ES-3662 REV. 12-85

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CENTRAL RESEARCH AND DEVELOPMENT DEPARTMENT

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April 10, 1989

CONFIDENTIAL

To: G. J. Patterson
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Lead Naphthenate
Cyclohexanecarboxylic Acid, Lead Salt
CAS Registry No. 50825-29-1

Lead naphthenate has been reviewed according to the February 1988 EQC "Guidelines for Control of Carcinogenic, Reproductive, and Developmental Risks Posed by Chemicals Made or Used within Du Pont."

## General Toxicity

Lead naphthenate (24% Pb) has very low acute oral toxicity with an LD50 in rats of 5100 mg/kg (7). Lead naphthenate is readily absorbed through the skin. A skin-loading experiment was conducted in three male subjects by applying 192 mg of lead naphthenate in 6 mL of an oil. After ten minutes, the lead content of the venous blood increased and reached more than 300% of its initial value 30 to 60 minutes after contact. The lead content of the venous blood returned to normal eight hours later (6). Lead can also be absorbed after an inhalation exposure. Workers exposed daily to an average lead naphthenate concentration of 96 ug/$m^3$ had a blood lead level of 63 ug/100 grams of blood (5).

## Carcinogenic Potential

Renal tumors were observed in mice dermally administered a 20% solution of lead naphthenate in benzene. In a group of 59 mice treated twice a week for up to 569 days, one renal carcinoma, four renal adenomas, and seven benign skin tumors were observed. No control group was included in this study (1,2).

Mutagenic Potential

Lead naphthenate was not mutagenic in the Ames test either in the presence or absence of a S-9 activation system derived from rat or hamster liver (3).

Developmental Toxicity

No information is available.

Reproductive Toxicity

No information is available.

AEL Review

These data were reviewed by the Haskell Laboratory AEL Committee on December 5, 1988.

An AEL of 50 ug/$m^3$ (8-hour TWA), adopted in 1980, was continued. Because of the ability of lead naphthenate to be absorbed through the skin, this limit will have a skin notation.

Also recommended was blood lead monitoring. Blood lead determinations represent the most effective way to monitor an individual for exposure to lead. Blood lead levels exceeding 60-80 ug Pb/100 g of blood indicate overexposure to lead.

A blood lead limit of 50 ug Pb/100 g blood was also continued. If blood lead exceeds this limit, action needs to be taken to determine the source of exposure and to initiate preventive actions. Instructions for determining blood lead concentrations have been prepared by the Medical Division. In these instructions, an airborne lead concentration of 30 ug/$m^3$ (8-hour TWA) is considered an action level. If airborne lead concentrations exceed 30 ug/$m^3$, biological monitoring must be done upon entry into the workplace and then every six months. If the blood lead level is 40 ug Pb/100 g blood or higher, blood leads must be done every two months until two consecutive levels are below 40 ug Pb/100 g blood.

This limit applies only to men, and women not of childbearing capability. For women of childbearing capability, direct exposure to lead should be avoided. However, contact with trace quantities and exposure to airborne lead levels equivalent to normal ambient background levels are permitted. Exposure to background levels correlates with a blood lead level of 30 ug Pb/100 g blood*. This lead level is not believed to be harmful to the fetus (4).

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* Note: This level is currently under review by the AEL Committee.

Summary

Kidney tumors were produced in mice dermally administered lead naphthenate. Because no control group was used, the statistical significance of these tumors cannot be determined. However, as kidney tumors have been produced by other lead compounds, e.g., lead acetate, the kidney tumors in these mice might be compound related. IARC reviewed these data and concluded that "although indicative of a carcinogenic effect of lead naphthenate on the kidney, these results cannot be evaluated, since no control mice were painted with the benzene vehicle alone".

We conclude that lead naphthenate should be considered a weak carcinogen in the mouse, producing kidney tumors. We further conclude that at the recommended AEL of 50 ug/m$^3$ (8-hour TWA), skin, the hazard is not significant.

No information is available on the developmental or reproductive toxicity of lead naphthenate. Lead compounds have been historically handled by Du Pont as developmental and reproductive hazards. As no information is available to show that lead naphthenate should not be included in this group, the control procedure described in Reference 4 (See AEL Review paragraph for details) would apply.

According to the guidelines for carcinogenic, reproductive, and developmental hazard designations, lead naphthenate will be categorized as c 1989, D 1989, and r 1989.

Note: Those receiving copies of this letter, if they have an interest, should coordinate any actions they plan to take with G. J. Patterson of the Automotive and Fabricated Products Department.

References


1. Baldwin, R. W. et al., Br. Cancer Campaign, 39:420 (1962) (J-2494).

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3. Cameron, T. P., NCI, Letter dated 8-23-83 to EPA (Cited in TSCA 8d Lead Naphthenate File, TSCA Fiche OTS 0512232-2 (AEL File 88-197).

4. Du Pont Co., Hazard Determination Letter dated March 23, 1981, Lead Exposure Levels in the Workplace.

5. Goldberg, R. et al., J. Occup. Med., 29(9):750-751 (1987).

References (Cont'd)

6. Rasetti, L. et al., Rass. Med. Indust. Ig. Lav., 30:71-75 (1961) (Cited in Van Peteghem, T. and H. De Vos, Br. J. Ind. Med., 31(3):233-238 (1974)).

7. Rockhold, W. T., Arch. Ind. Health, 12:477-482 (1955).

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April 10, 1989