BIO-MEDICAL RESEARCH
DOCUMENT DESCRIPTION FORM

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1–20	21–40	41–60	61 62
SHORT, RD	MINOR, JL	WINSTON, JM	11
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A Dominant Lethal Study in Male Rats	22
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Vinylidene Chloride -- Mutagenesis	24

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Brief Summary

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teristic of mercury treated rats. Decreased balancing ability and crossing of hind limbs suggested altered motor coordination. Rats treated with mercury often exhibited decreased body temperature, matting of fur about the eyes and perineal region, apparent vision impairment, decreased body weight, and diarrhea. (Authors' abstract)

Brain and Blood Lead in Acute Intoxication. H. Savolainen and J. Kilpio. *Scand J Work Environ Health* 3:104-197, 1977. Dept. of Industrial Hygiene and Toxicology, Institute of Occupational Health, Haartmaninkatu 1, SF-00290 Helsinki 29, Finland.

Administering lead acetate in drinking water to adult male rates resulted in an elevated lead content in blood and brain during 11 subsequent days. The brain and blood lead contents were proportional to each other although the interdependency changed according to the cumulative dose and equilibration period. The present data indicate that the permeability of the blood-brain barrier towards inorganic lead may be dose dependent and saturable with high doses of the metal. (Authors' abstract)

Effect of Butylated Hydroxytoluene and Other Antioxidants on Mouse Lung Metabolism. S. T. Omaye, K. A. Reddy, and C. E. Cross. *J Toxicol Environ Health* 3:829-836, 1977. California Primate Research Center, University of California, Davis, Calif.

Toxic doses of butylated hydroxytoluene (BHT), a phenolic antioxidant commonly used as a food additive, are known to produce lung damage. In this study, three days after a single ip injection of 62.5, 215, or 500 mg/kg BHT in mice there was a dose dependent increase in lung weight. This concentration dependence with injected BHT was accompanied by increases in lung DNA and nonprotein sulfhydryl levels and in whole lung tissue enzyme activities of glutathione (GSH) peroxidase, GSH reductase, glucose-6-phosphate dehydrogenase, and superoxide dismutase. The increased enzyme activities are considered to correspond to inflammatory and proliferative pulmonary changes resulting from acute lung cell injury and necrosis, which have been described previously, and cannot be construed as evidence for a primary oxidant induced pulmonary lesion. The mechanism of BHT induced lung changes may not be related to the antioxidant property of BHT, since vitamin E, n-propyl gallate, ethoxyquin, N,N'diphenyl-p-phenylenediamine, and the structurally similar compound, butylated hydroxyanisole did not appear to produce the gross anatomical or biochemical lung changes observed with BHT. (Authors' abstract)

A Dominant Lethal Study in Male Rats After Repeated Exposures to Vinyl Chloride or Vinylidene Chloride. R. D. Short, J. L. Minor, J. M. Winston, and C. C. Lee. *J Toxicol Environ Health* 3:965-968, 1977. Pharmacology and Toxicology, Midwest Research Institute, Kansas City, Mo.

Male CD rats were exposed six hours/day for five days/week to 0, 50, 250, or 1,000 ppm of vinyl chloride or 55 ppm of vinylidene chloride. Starting on week 11 of exposure, these males were mated with untreated females. There was no evidence of either preimplantation loss or postimplantation loss in pregnant females that resulted from these matings. Consequently, it was concluded that these exposures did not produce germinal mutation, as manifested by a dominant lethal effect, in male rats. (Authors' abstract)

Skin Tumor-Initiating Activities of the Twelve Isomeric Phenols of Benzo(a)pyrene. T. J. Slaga, W. M. Bracken, S. Dresner, et al. *Cancer Res* 38:678-681, 1978. Biology Division, Oak Ridge National Laboratory, Oak Ridge, TN 37830.

The skin tumor initiating activities of the 12 isomeric phenols of benzo(a)pyrene (BP) were determined in mice by use of a two-stage system of tumorigenesis. 11-Hydroxybenzo(a)pyrene was moderately active, whereas 2-hydroxybenzo(a)pyrene and BP were strong tumor initiators when applied topically to CD-1 mice and followed by twice-weekly applications of the promoter 12-0-tetradecanoylphorbol-13-acetate. 1-, 3-, 4-, 5-, 6-, 7-, 8-, 9-, 10-, and 12-hydroxybenzo(a)pyrene had less than 5% of the tumor initiating activity of BP when the data were expressed as papillomas per mouse. After 30 weeks of promotion, the number of papillomas per mouse was 8.4, 8.5 and 2.8, respectively, for the animals treated with BP, 2-hydroxybenzo(a)pyrene, and 11-hydroxybenzo(a)pyrene. A five-week latency period before the appearance of the first tumor was observed after the application of either 2-hydroxybenzo(a)pyrene or BP, whereas a slightly longer latency period of seven weeks was observed following application of 11-hydroxybenzo(a)pyrene. The time required for 50% of the animals to develop tumors was 13 weeks for animals treated with BP and 15 weeks for animals treated with 2- or 11-hydroxybenzo(a)pyrene. (Authors' abstract)

Cellular Attachment to Implanted Foreign Bodies in Relation to Tumorigenesis. D. J. Ferguson. *Cancer Res* 37:4367-4371, 1977. Dept. of Surgery, University of Chicago, Chicago, IL 60637.

Previous experiments demonstrating a reduction of tumorigenicity by roughening the surfaces of plastics implanted in rodents, or by increasing the pore size of cellulose filter implants, were repeated with observations on cellular attachment to these objects and to filters strengthened and made impermeable by bonding to plastic. Round 13 mm discs of methylmethacrylate implanted s.c. in A/BiF/F50+ mice produced sarcomas in 12% of mice at 64 weeks. Tumor incidence increased to 60% ($p < 0.001$) in mice receiving discs to which cellulose filters with pore sizes of 0.025 to 0.1 μm were bonded. No tumors occurred with discs covered by 0.45 μm filters, followed up to 83 weeks. Vinyl coverslips 15 mm square also produced no sarcomas when covered by 0.45 μm filters — plain vinyl produced sarcomas in 40% of mice at 64 weeks ($p < 0.001$). Sanding of vinyl surfaces reduced tumorigenicity ($p < 0.05$). Permeability, fragility, and storage capacity of filters are apparently not related to tumorigenicity. Surface roughness probably is related. Cells, mostly macrophages, were densely and uniformly attached to nontumorigenic surfaces from 24 hours to two years after implantation but were distinctly fewer and not uniformly distributed on tumorigenic surfaces. Topology favoring attachment was inherent in 0.45 μm filters and was produced in plastic by gouging irregular excavations 10 to 15 μm deep. — *R.E.E.*

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Source (Journal, Vol., Number, Pages, Date)

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TOXICOL. APPL. PHARMACOL., 9(3): 501-504,
Nov. 1966

Chronic oral toxicity to rats of a vinyl chloride-vinyl acetate copolymer.

Smyth, H.F., Jr.
Weil, C.S.

Rats Sc
vinyl chloride-vinyl chloride acetate

No toxic effect

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1–20	21–40	41–60	61 62
SOKAL J A	BARINSKI B	BIALEK J	11
KOLECKI R	MATYCH S	OLCZAK G	12
FALUS J	STETKIEWICZ J	STETKIEWICZ I	13

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Title of Report; end with space-hyphen-hyphen-space. Follow with Index Terms, separated from each other with comma-space. Avoid other punctuation; do not abbreviate.

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Experimental ~~Results~~ Studies on the Chronic	22
Toxic Effects of Vinyl Chloride in Rats --	23
DUBROVNIK	24

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EXPERIMENTAL STUDIES ON THE CHRONIC TOXIC EFFECTS OF VINYL CHLORIDE IN RATS

J. A. SOKAL, B. BARAŃSKI, J. MAJKA, R. ROLECKI, S. MATYCH, G. OLCZAK, J. PALUS, J. STETKIEWICZ, I. STETKIEWICZ and K. WRÓBLEWSKA

Institute of Occupational Medicine, Łódź, Poland

In the experiment, male rats were exposed to vinyl chloride at the concentrations of 50, 500 and 20,000 ppm, 5 hours a day, 5 days a week, during a total period of 10 months. After 1, 3, 6 and 10 months of the exposure, blood and urine analysis as well as histopathological examination of internal organs were performed in exposed and in control rats. Blood analysis included hematology and the following biochemical determinations: total protein, urea, phosphates, citrates, calcium, alkaline phosphatase, alanine and aspartate aminotransferases, γ-glutamyltranspeptydase and lactate dehydrogenase. In addition to the routine urine analysis acid mucopolysaccharides and hydroxyproline were estimated. After 10 months of exposure all rats were sacrificed, organ weights measured and the X-ray examination of the skeleton of some rats was performed.

As a result of chronic exposure to vinyl chloride the following toxic effects were observed in rats:

1. depression in body weight increase
2. increase in the relative weights of liver, kidneys, heart, and spleen
3. biochemical changes in blood (mainly increased activity of plasma lactate dehydrogenase and alkaline phosphatase)
4. slight hematological changes
5. morphological changes of the liver

Even at the concentration of 50 ppm some toxic effects were observed including morphological changes in the liver of some rats.

The results obtained showed that the chronic exposure to 50 ppm of vinyl chloride causes systemic toxic effects in rats.

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