COURTNEY M. PRICE
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American Chemistry Council *Good Chemistry Makes It Possible*

March 24, 2003

Dr. C.W. Jameson
National Toxicology Program
Report on Carcinogen
MD EC-14
P.O. Box 12233
Research Triangle Park, NC 27709

Dear Dr. Jameson:

Enclosed please find comments regarding the nomination of diethanolamine (DEA) for listing in the NTP *Report on Carcinogens*, Eleventh Edition. The comments are submitted on behalf of the Alkanolamines Panel of the American Chemistry Council.

For reasons discussed in the comments, the Panel believes that the available studies and data do not establish that DEA is "reasonably anticipated to be a human carcinogen" and therefore establish that NTP should accept the recommendations of RG1, RG2, and the NTP *RoC* Subcommittee and determine that listing of DEA in the *RoC* would not be appropriate.

If you have any questions, please contact Jon Busch of my staff at (703) 741-5633.

Sincerely yours,

BEFORE THE DEPARTMENT OF HEALTH AND HUMAN SERVICES
NATIONAL TOXICOLOGY PROGRAM

COMMENTS OF THE ALKANOLAMINES PANEL OF THE AMERICAN CHEMISTRY COUNCIL IN RESPONSE TO NTP'S CALL FOR FINAL PUBLIC COMMENT ON THE NOMINATION FOR LISTING DIETHANOLAMINE IN THE *REPORT ON CARCINOGENS*

National Toxicology Program,	)
Call for Public Comments on	)
10 Nominations, Proposed for Listing	)
in the *Report on Carcinogens*, Eleventh Edition	)

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March 24, 2003

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EXECUTIVE SUMMARY

The Alkanolamines Panel (Panel) of the American Chemistry Council submits these comments in response to the National Toxicology Program's (NTP) Call for Public Comments on 10 Nominations, Proposed for Listing in the *Report on Carcinogens,* Eleventh Edition. 68 Fed. Reg. 3033 (Jan. 22, 2003). The Panel members include major manufacturers of alkanolamines, including producers of DEA.

The Panel strongly supports the recommendations made by all three scientific peer review committees -- RG1, RG2, and, most recently, the NTP RoC Subcommittee -- not to list DEA in the *RoC* and urges NTP to adopt those recommendations. The Panel believes that all three committees have made the correct determination based on the scientific data and NTP's criteria for listing. The Panel is pleased that all three scientific peer review committees have found that the relevant data do not support the contention of the United Auto Workers (UAW) that DEA should be listed.

The Panel incorporates by reference: (1) its September 24, 2001, comments to NTP (Panel's Initial Comments), in response to NTP's initial request for comments on the nomination of DEA for possible listing in the *RoC*; (2) its December 21, 2001, letter and appended study; and (3) CTFA's September 17, 2001, comments.

The available scientific data clearly support the NTP Subcommittee recommendations not to list DEA in the *RoC* under NTP's criteria.

- There exists neither sufficient, nor limited evidence, from studies in humans which indicates that DEA causes cancer, or that shows DEA is known or is reasonably anticipated to be a human carcinogen under NTP's listing criteria.

  - The Panel concurs with the following conclusion in the RG2 Review Summary for DEA with regard to the epidemiological literature: "There are no human studies reported in which exposure solely to DEA is specifically mentioned. The human cancer studies on metalworking fluids are not relevant for the evaluation of the carcinogenicity of DEA because the effects of DEA cannot be separated from the other components of metalworking fluids."

  - UAW's September 4, 2002, comments and the epidemiological studies submitted to NTP shortly before the NTP RoC Subcommittee meeting do not provide any new significant and relevant information.

- The available experimental animal data do not meet NTP's listing criteria.

- Other data corroborate that DEA does not meet the listing criteria.

- International and U.S. authorities have determined that DEA should not be classified as presenting a carcinogenic risk to humans, thus corroborating the findings of RG1, RG2, and the NTP RoC Subcommittee that DEA should not be listed.

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INTRODUCTION

The Alkanolamines Panel (Panel) of the American Chemistry Council submits these comments in response to the National Toxicology Program's (NTP) Call for Public Comments on 10 Nominations, Proposed for Listing in the *Report on Carcinogens,* Eleventh Edition. 68 Fed. Reg. 3033 (Jan. 22, 2003). The Panel members include major manufacturers of alkanolamines, including producers of DEA.[1]

The Panel strongly supports the recommendations made by all three scientific peer review committees -- RG1, RG2, and, most recently, the NTP RoC Subcommittee -- not to list DEA in the *RoC* and urges NTP to adopt those recommendations.[2] The Panel believes that all three committees have made the correct determination based on the scientific data and NTP's criteria for listing. The Panel incorporates by this reference: (1) its September 24, 2001, comments to NTP (Panel's Initial Comments), in response to NTP initial request for comments on the nomination of DEA for possible listing in the *RoC*;[3] (2) its December 21, 2001, letter and appended study;[4] and (3) CTFA's September 17, 2001, comments.[5]

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1 Panel member companies are: BASF Corporation, The Dow Chemical Company, Equistar Chemical, L.P., Huntsman Corporation, and Ineos, L.L.C.

2 The votes of the RG1, RG2, and the NTP RoC Subcommittee against listing DEA in the *RoC* were by margins of 7/2, 9/0, and 8/1, respectively. 68 Fed. Reg. 3033, 3035 (Jan. 22, 2003).

3 Comments of the Alkanolamines Panel of the American Chemistry Council in Response to NTP's Request for Comments on the Nomination of Diethanolamine for Possible Listing in the *Report on Carcinogens* (Sept. 24, 2001).

4 Letter from Jonathon T. Busch to Dr. C.W. Jameson (Dec. 21, 2001) (appending Lehman-McKeeman, L.D., E.A. Gamsky, S.M. Hicks, J.D. Vassallo, M. Mar, and S.H.

The Panel agrees with and believes the evidence strongly supports the conclusion of all three scientific peer review committees that the relevant data do not support the contention of the United Auto Workers (UAW) that DEA should be listed in the *RoC*. The UAW nominated DEA for listing based on an NTP Technical Report[6] on the results of a two-year dermal bioassay on $B6C3F_1$ mice, which reported "clear evidence of carcinogenic activity of DEA in male and female mice."[7]

The following discussion summarizes the data that support the recommendations of the three scientific peer review committees. The discussion also responds to some of the erroneous comments that were submitted to NTP by the UAW on September 5, 2002, and addresses certain studies submitted by the UAW to NTP shortly before the November 19-20, 2002, meeting of the NTP RoC Subcommittee.[8]

Zeisel, "Diethanolamine Induces Hepatic Choline Deficiency in Mice." *Toxicological Sciences* (in press).

5 Letter from Gerald N. McEwen, Jr., Ph.D., J.D. to Dr. C.W. Jameson, regarding "Substances Under Review for Possible Listing in the Report on Carcinogens, Eleventh Edition (66 Federal Register 38430): Diethanolamine" (Sept. 17, 2001).

6 66 Fed. Reg. 38430 (July 24, 2001).

7 NTP (1999). Toxicology and Carcinogenesis Studies of Diethanolamine (CAS No. 111-42-2) in F344/N Rats and $B6C3F_1$ Mice (Dermal Studies). NTP TR 478, NIH Publication No. 99-3968.

8 DEA was considered at the NTP RoC Subcommittee meeting on November 19, 2002.

DISCUSSION

I. THE AVAILABLE SCIENTIFIC DATA CLEARLY DEMONSTRATE THAT DEA CANNOT BE LISTED IN THE *RoC* UNDER NTP's CRITERIA

Chemicals may be listed in the *RoC* if they are determined to be "known to be human carcinogens" or "reasonably anticipated to be human carcinogens"[9] within the meaning of NTP's listing criteria. For a chemical to be listed based on human evidence, studies in humans must indicate either: (1) there is sufficient evidence of carcinogenicity from studies in humans which indicates a causal relationship between exposure to the agent, substance, or mixture and human cancer ("known to be human carcinogen"); or (2) there is limited evidence of carcinogenicity from studies in humans which indicates that causal interpretation is credible, but that alternative explanations, such as chance, bias, or confounding factors, could not adequately be excluded ("reasonably anticipated to be human carcinogen").[10] For a chemical to be listed based on experimental animal data, there must be sufficient evidence of carcinogenicity from studies in experimental animals which indicates there is an increased incidence of malignant and/or a combination of malignant and benign tumors ("reasonably anticipated to be human carcinogen"): (1) in multiple species or at multiple tissue sites; or (2) by multiple routes of exposure; or (3) to an unusual degree with regard to incidence, site, or type of tumor or age at

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9 61 Fed. Reg. 50499-50500 (Sept. 26, 1996).

10 NTP, *10th Report on Carcinogens* at Introduction ("Listing Criteria" section), available at http://ehp.niehs.nih.gov/roc/tenth/intro.pdf. DEA has not been nominated based on human studies, and as discussed below, there are insufficient human data to raise an issue as to whether DEA may be listed based on human studies.

onset.[11] The listing criteria also indicate that other considerations may be taken into account, such as data indicating that the agent in question acts through mechanisms which do not operate in humans.

The available relevant data demonstrate that DEA does not meet any of these criteria for listing in the *RoC*.

A. There Exists Neither Sufficient, Nor Limited Evidence, from Studies in Humans Which Indicates That DEA Causes Cancer, or That Shows DEA Is Known or Is Reasonably Anticipated To Be a Human Carcinogen under NTP's Listing Criteria

There exists neither sufficient, nor limited evidence, from studies in humans which indicates that DEA causes cancer. The Panel concurs with the following conclusion in the RG2 Review Summary for DEA with regard to the epidemiological literature: "There are no human studies reported in which exposure solely to DEA is specifically mentioned. The human cancer studies on metalworking fluids are not relevant for the evaluation of the carcinogenicity of DEA because the effects of DEA cannot be separated from the other components of metalworking fluids."[12]

Although the NTP RoC Subcommittee's review summary has not yet been posted by NTP, by recommending against the listing of DEA, at least eight of the nine members of the

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11 *Id.*

Subcommittee made clear that they considered the available epidemiological literature of workers exposed to metalworking fluids to provide insufficient evidence that DEA causes cancer in humans. For example, in discussing the epidemiological data, Subcommittee members stated that any association of cancer with exposure to metalworking fluids cannot be attributed to DEA because the fluids were mixtures of many chemicals, including at least one known hepatocarcinogen.[13]

UAW's September 4, 2002, comments to NTP[14] provide no evidence that DEA causes cancer in humans. UAW's comments refer to two "recently published mortality studies identifying excess mortality from liver cancer among workers likely exposed to [DEA] through metal working fluids" that UAW maintains require further consideration.[15] UAW's comments acknowledge that because the metalworking fluids in these and other studies contain "at least five competing carcinogens," "the evidence that exposure to [DEA] is 'known' to be

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12 NTP, *Review Summary of the NTP Executive Committee Working Group for the Report on Carcinogens (RG2)*, at 1, available at http://ntp-server.niehs.nih.gov/newhomeroc/roc11/DEARG2RevSumm.pdf.

13 NIEHS, NTP, *Condensed Transcript of the Board of Scientific Counselors Report on Carcinogens (RoC) Subcommittee Meeting* (Nov. 19, 2002) at 225, 228, and 230.

14 Letter from Franklin E. Mirer, Ph.D., Director, UAW Health and Safety Department, to Dr. C.W. Jameson, National Toxicology Program, regarding "UAW Comments in Support of Listing Diethanolamine As 'Reasonably Anticipated To Be Carcinogenic to Humans.'" (Sept. 5, 2002) (Transmittal Letter) (appending UAW's comments dated September 4, 2002).

15 September 5, 2002, UAW Transmittal Letter.

carcinogenic to humans is limited."[16] UAW nevertheless suggests that these studies should be "given some weight in the rating of [DEA]."[17]

UAW's comments do not provide any new significant and relevant information.[18] UAW states that of the two "additional" worker studies which it claims show an increase in liver cancer mortality among workers exposed to metalworking fluids, the Eisen, *et al.* (2001) follow-up study is the strongest. This study is discussed, along with a number of other epidemiological studies, in the Draft Background Document prepared by NTP, and therefore was considered by RG1, RG2, and the NTP RoC Subcommittee. The updated NCI cohort, which is the Kazerouni, *et al.* (2000) study referenced in UAW's comments, is not cited in the Draft Background Document, and it is not clear whether RG1 and RG2 considered it. The NTP RoC Subcommittee, however, was presented with comments on that study by UAW[19] and heard and considered testimony on behalf of the Panel critiquing the study.[20]

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16 September 4, 2002, UAW Comments at 8.

17 *Id.* at 10.

18 In addition to discussing epidemiological data, the UAW September 4, 2002, comments, at 7, also refer to the "recent" toxicology studies on mice (Al-Humadi *et al.*, 2000), which reported liver toxicity and adverse male reproductive effects from dermal exposure to metalworking fluids. (This is the same study submitted by UAW as an attachment to a letter to NTP dated November 7, 2002.) These findings, in an inappropriate model for human dermal absorption of chemicals, provide no convincing information pertinent to NTP's criteria for listing DEA. Liver toxicity alone does not constitute evidence of carcinogenicity.

19 *See* September 4, 2002, UAW Comments at 6-7.

20 *See* November 19, 2002, Transcript at 218-219 (Testimony by Dr. William Stott, on behalf of the Panel (misspelled as Dr. Stock in transcript).

The Eisen, *et al.* (2001) study and other epidemiological studies of workers exposed to metalworking fluids do not justify any conclusions regarding DEA's potential carcinogenicity because each has substantial weaknesses such as, in particular, confounding due to exposure to a number of other potential carcinogens found in metalworking fluids. The "other recently published" mortality study (presumably Kazerouni *et al.*, 2000) discussed in UAW's comments suffers from at least the same deficiencies and in fact, as admitted by UAW, "may be among the weaker studies."[21] Contrary to UAW's suggestion, the findings of the Eisen, *et al.* (2001) study and the NCI cohort study (Kazerouni *et al.*, 2000), as well as the other epidemiological studies referenced, are so seriously confounded by exposure to multiple carcinogens and a variety of other factors, that those studies cannot be considered to provide "limited evidence" of carcinogenicity indicating that a "causal interpretation [with regard to DEA] is credible" under the NTP *RoC* criteria. In particular, the Eisen, *et al.* (2001) study did not measure alcohol or hepatitis incidence, major risk factors for liver cancer, or noncancer deaths such as liver disease. The RRs measured in the study did not demonstrate dose-response with soluble nor synthetic metalworking fluid exposure (mg/m^3-years). Indeed, RRs for over 2 mg/m^3-years exposures were less than 1.00. Finally, differences in coding used for cause of death over time led to the listing of tumors of biliary tract (gall bladder, extrahepatic bile duct, ampulla of Vater) as liver cancer. Analysis of the latter has suggested that biliary cancer rather than liver cancer was elevated in those study groups having an increased RR.

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[21] September 4, 2002, UAW Comments at 6.

The Kazerouni, *et al.* (2000) study added 13 additional years to the previous cohort study reported by Decoufle (1978).[22] No distinction between metalworking fluids, and thus potential exposure to alkanolamines and other materials unique to semi- and synthetic fluids, was attempted (grouped as "oil mists"). The only increased SMR was limited to short-term workers. In contrast, men with heavy exposure to oil mists for five or more years had no increased risk of liver cancer (SMR = 0.82) indicating a lack of dose-response and suggests a nonoccupational cause for the short-term SMR. Accordingly, the epidemiological studies on workers exposed to metalworking fluids can be afforded no weight in evaluating the carcinogenicity of DEA.

B. The Available Experimental Animal Data Do Not Meet NTP's Listing Criteria

The available experimental animal data do not provide any probative evidence that DEA induces malignant or a combination of malignant and benign tumors in multiple species or at multiple tissue sites, by multiple routes of exposure, or to an unusual degree. Accordingly, the animal data no not satisfy the criteria for listing in the *RoC*. This conclusion is established by the following considerations:

- Dermal administration of DEA was not carcinogenic in the NTP dermal bioassay on rats.
- While the NTP report of the two-year dermal bioassay in $B6C3F_1$ mice reported a significant increase in benign and malignant liver tumors in

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22 The previous cohort study was reported by: Decoufle, P. (1978). "Further analysis of cancer mortality patterns among workers exposed to cutting oil mists." *J. Natl. Cancer Inst.* 61:1025-1030.

both male and female mice,[23] as concluded by RG2 in its Review Summary, the study reported a significant increase only of renal adenomas (in male mice). No significant increase in renal tubule carcinomas was observed. Accordingly, the renal tumors did not represent a second tumor site within the meaning of NTP's listing criteria.[24]

- The hepatocellular tumors cannot be considered to have been increased to an unusual degree for the reasons stated in the Panel's Initial Comments.[25] Among other items, as demonstrated in the Panel's Initial Comments, an unusual degree finding cannot be made because: (1) the incidence of liver tumors in the vehicle (*i.e.*, ethanol-treated) control female and male mice in the NTP DEA study was unusually high, *i.e.*, the "baseline" incidence of liver tumors in mice not treated with DEA was high; (2) experts on rodent liver carcinogenesis have long accepted that liver tumors in rodents develop in a morphologic sequence of lesions along a continuum with liver adenomas preceding and being able to progress into carcinogens and hepatoblastomas; (3) the $B6C3F_1$ mouse has an unusually high susceptibility to chemical-induced tumor formation; and (4) the ethanol vehicle used in the NTP study for administering DEA is a confounding factor in interpreting the results for the reasons given in the Panel's Initial Comments.[26] Dr. Allan Smith, a member of the RoC Subcommittee, dismissed the liver tumors as having been induced to an unusual degree on

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23 As discussed in detail in the Panel's Initial Comments, the Panel believes that the NTP mouse study has a number of technical limitations that preclude its use for evaluating whether DEA should be listed. *See* Panel's Initial Comments at ii and 12-17.

24 The RG1 Summary also concluded that renal tubule carcinomas were not significantly increased and noted that a majority of the RG1 members found that the "carcinogenicity data was a single tumor type (liver) in one species (mice) and thus did not meet the criteria for listing in the [RoC]." NTP, *Review Summary of the National Institute of Environmental Health Sciences (NIEHS/NTP) RoC Review Committee (RG1)*, at 1, available at http://ntp-server.niehs.nih.gov/newhomeroc/roc11/DEARG1RevSumm.pdf. While an NTP RoC Subcommittee Summary Report has not yet been posted, this clearly must have been the conclusion of at least eight of the nine voting members of the Subcommittee.

25 *See* Panel's Initial Comments at 8-10, as well as other factors described therein.

26 *Id.* The incidence of tumors in ethanol-treated controls was: 66%, 64%, and 10% for adenomas/carcinomas combined, adenomas, and carcinomas, respectively, in females; and 78%, 62%, and 24% for adenomas and carcinomas combined, adenomas, and carcinomas, respectively, in males. Response in the female controls was outside the NTP historical control incidences. *See* Panel's Initial Comments, Attachment 2 (Diethanolamine: A Conversation with OEHHA Staff, by W. T. Stott, Ph.D.), Table A; NTP Bioassay at 43-44.

the grounds that there were extremely high incidences of liver tumors in the controls.[27]

- The National Institute of Environmental Health Sciences' (NIEHS) Tg.AC transgenic mouse study on DEA was negative.[28]

- The NTP bioassays on DEA condensates should not be considered to provide any evidence of carcinogenicity of DEA at any tissue site. This conclusion follows from numerous factors, which are discussed in detail in the Panel's Initial Comments.[29] For example, the DEA condensate studies were bioassays of complex mixtures of imprecise composition of many chemicals, of which DEA comprised only a small and quantitatively unknown proportion.[30] Therefore, as the International Agency for Research on Cancer (IARC) concluded, the condensate studies "were not designed as, and did not represent, conventional or adequate carcinogenesis bioassays of [DEA]."[31] In the Review Summary for RG2, RG2 also concludes that the condensate studies "do not provide direct evidence for the carcinogenicity of DEA alone."[32]

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27 November 19, 2002, Transcript at 227-228. *See also id.* at 221 (Testimony of Dr. Stott).

28 Spalding, *et al.* (2000). "Response of Transgenic Mouse Lines $p53^{+/-}$ and Tg.AC to Agents Tested in Conventional Carcinogenicity Bioassays." *Toxicol. Sci.* 53: 213-223.

29 Panel's Initial Comments at 27-31.

30 *See id.* at 28-29.

31 IARC, *Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 77 -- Some Industrial Chemicals* (2000) at 362.

32 Review Summary for RG2 at 1. In addition, in presenting the NTP Background Document on DEA at the NTP RoC Subcommittee meeting, Ms. Ruth Lunn (NIEHS) concluded that any increase in tumor incidences observed in the condensate studies cannot be attributed to DEA because the condensates were complex mixtures and contain other substances. November 19, 2002, Transcript at 208.

C. Other Data Corroborate That DEA Does Not Meet the Listing Criteria

1. Extensive Evidence Shows That DEA Is Not Genotoxic

As discussed in detail in the Panel's Initial Comments, DEA has been extensively tested for potential genotoxicity in numerous tests and test systems and virtually uniformly has been found to be negative.[33] The RG2 Review Summary also concludes that DEA does not appear to be genotoxic. Moreover, the presentation of the conclusions of the NTP Background Document for DEA at the NTP RoC Subcommittee meeting also indicated that DEA has been tested for genotoxicity in various systems and does not appear to be genotoxic.[34] Further, IARC, in its 2000 Monograph, concluded that the data available to the Working Group do not indicate that DEA is genotoxic.[35]

2. Mechanistic Research Specifically on DEA Indicates That, to the Extent DEA Can Potentially Induce Tumors in Mice, It Does So by a Mechanism That Is Not Relevant to Humans

As discussed in detail in the Panel's Initial Comments, a number of studies have been conducted to elucidate the mechanism by which DEA potentially may induce tumors in mice. This research provides convincing evidence that to the extent DEA can induce tumors in mice, it does so by causing chronic choline deficiency. From this research and other factors

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33 *See* Panel's Initial Comments at 25-27.

34 November 19, 2002, Transcript at 209-210 (Presentation by Ms. Ruth Lunn, NIEHS).

35 IARC Monograph at 369-372 and 374.

described in the Panel's Initial Comments, it is clear that any reasonably anticipated human exposures to DEA that could result from the normal production and/or use of DEA or products containing DEA would not cause choline deficiency in humans. Additional research has been published since submission of the Panel's Initial Comments. This has included a very comprehensive research study by Lehman-McKeeman, *et al.* (2002) (*Toxicol. Sci.* 67:38-45)[36] "demonstrat[ing] that DEA treatment causes a spectrum of biochemical changes with choline deficiency in mice and demonstrat[ing] a clear dose-concordance between DEA-induced choline deficiency and hepatocarcinogenic outcome." In addition, recent data presented by L. Kamendulis, *et al.* (2003) (*Toxicol. Sci.* 72:S-1, Abstr. No. 1155) at the Society of Toxicology Annual Meeting demonstrated a lack of DEA or choline deficiency stimulated cell proliferation in cultured human hepatocytes in contrast to responses obtained with rat and mouse hepatocytes.

Accordingly, the mechanistic research alone establishes that DEA does not appear to pose a carcinogenic risk to humans.

II. INTERNATIONAL AND U.S AUTHORITIES HAVE DETERMINED THAT DEA SHOULD NOT BE CLASSIFIED AS PRESENTING A CARCINOGENIC RISK TO HUMANS, THUS CORROBORATING THE FINDINGS OF RG1, RG2, AND THE NTP RoC SUBCOMMITTEE THAT DEA SHOULD NOT BE LISTED

IARC recently found that the weight of the scientific evidence does not establish that DEA is likely to be a carcinogenic risk to humans, thus supporting the views of the Panel, as well as of RG1, RG2, and the NTP RoC Subcommittee. Based on a weight of the evidence

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[36] The Panel submitted a preprint of this study to NTP with a letter dated December 21, 2001 (from Jonathon T. Busch to Dr. C.W. Johnson).

review of the relevant scientific literature, IARC concluded that DEA is "not classifiable as to its carcinogenicity to humans" and therefore is a Group 3 chemical.[37]

Significantly, too, the Office of Environmental Health Hazard Assessment (OEHHA), the lead agency for the implementation of California's Proposition 65, announced earlier this month that it has withdrawn its previous proposal to list DEA as a chemical known to the State to cause cancer. OEHHA had originally announced in 1999 that it was considering the listing of DEA under Proposition 65 under the statute's "authoritative bodies" procedure based on the findings of the NTP report on the dermal bioassay on mice and solicited public comment on that proposal. On March 7, 2003, OEHHA issued a notice which states that considerable scientific information has been released subsequent to the release of the NTP report and that OEHHA, after consideration of this information, has determined not to proceed with the listing of DEA via the authoritative bodies listing mechanism.[38]

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37 IARC Monograph at 374.

38 OEHHA, "Decision Not to Proceed with the Listing of Diethanolamine via the Authoritative Bodies Listing Mechanism" (Mar. 7, 2003), available at http://www.oehha.ca.gov/prop65/CRNR_Notices/admin_listing/process_procedures/DEAnog.html.

CONCLUSION

For the reasons discussed above, the Panel believes that the available studies and data do not establish that DEA is "reasonably anticipated to be a human carcinogen" and therefore establish that NTP should accept the recommendations of RG1, RG2, and the NTP RoC Subcommittee and determine that listing of DEA in the *RoC* would not be appropriate.