truly marine angiosperms such as *Zostera* or *Thalassia*. Aldehyde production from bacteria is too limited to be of consequence in sediments rich in organic matter.

A freshwater sediment from an inlet near the northeastern shore of Barro Colorado Island, Gatun Lake, Panama, was also examined. This sediment is rich in partially degraded organic material derived from a low-lying tropical rain forest inundated during the formation of Gatun Lake, in the development of the Panama Canal. Oxidation with alkaline nitrobenzene yielded chiefly vanillin and syringaldehyde—a result consistent with the source material, mainly residue of deciduous forest trees.

Postglacial peat from Montclair bog, North Quincy, Mass., was examined. The contribution of coniferous wood to this temperate, freshwater deposit is reflected in the relatively high yield of vanillin. The correspondence between the aldehydes generated from peat specimens and the parent plant was clearly demonstrated in earlier studies with peat and soil (*2*, *13*).

On oxidative degradation of nonmarine ancient carbonaceous sediments, small yields of *p*-hydroxybenzaldehyde and vanillin were obtained from Mesozoic and younger specimens. Syringaldehyde was not detected. Lignin remnants may be the progenitors of the aldehydes from these older sediments as well. In sediments, where conditions are anaerobic and anoxic, lignin persists. Lignin is rapidly degraded only under aerobic conditions, or when oxygen can be transferred or dehydrogenation can take place (*11*).

Although older sediments are markedly different in many respects, their aldehyde yields are quite similar, and of the same order of magnitude as those of recent marine sediments.

A sample of Chattanooga shale (Upper Devonian) from northwest Georgia yielded *p*-hydroxybenzaldehyde and vanillin in amounts like those from Mesozoic sediments. This is not surprising, since the Chattanooga shale is rich in humic material (*16*). The specimen examined may have been rich in woody matter, having been collected near the probable shoreline of the Chattanooga Sea during its period of greatest extent.

Investigation of several Precambrian samples did not afford equivocal results. No aldehydes were detected from a Middle Precambrian anthraxolite, an anomalous, anthracite-like carbonaceous substance containing about 95 percent carbon and less than 0.1 percent extractives. Michigamme coal, also of Middle Precambrian age, however, gave approximately 0.01 mg of vanillin per gram of sample. Confirmation by other means is needed because the vanillin yield from our controls approaches this amount.

Paleobotanical evidence suggests that the capacity of plants to synthesize lignin did not evolve until late Silurian and early Devonian time. However, analysis of crude oil 1 billion years old from Precambrian Nonesuch Shale (Upper Michigan) gave 0.07 mg of *p*-hydroxybenzaldehyde and 0.14 mg of vanillin per gram of sample. This might be due to contamination, however, since the precise history of the specimen is not known.

Thus, it seems that phenolic aldehydes can be generated from plant materials of greatly differing ages. The phenolic aldehydes can be separated and identified; their ratios appear to be related to the chemical structure of the lignin in the initial plant. The phenolic aldehydes from the sediments analyzed in this study may be a geochemical index of the contribution of lignin to the organic sediments.

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14 November 1969; revised 9 February 1970 ■

Mutagenicity of Trimethylphosphate in Mice

Abstract. *Subtoxic concentrations of trimethylphosphate, administered orally or parenterally to male mice, produced mutagenic effects, dependent on dosage, in the dominant lethal assay.*

Trimethylphosphate (TMP) is used as a gasoline additive, at a concentration of approximately 0.25 g per gallon, for controlling surface ignition and spark plug fouling (*1*). It is also used as a methylating agent (*2*), a chemical intermediate in production of polymethyl polyphosphates (*3*), a flame retardant solvent for paints and polymers, and a catalyst in preparation of polymers and resins (*1*). Recently the related triethyl phosphate has been proposed as a food additive for stabilizing egg whites (*4*).

Trimethylphosphate is weakly toxic to rodents when administered orally, parenterally, or cutaneously (*5*); long-term administration of high doses in rats induces weight loss and paralysis. The compound is rapidly degraded to dimethyl phosphate, and S-methylcysteine has been identified as a urinary metabolite, an indication that it functions as an alkylating agent in vivo

Table 1. Mean early deaths per pregnancy in female mice mated with males treated with trimethylphosphate (TMP).

Week of mating after drug administration	Control	TMP (mg/kg intraperitoneally)		Control	TMP (mg/kg intraperitoneally)					Control	TMP (mg/kg by gavage)	
		200	1000		500	850	1250	1500	2000		500	1000
					Early deaths per pregnancy (mean)							
1	0.33	0.08	1.25	0.57	0.00	0.30	1.00	0.56	1.89	0.00	0.90	4.00
2	.05	.65	1.82	.53	.60	.75	1.80	2.13	3.70	.23	4.08	0.00*
3	.47	.47	0.69	.20	.67	.43	0.55	0.75	1.29	.21	0.77	.56
4	.68	.55	.17	.45	.29	.50	.11	.30	0.00	.18	.24	.36
5	.47	.00	.33	.40	.30	.08	.33	.50	.25*	.10	.25	.00
6	.67	.20	.22	.62	.30	.27	.20	.15	.13	.13	.12	.12
7	.25	.58	.32	.21	.29	.50	.23	.25	.40*	.40	.06	.40
8	.39	1.10	.43	.25	.25	.70	.14	.36	.29	.07	.21	.13
					Males mated (No.)							
	10	7	9	10	5	5	5	5	5	7	10	10†
					Females pregnant per week (mean)							
	18	13	18	19	10	10	10	10	7	13	15	16

* Six or fewer females pregnant. † One male died in 2nd week.

(*6*); unlike other alkylphosphates, TMP also alkylates glutathione in vitro (*7*). Litter sizes of the F_1 progeny of untreated females mated with males given TMP orally or parenterally are reduced (*6*), presumably by induction of dominant lethal mutations. Mutagenicity of TMP in mammals is also suggested by the fact that it can serve as an alkylating agent and by the fact that it induces reverse mutations in *Neurospora* (*8*). For these reasons, it was considered important to determine the mutagenicity of TMP in mammals directly by the dominant lethal assay (*9*). Dominant lethal mutants are convenient indicators of major genetic damage and have been used for measuring effects of x-rays and chemical mutagens in mammals; the genetic basis for dominant lethality is the induction of structural and numerical chromosomal aberrations, such as translocations and aneuploidies, leading sequentially to loss of nonviable zygotes before implantation, early fetal deaths, and sterility and semisterility in F_1 progeny (*9*).

Trimethylphosphate, freshly prepared in distilled water, was administered to male Swiss (ICR/Ha) mice 56 days old, in 0.1-ml volumes by single intraperitoneal injection, in dosages ranging from 200 to 2000 mg/kg, or by gavage daily for 5 days, in individual doses of 500 or 1000 mg/kg, equivalent to total doses of 2500 or 5000 mg/kg. After treatment, each test and concurrent control male was caged for 1 week with three virgin female Swiss mice. Females were replaced weekly and consecutively for a total of 8 weeks, representing the duration of the spermatogenic cycle. Females were autopsied 13 days after the midweek of their caging and presumed mating; these untimed pregnancies thus ranged from 9 to 15 days. Each female was scored for pregnancy, and for numbers of total implants, as represented by living implants, early fetal deaths, and occasional late fetal deaths; corpora lutea were not counted. The percentage of pregnancies and mean numbers of total implants, including living fetuses and early fetal deaths, were determined weekly for each group. Analysis of vari-

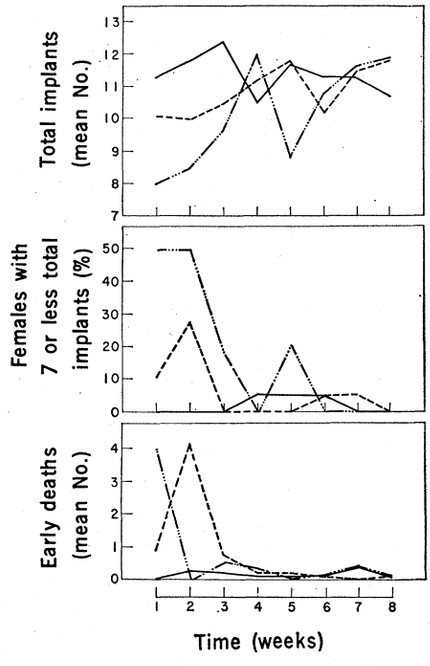

Fig. 1. Numbers of total implants and early fetal deaths in females mated over an 8-week period following treatment of males with trimethylphosphate by gavage on five successive days. Numbers of total implants and early fetal deaths are mean values per pregnant female. Time refers to weeks of mating after termination of treatment of males. (——) Control; (----) 500 mg of TMP per kilogram daily; (——•••——) 1000 mg of TMP per kilogram daily.

Table 2. Analysis of variance; I, intraperitoneally; G, gavage.

Time (weeks)	Dosage (mg/kg)	Route	Within subclass mean square (degrees of freedom)	*F* values (degrees of freedom)		
				Dose	Week	Interaction
			Total implants per pregnancy			
1–3	200, 1000	I	4.69(137)	5.33*(2)	1.96(2)	1.06(4)
4–8	200, 1000	I	5.10(227)	2.06(2)	1.38(4)	0.49(8)
1–3	500–2000	I	4.22(182)	0.29(5)	4.79*(2)	1.25(10)
4–8	500–2000	I	4.21(302)	.19(5)	2.30(4)	1.82‡(20)
1–3	500, 1000	G	5.64(95)	12.96*(2)	1.69(2)	0.24(4)
4–8	500, 1000	G	3.91(198)	0.22(2)	1.29(4)	3.01*(8)
			Early deaths per pregnancy †			
1–3	200, 1000	I	0.91(137)	14.65*(2)	0.73(2)	3.40‡(4)
4–8	200, 1000	I	0.74(227)	2.83(2)	.64(4)	2.45‡(8)
1–3	500–2000	I	1.02(182)	7.86*(5)	5.64(2)	1.19(10)
4–8	500–2000	I	0.51(302)	1.01(5)	0.07(4)	0.85(20)
1–3	500, 1000	G	1.19(95)	13.32*(2)	7.03*(2)	14.03*(4)
4–8	500, 1000	G	0.35(198)	0.03(2)	0.07(4)	0.87(8)

* Significant at $P = .01$. † Data transformed by Freeman-Tukey Poisson transformation before analysis. ‡ Significant at $P = .05$.

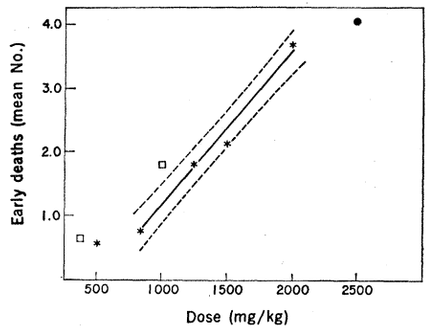

Fig. 2. Dose-response relationship for numbers of early fetal deaths per pregnancy in females mated in the 2nd week after treatment of males with trimethylphosphate. Numbers of early deaths are mean values per pregnant female. Mean (—) and 95 percent confidence interval (----) for linear response of four highest doses of five dosages administered by single intraperitoneal injection. Superimposed points from other experiments showing general agreement. □ Single intraperitoneal injection at two dosages. • Gavage on five successive days at 500 mg/kg each (cumulative dose of 2500 mg/kg).

ance of dose as a function of weeks after TMP administration was performed with matrix inversion techniques.

Trimethylphosphate generally was not toxic at the doses tested. One male that received five oral doses of 1000 mg/kg did die (Table 1). Pregnancy rates in females mated with test and control males did not differ consistently; however, the incidence of pregnancy was generally reduced at the highest total dosages. Weekly means of total implants per pregnant control ranged from 10.5 to 12.6. After injection of 200 and 1000 mg/kg, reduction in numbers of total implants during the first 3 weeks of mating was significant and related to dosage (Table 2); however, when TMP was injected over a wider range of dosages, 500 to 2000 mg/kg, no distinct reductions related to dosage were observed, although lower numbers of total implants were apparent in the 2nd week. After gavage with higher total doses, reduction in numbers of implants in the first 3 weeks of mating was significant and related to dosage (Fig. 1, Table 2); at the highest dosage, implants were also reduced at the 5th week. This is also apparent from the alternate criterion of proportion of females with reduced numbers of total implants (Fig. 1). At the higher total oral doses, losses before implantation were consistent and related to dosage in the first 3 weeks of mating (Fig. 1).

Mean numbers of early fetal deaths per pregnant control ranged from 0 to 0.68 with a mean of 0.33 (Table 1). A highly significant increase in early deaths occurred during the first 3 weeks of mating for all experiments (Table 2). This is manifested as a dosage-dependent increase in early fetal deaths occurring in the second mating week at all dosages, except the highest oral dose where effects were noted earlier (Fig. 1); absence of early fetal deaths at the 2nd week of mating for the latter dosage probably reflected reduced pregnancies (Table 1) and losses before implantation (Fig. 1). The dose-response regression for early deaths in the second mating week was linear over the four highest doses injected intraperitoneally, with a slope of 1.2 (± 0.1 early deaths per 500 mg); the effects of TMP appear cumulative and depend on total dose administered irrespective of route (Fig. 2).

In these experiments, single parenteral and repeated oral administration of less than toxic concentrations of TMP to male mice produced both antifertility and mutagenicity dependent on dosage. Antifertility effects, as manifested by reduced pregnancies, were less clearly defined and evident only at highest total dose. Mutagenic effects, as manifested by increased numbers of early fetal deaths and by losses before implantation, were restricted to matings during postmeiotic stages of spermatogenesis. As previously reported for mice treated with the mutagens tris(1-aziridinyl)phosphine oxide (TEPA) and tris(2-methyl-1-aziridinyl)phosphine oxide (METEPA), the time and dose-response relations for both losses before implantation and for increased numbers of early fetal deaths are similar. At high dosages, early deaths occurred in females with reduced number of total implants and in those with normal numbers of implants (*9*). Our results indicate a higher degree of mutagenic sensitivity of mice to TMP than suggested by previous data (*6*); in both studies, however, TMP appeared equally active after oral or parenteral administration.

Evaluation of potential human genetic hazards due to TMP requires data, presently generally unavailable, on precise conditions of its industrial use, including concentration in fuels. Additional information is also required on the concentration of unreacted TMP, and of any biologically active pyrolysis products, in automobile exhaust.

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10. Supported by NIH grants C-6516 and FR-05526 and by National Air Pollution Control Administration contract PH-86-66-169.

2 March 1970

Aerial Vision: Unique Adaptation in an Intertidal Fish

Abstract. Mnierpes macrocephalus, *a clinid fish of rocky shores of the eastern tropical Pacific, makes frequent terrestrial sojourns. The normal fish eye is myopic in air because of curvature of the cornea. This is overcome in* Mnierpes *by the presence of two flattened corneal surfaces.*

In aquatic vision, fishes rely solely on the movement of the round crystalline lens for visual accommodation. The cornea does not function in image formation because its refractive index is the same as that of water. In air, fish vision becomes myopic because the refractive power of the curved cornea