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Document Title	INITIAL SUBMISSION: LETTER FROM DUPONT TO USEPA W/SUMMARY OF A 2-WK INHALATION STUDY OF PERFLUOROACETYL FLUORIDE IN MALE RATS, DATED 091300		
Chemical Category	PERFLUOROACETYL FLUORIDE		

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DuPont Haskell Laboratory
for Toxicology and Industrial Medicine
Elkton Road, P.O. Box 50
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September 13, 2000

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Attention: 8(e) Coordinator
Office of Pollution Prevention and Toxics
U.S. Environmental Protection Agency
401 M Street SW
Washington, DC 20460-0001

MR 3-356

Dear 8(e) Coordinator:



Perfluoroacetyl Fluoride (PAF)
CAS# 354-34-7

8800000220

This letter is to inform you of the results of a 2-week inhalation study in male rats with the above referenced substance.

Four groups of ten male rats each were exposed six hours/day, four to five days/week, for a total of nine exposures. On the day following the last exposure, blood and urine samples were collected for clinical analysis, and 5 rats per group were sacrificed for anatomic pathology. All remaining rats were allowed to recover for a 14-day period. At the end of the recovery period, blood and urine samples were collected for clinical pathology analysis, and all surviving rats were sacrificed for anatomic pathology.

The targeted concentrations for the study were 0, 1, 10 and 40 ppm. The analytically determined mean concentrations of PAF were 0, 1.4, 10, and 36 ppm, respectively. All animals in the high dose group (40 ppm) were sacrificed the day after their sixth exposure due to the severity of their clinical signs of toxicity (irregular respiration and lung noise) and severe body weight losses. No clinical pathology samples were collected for the 40 ppm group. The irregular respiration in the high level group was observed during each of their six exposures. Irregular respiration was observed in several of the intermediate level animals during exposures six through nine. Animals in low level group appeared normal throughout all nine exposures.

Statistically significant increased lung absolute weight, % of body weight, and brain weight ration were present at sacrifice in the 10 ppm group. The weight increase correlated microscopically with inflammation, bronchiolar hyperplasia, and perivascular/peribronchiolar edema and was considered compound-related. Gross pathological observations considered compound-related were lung discoloration and large mediastinal lymph nodes at 10 and 40 ppm. Microscopic findings included changes in the lungs at 1 and 10 ppm the day after the ninth exposure (test day 11) and in the 40 ppm group on test day 8 (high group sacrificed after the sixth exposure). Subacute to chronic centriacinar inflammation and bronchioalveolar hyperplasia were present in 4/5, 5/5, and 10/10 rats at 1, 10, and 40 ppm, respectively. Perivascular and peribronchiolar edema was present in all rats at 10 and 40 ppm. The severity of inflammation and hyperplasia was minimal at the 1 ppm level and mild to severe at 10 and 40 ppm.

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In 14-day recovery rats, similar lung lesions, though not as severe, were present in all 10 ppm rats. Minimal subacute/chronic centriacinar inflammation was present in 2/5 recovery rats at 1 ppm. These findings appear to be reversible with time.

Under these experimental conditions, the findings described above appear to be reportable, based upon EPA guidance regarding the reportability of such data under TSCA Section 8(e) criteria.

Sincerely,

A handwritten signature in black ink that reads "A. Michael Kaplan". The signature is written in a cursive style and is followed by a horizontal line.

A. Michael Kaplan, Ph.D.
Director - Regulatory Affairs

AMK/RS:clp
(302)366-5260

CERTIFICATE OF AUTHENTICITY

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