From: US112388--VM01 Date and time 10/12/95 03:52:07
To: US065823--USSP01 BUTENHOFF, JOHN US088010--USSP01 ZIMMERMAN, DALLAS
US265515--ALLIN1 ZOBEL, LARRY R. M US312588--ALLIN1 MANDEL, JEFF
BE100008--DIEVMB COX, BOB

FC 1225/-4
FC-143

From: Roger G. Perkins 3M 612/733-3222
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Subject: APME Meeting

The October 10, 1995 meeting of the APME Toxicology Committee was heldat the APME offices in Brussels; G. Malvinerno (Ausimont), R. Jung (Hoechst), G. Kennedy (duPont), M. Mistrorigo (Miteni), C. Elcombe (ICI), D. Farrar(ICI), Bob Cox (3M) and this writer were present.

ICI toxicologists are very strongly espousing the position that APFO is an animal carcinogen based on the tumors in the liver, pancreas and testis seen in the duPont study in male rats only at a dose of 300 ppm in the diet. They and others present contend that the benign tumors are simply early lesions on a continuum that ultimately leads to malignant tumors. (I do not agree with their interpretation and stated this.)

Most of the other issues discussed hinge in part on this first issue.

They are still very strongly in favor of 3M publishing data in a peer reviewed journal that would put the Gilleland October 1993 publication into the proper perspective. They would like for 3M to organize an effort to get the epidemiologists and physicians from the various companies in contact with each other.

We spent a considerable time discussing the in-vitro testing for genotoxicity. I explained that 3M testing of FC-143 was delayed because of prioritization in a larger toxicity testing plan.

The occurence of peroxisome proliferation was discussed in terms of the liver tumors and the relevance to man. There is some puzzlement about the mechanism for occurence of the acinar tumors, but Cliff Elcombe did remind us that the acinar cells and hepatocytes have a common embryonic origin and that some tumors in the acinar cells have an appearance very similar to hepatic cell tumors. The topic of APFO as an endocrine disruptor was raised and the statement made that the committee or producers need to develop a position paper on this issue.

There was discussion of mechanistic considerations related to tumor formation. This included a proposal that Elcombe outline an approach to look at acinar cells in culture as regards APFO effects on cell proliferation, etc.

I mentioned our study in progress of rat and human liver in culture and responses to several FC. There was interest in whether peroxisome proliferation would be studied.

Those persons representing APFO users are very anxious for the APFO manufacturers to make a decision on EU labeling of APFO and specifically are speculating whether risk phrase R40 applies.

One action point is to get the 3M environmental data and duPont environmental data collected into a single review to determine if data gaps exist.

The next meeting is proposed to occur during the week of January 15, 1996; location possibly Rome or Brussels.

since the meeting Bob Cox and I have discussed the meeting. It appears that 3M might benefit in using the ECETOX forum to establish a group whose goal would be the preparation of either a technical report or criteria document on perfluorooctanoic acid and its salts and related compounds in which current toxicology and possibly ecotoxicology data would be summarized. It appears that such a paper is the goal of the APME toxicology committee, but that with so many other issues on their agenda that some other route to this goal may be faster.