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Subject Environmental Defense comments on Dichloroacetyl Chloride (CAS# 79-36-7)

(Submitted via Internet 2/11/05 to oppt.ncic@epa.gov, hpv.chemrtk@epa.gov, boswell.karen@epa.gov, chem.rtk@epa.gov, lucierg@msn.com and Edwin.L.Mongan-1@usa.dupont.com)

Environmental Defense appreciates this opportunity to submit comments on the robust summary/test plan for **Dichloroacetyl Chloride (CAS# 79-36-7)**.

The test plan and robust summaries for dichloroacetyl chloride (DCAC) were submitted by E. I. du Pont de Nemours and Company. In general, the submission was informative and written in an objective manner. However, no information was provided on the applications and uses of DCAC, so it is impossible to evaluate the potential for worker exposures, environmental releases and human exposures from the general environment or from consumer products.

The sponsor contends that existing data are adequate to meet all SIDS endpoints. This contention relies heavily on the use of surrogate data from dichloroacetic acid (DCA), which is the presumed hydrolytic product of DCAC. The test plan states that the half life of DCAC in water is less than one second and the expected products are DCAC and hydrochloric acid. While we expect this is true, we recommend that experimental data be obtained to unequivocally demonstrate that DCAC is converted entirely to DCA when in contact with water. This is important, as the foundation of the test plan for DCAC rests on the assumption of rapid and quantitative formation of DCA.

In regards to the other endpoints, we agree that they are addressed adequately by existing data with the exception that a reproductive toxicity study needs to be conducted because a study in male rats indicates that DCA is a testicular toxin and no data are available to assess the reproductive toxicity in female rodents. The reproductive toxicity study should use rats exposed to DCAC via oral exposures. The robust summaries indicate that severe testicular lesions were caused in rats by oral exposures to DCA but inhalation exposures to DCAC did not cause these lesions. If DCAC is entirely converted to DCA in the body, why didn't DCAC cause the same testicular lesions? Are there pharmacokinetic data available from inhalation and oral exposures to explain this apparent discrepancy?

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The existing repeat dose studies were conducted via multiple routes of exposure in more than one species and there are two cancer bioassays described in the robust summaries. Taken together, these studies are more than adequate to address the repeat dose endpoint. These studies indicate that DCAC and DCA are toxic chemicals, as nasal tumors were observed in rats following inhalation doses of 2 ppm DCAC and liver tumors occurred in rats following DCA exposure. Studies in dogs and rats indicate that DCA is toxic to the brain, lung, testes and prostate at doses as low as 12.5 mg/kg/day for 30 days. A NOAEL was not achieved in most of these studies. The testicular lesions were severe and widespread. Unfortunately, these studies, in most cases, did not use female animals. Given its toxicity, we hope that workers using or producing DCAC are fully protected from exposure and the workplace exposure limit is sufficiently protective.

Both DCA and DCAC are positive in *in vitro* and *in vivo* tests for genetic toxicity. Are data available regarding the mechanism of mutagenicity?

Thank you for this opportunity to comment.

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