

FROM:

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As per our 1/10 Conversation:

In May 1989, I copied CFR on our action plan for lead naphthenate. We had been treating Pb naphth as D and r since at least 1981 (organic lead compounds) so there was no change in our historical handling precautions. The only thing new from the Haz Det letter was the small c designation, which has been communicated to APD employees by C. Lucas, as per the attached instructions.

George

N 27662

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May 23, 1989

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HAZARD REVIEW
LEAD NAPHTHENATE

Haskell Lab has completed a Hazard Review of lead naphthenate. Haskell's findings can be summarized as follows:

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/m³ (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 considered by Du Pont as developmental toxins. As there is no information that lead naphthenate should not be included in this group, the control procedures for lead compounds should be in effect. These are as follows:

An AEL of 50 ug/m³ (8-hour TWA), adopted in 1980, has been continued. Because of the ability of lead naphthenate to be absorbed through the skin, this limit will have a skin notation.

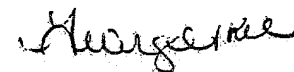
Also recommended was blood lead monitoring. Blood lead determinations represent the most effective way to monitor an individual for exposure to lead. Blood lead levels exceeding 60-80 ug Pb/100 g of blood indicate overexposure to lead.

A blood lead limit of 50 ug Pb/100 g blood was also continued. If blood lead exceeds this limit, actions 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/m³ (8-hour TWA) is considered an action level. If airborne lead concentrations exceed 30 ug/m³, 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 if 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.

In addition, lead compounds have historically been classified as r, or less potent reproductive toxins. This means that although reproductive toxicity occurred, it did so only at or just below dose levels resulting in other signs of toxicity. There is no evidence lead naphthenate should not be considered typical of this group.

In summary, according to the guidelines for carcinogenic, reproductive, and developmental hazard designations, lead naphthenate will be categorized as c 1989, D 1989, and r 1989. Lead naphthenate is already on the Restricted Materials List as a developmental hazard. There need be no change to the list as a result of these findings. Please advise employees of these conclusions.


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GJP/kle