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8EHQ-06-16452

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March 10, 2011

VIA FEDERAL EXPRESS

Attn: TSCA Declassification Coordinator
U.S. Environmental Protection Agency
Office of Pollution Prevention and Toxics
Document Control Office (7407M)
Washington, D.C. 20460

Public Copy

Re: Declassification Activity - TSCA §8(e) Submission
Originally Assigned 8EHQ Number: 8EHQ-06-16452 (letter dated 05.04.06)
Originally Assigned Bar Code: 88060000224
CAS number: 2923-26-4
Supplemental Submission - Revised Public Copy of Submission

Dear TSCA Declassification Coordinator:

This submission is made in connection with the EPA 2010 CBI Declassification Challenge initiative.

Please find enclosed a revised public copy of the above-identified submission. Any information still claimed as confidential business information (CBI) in the attached report has been redacted and replaced by brackets. The originally assigned 8EHQ number has been added by the submitter to the first page of the enclosed revised public copy of the submission.

Very truly yours,

Enclosure



PUBLIC COPY

May 4, 2006

Via Federal Express

Document Processing Center (Mail Code 7407M)
Room 6428
Attention: 8(e) Coordinator
Office of Pollution Prevention and Toxics
U.S. Environmental Protection Agency
1201 Constitution Ave., NW
Washington, D.C. 20460

Dear 8(e) Coordinator:

Sodium Perfluorohexanoate
CAS# 2923-26-4
Generic Name: Perfluorocarboxylate salt

This letter is to inform you of the results of two recently conducted two-week oral gavage range-finding studies to set dose levels for a 90-day study with the above-referenced test substance.

In the first study, male and female CrI:CD(SD) rats were dosed daily with 0, 5, 10, 30, or 50 mg/kg/day by oral gavage. There were no in-life, clinical pathology, or anatomical pathology findings in the study. In a second study, male CrI:CD(SD) rats (5/dose level) received a daily dose of 0, 500 or 1000 mg/kg/day for two weeks via oral gavage. The 0 ppm group received water. At sacrifice, the animals were weighed, had their livers removed and weighed, and underwent a gross pathology exam of the stomach only. Blood was collected via the vena cava for clinical pathology. Clinical observations were determined weekly and body weight was measured daily throughout the study. Clinical pathology evaluations (hematology and clinical chemistry) were performed on fasted rats at terminal sacrifice. No histopathology was performed in this study.

Test substance-related effects on hematology and clinical chemistry occurred in rats dosed at 500 and 1000 mg/kg/day. In addition, an increase in absolute and relative liver weights were observed in rats receiving 500 and 1000 mg/kg/day test substance. The hematology effects were minimal (500 mg/kg/day) to moderate (1000 mg/kg/day) decreases in red cell mass parameters (red cell count, hemoglobin, and hematocrit) associated with accelerated red blood cell production, as indicated by increased reticulocytes. A decrease in red cell mass parameters was likely due to premature red cell destruction (hemolysis). Increased platelet counts, secondary to accelerated red blood cell production was observed in animals receiving 1000 mg/kg/day. Clinical chemistry measurements indicated a mild increase in aspartate aminotransferase and alanine aminotransferase activities (1000 mg/kg/day). In addition, mildly increased alkaline phosphatase activity was observed in 2/5 rats at 1000 mg/kg/day. Rats dosed with 500 and 1000 mg/kg/day test substance exhibited mildly decreased cholesterol and triglycerides and decreases in total protein due to decreases in globulin. The magnitude of the protein effects was minimal at 500 mg/kg/day and mild in animals receiving 1000 mg/kg/day.

Under these experimental conditions, the findings described above are being reported in accordance with the guidance given in the EPA TSCA Section 8(e) Reporting Guide (June 1991).

Sincerely,