	•	111.3	•
Lymphatic & hematopoietic tissue (except leukemia)	211.5	•	211.3	•
Leukemia	2/1.0	•	11.9	*
All other	312.6	a	312.4	*
Anemias	2/.2	*	11.1	*
Cardiovascular disease	46164.2	72	43157.3	75
Influenza and pneumonia†	312.9	*	312.6	*
Bronchitis, emphysema & asthma	312.3	*	212.1	•
Cirrhosis of liver	313.6	*	313.2	*
Symptoms, senility & ill-defined conditions	111.4	a	111.2	*
Accidents	2/12.0	•	2110.8	*
Suicide	513.4	147	5/3.1	161
Residual causes	4115.4	*	4/13.6	•
No death certificate available (one person killed in military action)	3/—	—	3/—	—

*Less than five observed deaths.

†Includes one person for whom myeloblastic leukemia was listed under "other significant conditions" at death.

blastic leukemia which was listed under "other significant conditions." The first decedent's employment at the company began in 1946, and he spent two years in an area where potential exposures included nitrobenzene and bromine. From 1948 to March 1950, he had several short work assignments with varied exposures. From March 1950 through July 1960, he worked in a location where he had 65 months of potential exposure to vinyl and vinylidene chloride that could have been over 200 ppm. From August 1960 until March 1971, he worked in an alkyl benzene area where his exposure was categorized as low (estimated dosage, 545 ppm months). No family history of cancer was noted for this individual, who died in 1971 at age 45.

The second decedent was employed for 30 months by the company; 18 months (October 1950 to April 1952) involved potential exposure to very low concentrations of benzene (< 2 ppm TWA dosage: 18 ppm months). This person died in 1965 at age 51. Other occupational history included four years in the military and employment from 1948 to 1950 in a saw mill which manufactured veneer. Data on family medical history are lacking.

The third death was categorized as bronchopneumonia bilateral, with myeloblastic leukemia listed under "other significant conditions." This decedent began working at Dow Chemical in 1943. Industrial hygiene surveys taken in his work areas between 1943 and 1955 indicated he had probable exposure to *p*-chlorophenol, ethyl chloride, phenetidine, acetic anhydride, and phenacetin dust. From 1955 until his retirement in 1962, the decedent was potentially exposed to low levels of benzene (estimated dosage, 305 ppm months). Family history included mother's death due to cancer. He died in 1970 at the age of 72.

In Table 7, selected causes of death in the population (less the group exposed to arsenicals, asbestos, and vinyl chloride) are examined with respect to career dosage and interval since initial exposure to benzene. Prior analysis by production area had revealed no statistically significant differences in mortality (Production Area I, SMR = 89; Production Area II, SMR = 90; Production Area III, SMR = 72). For any dosage there were no significant increases in mortality relative to the U.S. population, by cause-of-death category (Table 7). In the interval of less than 15 years since first exposure, the SMRs were considerably

Table 7.—Observed and Expected Deaths* by Interval Since First Exposure and Estimated Career Benzene Dosages for Selected Causes of Death, 1940-1973

Cause of Death and Career Dose Category	Interval Since First Exposure: Total Obs/Exp	Total SMR	<15 Years Obs/Exp	<15 Years SMR	15-24 Years Obs/Exp	15-24 Years SMR	25+ Years Obs/Exp	25+ Years SMR
All causes								
Total	911114.1	80	17140.6	42	40141.9	95	34131.6	108
0-499 ppm months	41165.1	63	12/27.5	44	18124.0	75	11/13.6	81
500-999 ppm months	18116.2	111	215.9	†	1115.9	186	514.4	114
1000+ ppm months	31132.8	98	3/7.2	†	11112.0	92	18113.6	132
Cardiovascular disease	43157.3	75	5/17.6	28	18/22.6	80	20117.1	117
0-499 ppm months	15/32.9	46	3/12.0	†	6/13.4‡	45	6/7.5	80
500-999 ppm months	9/7.7	117	1/2.5	†	5/2.9‡	172	312.3	†
1000+ ppm months	19/16.7	114	113.1	†	7/6.3	111	11/7.3	151
Malignant neoplasms	24120.3	118	6/6.3	95	1017.5	133	816.5	123
0-499 ppm months	11111.2	98	4/4.3	†	5/4.2‡	119	212.7	†
500-999 ppm months	512.9	172	110.9	†	3/1.1‡	†	110.9	†
1000+ ppmmonths	816.2	129	111.1	†	212.2	†	512.9	172
All other a —	22/36.5	60	6116.7	36	10111.8	85	618.0	75
0-499 ppm months	14121.0	67	5111.2	45	6/6.3‡	95	313.4	†
500-999 ppm months	415.6	†	0/2.5	†	2/2.0‡	†	111.1	†
1000+ ppm months	519.9	51	113.0	†	2/35	†	2/35	†

*Based on age-specific death rates (5-year interval) combined by indirect method.

†No cause of death was determined for one person in each of the two lower dose categories.

‡Less than five observed deaths.

lower than expected for cardiovascular disease and all other causes, as might be anticipated in view of preselection factors.

Discussion

No statistidy significant increases in total mortality relative to the US. white d e population were observed with respect to production area or estimated career dosage of benzene. Exposure and health records were reviewed on an individual basis for several employees with medical findings associated in the literature with benzene poisoning. Three case histories of employees with leukemia wen reviewed. The three leukemias were myelocytic with two being classified as acute, Vigliani reported in his investigations that benzene-related leukemias were mostly acute hemocytoblastic or myeloblastic.[9] In our population the expected incidence of leukemia, excluding lymphocytic or monocytic cell types, was 0.8 cases (based on the Third National Cancer Survey, Incidence Data) compared with three observed cases ($P < .047$).

In these cases, varied work histories and the lack of medical history made a retrospective assessment of the possible relationship to benzene exposure very judgmental. Prior employment with a veneer company further complicated the assessment for one person as Milham has reported increased myelocytic leukemia in similar work situations.[10] Ward et al.[11] have suggested that in cases of leukemia associated with benzene exposure, genetically-controlled sensitivity and other environmental factors—especially during childhood—should be investigated. These researchers have been unsuccessful in producing leukemia in laboratory animals subcutaneously exposed to benzene.

An assessment of the case history of the individual with aplastic anemia is similarly difficult. In a series of over 2,000 deaths dating back to 1940 known to the company, this was the only death due to aplastic anemia. From an environmental standpoint, his potential exposure to numerous chemical agents including arsenical compounds, and the latency with respect to benzene exposure, are factors to be considered.

A review of multiphasic health inventories for 282 employees in the current study has been undertaken and will be reported when complete.

* * * * * * * * * *

Submitted for publication December 30.1975; revised, accepted April 28, 1976.

Article copier are available from: M. Gerald Ott, Corporate Medical Dept., The Dow Chemical Company, 2030 Dow Center, Midland, MI 48640.

* * * * * * * * * *

REFERENCES

1. Hunter, D. 1969. *The diseases of occupations*, 4th ed., pp. 506-521. London: The English Universities Press.
2. Erf, L. F. and Rhoads, C. P. 1939. The hematological effects of benzene (benzol) poisoning. *J Ind Hyg Toxicol* 21:421-35.
3. de Gowin, R L. 1963. Benzene exposure and aplastic anemia followed by leukemia fifteen years inter. *JAMA* 185:748-51.
4. Vigiliani, E. C., and Saita, G. 1964. Benzene and leukemia. *N Engl J Med* 271:872-76.
5. Thorpe, J. J. 1974. Epidemiologic survey of leukemia in persons potentially exposed to benzene. *J Occup Med* 16:375-82.
6. Stewart, R. D.; Dodd, H. C; Baretta, E D.; Schaeffer, A. W.; and Mutchler, J. E. 1967. Chronic overexposure to benzene vapor. *Toxicol Appl Pharmacol* 10:381.
7. Ott, M. C.; Langner, R. R.; and Holder, B. B. 1975. Vinyl chloride exposure in a controlled industrial environment. *Arch Envimn Health* 30:333-39.
8. Ott. M. G.; Holder, B B; and Langner, R R 1976. Determinants of mortality in an industrial population. *J Occup Med* 18: 171-77.
9. Vigliani, E. C., and Forni, A. 1976. Benzene and leukemia. *Environ Res* 11:122-27.
10. Milham, S. 1976. Neoplasia in the wood and pulp industry. *Ann NY Acad of Sci* 271:294-300.
11. Ward, J. M.; Weisburger, J. H.; Yamamoto, R. S.; Benjamin, T.; Brown, C. A.; and Weisburger, E. K. 1975. Long-term effect of benzene in C57BL/6n mice. *Arch Environ Health* 30:22-25.

Residence under an Airport Landing Pattern as a Factor in Teratism

F. NOWELL JONES, Ph.D.
Department of Psychology
JUDY TAUSCHER
School of Public Health
University of California
Los Angeles, California

ABSTRACT

An analysis of all Lor Angeles County birth records for the years 1970, 1971, and 1972 reveals a higher incidence of reportable birth defects in those census tracts lying wholly or partly within the 90 dbA loudness contours under the landing pattern at Los Angeles International Airport than in the rest of the County. While not proving that noise is responsible for the increase in teratism, these results point to a potentially important physical environmental effect with significant public health implications.

RECENTLY, considerable attention has been given to the possible mental health effects of aircraft noise on the residents of affected areas near airports. The original impetus appears to have stemmed from a report by Abey-Wickrama et al. who found a higher incidence of psychiatric problems among persons living under the Heathrow flight patterns than among those who lived in comparable, but quieter areas.[1] McLean and Tarnopolsky[2] have reviewed this and other similar reports and have reached the conclusion that the evidence for psychiatric casualties from noise is not compelling, although there is room for further research. Ando and Hattori[3] report that babies born to mothers living near the noisy Osaka airport are of relatively low weight, presumably because of stress to the mothers. It has also been reported that teratogenic effects occur in pregnant rats upon exposure to noise.[4] A British report[5] remarks that the still-birth rate is higher in Hounslow (in part, a very noisy district under the Heathrow traffic pattern) than elsewhere in Greater London. The analyses which we report here tend to confirm the above findings, in that more than expected abnormal births were found to occur to mothers residing in the noisiest census tracts on the approaches to Los Angeles International Airport.

The authors were able to obtain taped compilations of all births recorded in Los Angeles County for the years 1970,1971, and 1972. The data available included, among other things, observable birth defects, race, and census tract of residence. Based on previous work by Meecham and Smith[12] we chose tracts lying wholly or in part within the 90 dbA contour around the landing pattern. This pattern, which was altered for night landing in 1974, extends roughly eastward over a populated area. The "noise area" was divided for all practical purposes into a largely black, eastern region, and a largely white, western region (Spanish surname births were too few for reasonable statistical treatment). Approximately one-half of the births in the noise area were black. (It should be noted that the largely white area was the noisier.) Finding proper control tracts presented a difficult problem; since the noise tracts were middle-of-the-road so far as the white population was concerned, and more affluent than average so far as the black population was concerned, we chose to use the remainder of the entire county for our comparisons. This choice biases the results against finding noise effects because there are other airports (not so heavily used) and many freeways scattered throughout the county. Figure 1 shows the location of the target