January 24, 2005

Environmental Protection Agency
Public Information and Records Integrity Branch (7502C)
Office of Pesticide Programs
1200 Pennsylvania Ave., NW
Washington, DC 20460-0001

Re: Captan; Cancer Reclassification; Amendment of Reregistration Eligibility Decision; Notice of Availability (http://epa.gov/EPA-PEST/2004/November/Day-24/)

Federal Register: Federal Register: November 24, 2004 (Volume 69, Number 226) Page 68357-68360
Submitted by email to opp-docket@epa.gov and jennings.susan@epa.gov

Attention: Docket ID Number OPP-2004-0296

Dear Sir or Madam;

These comments are being submitted on behalf of the Natural Resources Defense Council (NRDC) and co-signers. NRDC is a non-profit organization dedicated to the protection of human health and the environment. We use law, science and the support of more than 1 million members and online activists to protect the planet's wildlife and wild places and to ensure a safe and healthy environment for humans and all living things. We do not have any direct or indirect financial or fiduciary interest in the manufacture or sale of captan or any other pesticides.

These comments detail our concerns with the Agency decision to weaken the classification of captan from “probable” to “not likely” carcinogenic at relevant exposures. NRDC challenges this process, which was completed entirely outside of the legally-mandated full scientific scrutiny and opportunity for full public participation required by the Federal Advisory Committee Act (FACA), 5 U.S.C. Appendix 2, the Administrative Procedure Act (APA), 5 U.S.C. §551 et seq., and the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), 7 U.S.C. §136. For example, EPA cannot lawfully use the advice provided by an obviously financially interested party (in the form of the recommendations of the industry’s Captan Task Force and the industry-funded Toxicity Excellence in Risk Assessment, or TERA), which was provided without observance of the process required by FACA, much less the dictates of the APA and FIFRA. FACA requires a balanced and chartered advisory committee to be constituted to provide such advice. The APA requires that in conducting its administrative activities, EPA must provide all sides an equivalent opportunity to participate, and that it is arbitrary, capricious, and not in accordance with law to proceed using a process that relies upon the arguments and comments of one side. Moreover, FIFRA §25(d) & (e), 7 U.S.C. §136w(d) & (e), requires EPA to convene a Scientific Advisory Panel (SAP) peer review panel prior to relying upon such a study or change in classification for captan. We therefore request that the Agency conduct and document its own full and independent scientific review of data relevant to captan’s carcinogenicity, and that this review be fully vetted in through its expert Scientific Advisory Panel. At the very least, the review submitted by the Captan Task Force (CTF) must go through an internal Agency scientific review, followed by a public review by its expert Scientific Advisory Panel. Anything less will be unlawful and scientifically unsound.

AGENCY ACTIONS:
Amendment to the 1999 Captan RED.

In 1999, EPA issued a RED for captan under section 4(g)(2)(A) of FIFRA. EPA has modified certain captan label requirements including: Double notification for all agricultural uses of captan; verbal notification of eye concerns associated with captan for 7 days following application; wettable powders applied aerially to be formulated in water soluble packages; reductions in the dichondra ornamental grass use rate; establishing a Re-Entry Interval for ornamentals, blackberries, blueberries, dewberries, raspberries, and grapes of 48 hours; removing the dust/mist respirator requirement for handling bags of treated seed.

Cancer reclassification.

When the RED was issued, captan retained its previous classification as a B2 chemical carcinogen (probable human carcinogen). Cancer risk from captan was quantified using the Agency's default approach described in the Agency's 1986 Cancer Risk Assessment Guidelines. After multiple requests by the Captan Task Force (CTF), and industry partnership between two captan registrants, the Makhteshim-Agan of North America, Inc. and Arvesta Corp1, the EPA agreed in 2001 to consider a re-evaluation of captan’s carcinogenicity by the science-for-hire group, Toxicology Excellence for Risk Assessment (TERA). The CTF provided funds for TERA to recruit and manage the process of reviewing the captan cancer mode of action data, even going so far as to generate a “peer” review of their own assessment, also funded by the CTF. This Peer Review Panel supported the CTF position, concluding that captan acted through a non-genotoxic threshold mode of action.

The Agency accepts the proposed mode of action as set forth by the CTF that suggests that:
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captan induces adenomas and adenocarcinomas in the duodenum of the mouse by a non-genotoxic mode of action involving cytotoxicity and regenerative cell hyperplasia that exhibits a clear dose threshold. These responses are reversible following cessation of captan exposure. There is a strong causal association (dose-response, temporality) indicating that tumor formation is secondary to cytotoxicity and hyperplasia and that the latter is a key event in the sequential cascade of events leading to cancer."
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In September 2004, the Agency, in accordance with the EPA 1999 Proposed Guidelines for Carcinogen Risk Assessment, classified captan as `not likely to be a human carcinogen at dose levels that do not cause cytotoxicity and regenerative cell hyperplasia' and `likely to be carcinogenic to humans following prolonged, high-level exposures causing cytotoxicity and regenerative cell hyperplasia.' The new cancer classification considers captan to be a potential carcinogen at prolonged high doses that cause cytotoxicity and regenerative cell hyperplasia. These high doses of captan are many orders of magnitude above those likely to be consumed in the diet, or encountered by individuals in occupational or residential settings.

CAPTAN USE

Captan was first registered as a pesticide in the U.S. 19512. The Toxic Releases Inventory (TRI) for industrial captan releases reports that in 2002, the most recent year for which data is compiled3, there was 566 pounds of fugitive air emissions, 932 lbs of point source air emissions

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1 Makhteshim-Agan of North America, Inc 551 Fifth Avenue, Suite 1100 New York NY 10176 and Arvesta Corporation 100 First Street, Suite 1700 San Francisco, CA 94105

2 EPA Office of Pesticides EPA-738-F99-015 Carbaryl RED facts. September 1999
Captan is a fungicide used to control diseases on orchard crops, seed treatments, ornamentals, lawns and turf, and is also used as an in-can preservative in adhesives and paint. Formulations include dust, emulsifiable concentrate, flowable concentrate, water dispersible granules, wettable powder, and a variety of others. Captan is applied by sprayers, chemigation equipment, power duster, liquid seed treater, paintbrush, tank-type sprayers, and other application methods. Captan is also applied as a post-harvest dip to apples, cherries and pears.

Currently, 158 captan products are registered, of which nine are manufacturing-use products. In 1989, EPA published the Position Document (PD4) "Captan; Intent To Cancel Registrations; Conclusion of Special Review" (54FR8116) on February 24, 1989. However, after receiving additional data, EPA decided to allow the continued registration of the following uses: all non-food uses, seed treatments, and certain food uses listed in the PD4 (almonds, apples, apricots, blackberries, blueberries, celery plant-beds, cherries, dewberries, eggplant plant-beds, grapes, green onions, lettuce, mangos, nectarines, peaches, post-harvest pears, pepper plant-beds, pimento plant-beds, plums/prunes, raspberries, spinach plant-beds, strawberries, taro and tomato plant-beds).

CAPTAN TOXICITY
EPA selected developmental endpoint in rabbits, with a NOAEL of 10 mg/kg/day, as a basis for determining the acute Reference Dose and the short- and intermediate-term dermal risk assessments. A three generation reproduction study in rats is the basis for the chronic RfD. The NOAEL in the study was 12.5 mg/kg/day.

The carcinogenicity of captan has been reviewed in 1986, 1989, 1995, 1999. Each time, the classification of captan as a B2 probable human carcinogen had been retained, most recently based on increased incidence of intestinal tumors in mice and rats. To estimate human cancer risks, the Agency used a linear, low dose extrapolation (presume no safe exposure level) approach for captan. In 1999, based on intestinal tumors in mice, a slope factor\(^5\) (Q1\(^*\)) of 2.4x10-3 (mg/kg/day)-1 was calculated.

INDUSTRY-PAID SCIENCE, INDUSTRY-PAID PEER REVIEW
While nothing in these comments is intended to impugn the integrity or morals of the members of the committee, we are concerned that the EPA is re-classifying captan without any

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3 TRI On-site and Off-site Reported Disposed of or Otherwise Released (in pounds), for facilities in All Industries, CAPTAN, U.S., 2002. Data source: 2002 Data Update as of August 2, 2004

4 The "unknown" category of disposal indicates that a facility is not aware of the type of waste management used for the toxic chemical that is sent off-site. Therefore, EPA has categorized this method as the least desirable type of waste management (environmentally least desirable) and has included it as a type of disposal or other release for reporting purposes. Data from Section 6.2, Code M99, on the TRI Form R.

5 A slope factor is an estimate of a carcinogen’s potency, characterized as a plausible upper bound on the increased human cancer risk from lifetime exposure to an average dose of 1 mg/kg-d. That is, the slope factor estimates a bound on the risk per mg/kg-d, accordingly, the slope factor is expressed in units of inverse lifetime-average dose, or (mg/kg-d)-1. Multiplying a slope factor by a lifetime-average dose (in mg/kg-d) yields a plausible upper bound on the increased probability of developing cancer from exposure to the carcinogen.
peer review by a committee that is subject to the Federal Advisory Committee Act to ensure a fair and balanced committee, and notification of the peer review in the federal register so the public is given an opportunity to submit comments in a timely manner to inform the review where appropriate. Instead, the document supporting the weaker cancer classification of captan was reviewed by a private science-for-hire organization without notice in the federal register, and without any participation by the public. NRDC challenges this process, which appears to have been completed outside of full scientific scrutiny, outside of full opportunity for public participation, and outside of the FACA, APA, and FIFRA procedures that are supposed to guide this process.

The peer-review of the Captan Task Force (CTF) scientific analysis was sponsored by the CTF. That is, the captan registrants paid for a peer-review of their own report. To provide a thin degree of separation, the peer review was conducted by an “independent” organization, the Toxicology Excellence in Risk Assessment (TERA). TERA is a non-profit organization claiming to be “dedicated to the best use of toxicity data in risk assessment”. The TERA “independent” review panel included eight members; four consultants from private companies, two industry employees, and two government employees. While TERA claims to have vetted all review panel members through its conflict of interest process, its conflict policy is far more permissive than the Federal Advisory Committee Act (FACA) guidelines. In particular: a) TERA has no requirement to require the membership of the advisory committee to be fairly balanced in terms of the points of view represented, and b) if financial conflicts are identified, then TERA has a plan to “manage” the conflict while still retaining the conflicted individual. For the members of

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6 http://www.tera.org/

7 TERA report of peer review meeting. Cancer assessment for captan. September 3-4, 2003. page 1

8 M Gargas, The Sapphire Group; D Goodman, Covance Laboratories, Inc.; A Shipp, Environ Health Sciences Institute; G Hard, consultant.


10 M Moore, FDA; L Sirinek, Ohio EPA

11 All Federal Advisory Committees must comply with the Federal Advisory Committee Act (FACA) (5 U.S.C. App. C) and related regulations. The Federal Advisory Committee Act (FACA) pertains to federal advisory committees established by US House and/or Senate legislative action (see http://www.epa.gov/ocem/faca/fed_adv_comm_act.htm for a complete description). Essential elements of FACA relevant to this position paper include the following: “In considering legislation establishing, or authorizing the establishment of any advisory committee ….. such legislation shall— (1) contain a clearly defined purpose for the advisory committee; (2) require the membership of the advisory committee to be fairly balanced in terms of the points of view represented and the functions to be performed by the advisory committee; (3) contain appropriate provisions to assure that the advice and recommendations of the advisory committee will not be inappropriately influenced by the appointing authority or by any special interest, but will instead be the result of the advisory committee's independent judgment”.

12 The TERA conflict policy states that, “An individual may have a conflict of interest that might influence their scientific opinions on an assessment. Situations might include: 1) An individual’s employer may manufacture, purchase, use, or dispose of the chemical under review. 2) An individual’s employer may be involved in other significant activities for that chemical. 3) An individual may hold financial interests in companies producing, using, disposing of, or selling the chemical under review. 4) An individual (or the individual’s employer) has taken a public position on an issue. 5) An individual may work for a government agency or program that regulates the chemical”. http://www.tera.org/peer/coi.html
the captan peer review, TERA determined that none of the panel members had a conflict of interested, defined by TERA as, “authorship or previous review of this document”, “employed by the Sponsor or Author organizations”, “currently receiving financial support” from the Sponsor or Author, or “those with direct personal financial interests in the outcome of the review”.

**SCIENTIFIC CONCERNS ARISING FROM THE CTF SCIENTIFIC ANALYSIS OF CAPTAN, AND UNRESOLVED IN THE TERA REVIEW**

*Executive summary (CTF review, p. 1-2)*

The CTF review asserts that the “rat bioassays provides no evidence that captan is associated with increased incidences of either renal tumors in males or of uterine sarcomas in females”. It is not clear why renal tumors in male rats and uterine sarcomas in females were dismissed. In any case, absence of evidence is not absence of carcinogenic potential. It is scientifically inappropriate to conclude that “captan’s tumorigenic potential is restricted to one tumor type in a single animal species”, after inappropriately disregarding existing data that indicates otherwise.

The CTF review claims that, “an epidemiology study of limited power involving 410 employees of a captan manufacturing plant in the U.S. provided no evidence of increased duodenal cancer or other oncogenic effects”. An inadequate epidemiology study provides no assurance of a lack of carcinogenic potential.

The CTF review concludes that, “captan is not genotoxic in intact animals”. However, the review acknowledges that captan is mutagenic in bacterial and eukaryotic test systems. The CTF review then notes that mutagenic activity is “eliminated or substantially decreased” in systems where proteins or thiols (-SH) have access to the acknowledged genotoxic metabolites of captan. It is plausible, then, that the genotoxicity of captan is not due to captan *per se*, but rather its cleavage products – THPI and/or thiophosgene. A direct-acting bacterial mutagen should be considered as a potential genotoxic agent in animals especially at sights of contact; if the metabolites are genotoxic, then sites where they are produced or distributed are a concern. Although more data is needed on duodenal stem cell dosimetry of captan and its metabolites, if half-life of the parent compound is less than 1 second, as the CTF claims (CTF review, p. 7), it is not expected that organ toxicity would be observed, except at sites of initial contact or cleavage. If THPI induces toxicity or genotoxic effects, then sites of its distribution are at risk. The fact that toxicity occurs in the duodenum and not in the forestomach implies that cleavage products are driving these effects; this is supported by metabolic data reported in the CTF review that, “THP1 and its metabolites were found in the duodenum, blood and urine” (CTF review, p. 8). These data available support the genotoxicity captan metabolites.

The CTF review claims without adequate supporting data that a “nongenotoxic mode of action for captan is proposed in which tumors are a secondary consequence of a cascade of non-neoplastic events”, but provides no evidence for this proposal. Where are data supporting the CTF claim that DNA damage in duodenum of mice leading to neoplasia was spontaneous and not induced by captan? A nongenotoxic mode of action is claimed but not shown.

In absence of data, the CTF claim that dermal or inhalation exposures would not be carcinogenic at any dose levels is unsupported. Given data demonstrating that captan and its metabolites are mutagenic, DNA-reactive agents, NRDC is highly concerned that an increased cancer risk by these routes of exposure is biologically plausible.

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Metabolism and Pharmacokinetics (CTF review, p. 7-9)

If thiophosgene is effectively deactivated with glutathione and other thiols, as the CTF review claims, then what caused the adenomas and adenocarcinomas in the proximal duodenum, reported in both sexes of mice?

Inconsistent statements in the CTF review, such as: “captan is rapidly absorbed and distributed” and “captan is not absorbed as parent compound” (CTF review, p. 8), are distressing.

The claim that captan is absorbed from the gut in the form of hydrolysis products was demonstrated in the oral ingestion studies described in the review. However, to adequately quantify the absorption and elimination kinetics of captan and its toxic metabolites (thiophosgene and THP1), data are needed describing the blood time-course following gavage and iv administered captan.

Hazard Identification (CTF review, p. 9-14)

How stable is captan in feed (it reacts rapidly with thiol compounds)? Do we know the dose of parent compound ingested by rats and mice? If the captan is degraded in feed, then the rodents in the study would be developing tumors at lower actual dose than the exposure amount indicated in the study, suggesting that captan is a more potent carcinogen than otherwise indicated. These data, if available, should be provided. If these data are not available, they should be generated and submitted for review.

In the Daly & Knezevich study (1983 Chevron Chemical Company report; re-evaluated by Robinson in a 1994 Zeneca report), duodenal tumors were induced in female CD1 mice at 800 and 6000 ppm, and in males at 6000 ppm, but focal hyperplasia of the duodenal mucosa was reported in both males and females only at 6000ppm. No toxicity at other organ sites was reported in this analysis; were other sites evaluated? It is concerning that the duodenal tumor rate in this study is so high in controls (2/91 males and 3/85 females); whereas the National Toxicology Program (NTP) historical control rate is 0/572. Was there any outside/independent pathology review of these lesions?

The rat study by Goldenthal et al (1982) reported liver hypertrophy and increased organ weights for kidney, heart, brain, and liver. How did this occur if captan has half-life of less than 1 second, as the CTF report asserts? The study also reported a significant increasing trend in males for combined adenoma and carcinoma of the kidney. Kidney tumors, especially carcinomas, are rare in control rats (~1%), and the trend is significant, suggesting the strength of these findings. The CTF review attempts to dismiss these findings by suggesting that the data was only “borderline” significant, and a dose-related trend was evident only for combined adenomas and carcinomas. However, “borderline” significant is still significant, and cannot be disregarded. And, combining adenomas and carcinomas is reasonable and the general practice of both the EPA and the NTP. Was the mortality rate affected by treatment? Why were animals that died early excluded from the statistical analysis? An appropriate statistical analysis should be done that includes all study animals, even those that died early in the study, consistent with EPA policy.

Human Epidemiology (CTF review, p. 13-14)

The epidemiology study discussed in the CTF review is inadequate to draw conclusive evidence that captan is or is not carcinogenic. It is an unpublished study submitted as a report to Tomen Agro, Inc in 2000. The study had no exclusion criteria; anyone who worked at least one day at a captan manufacturing plant was included. It included only 410 employees. There were no data on individual exposure histories. This study provides no useful information on the carcinogenicity of captan in humans, and should not be used to suggest, as the CTF review does, that, “employees at the captan plant were not at risk of cancer-related deaths”.

NRDC comments on Captan RED, OPP-2004-0296, Jan 24, 2005
A recently published epidemiology study of women farmers reported possible risks of breast cancer associated with captan exposure. The study included over 30,000 women with no history of breast cancer. Information on pesticide use and other information was obtained by self-administered questionnaire at enrollment from the women and their husbands. The study is very robust, and further follow-up will be helpful to clarify the link between captan and women’s cancer risks. A 2001 epidemiology study that the CTF review failed to cite reported a significant link between non-Hodgkin’s lymphoma and captan exposure (OR adj, 2.51; 95% CI, 1.32–4.76).

Genetic Toxicology (CTF review, p. 14-20)

The CTF review asserts that captan is not genotoxic, despite acknowledging that, “the genetic toxicology of captan remains a controversial area”, that is, poorly understood. Understanding the reactivity of captan and its metabolites with DNA requires identification of DNA adducts; such data, if it exists, is not discussed in the review. The CTF report attributes mutagenicity of captan to its metabolite, thiophosgene, but this is based on circumstantial evidence, and not robust data. Are mutations induced in the absence of S9 or cysteine proportional to thiophosgene production? The CTF report acknowledges that, “the mechanism by which captan induces these mutations is not clear”, but concludes that, “regardless of the mechanism involved, it is clear that captan has the potential to induce mutations when sensitive targets are exposed”. The EPA Draft 2003 Cancer Guidelines Supplemental are clear that when infants or children may be exposed, the default assumption is to assume no safe level of exposure, and use a linear low dose extrapolation for mutagens. Why has EPA abandoned this default assumption, despite acknowledging that the mechanism of gentoxicity of captan is poorly understood?

The CTF review claims that captan tests overwhelmingly negative for \textit{in vivo} mutagenicity, despite positive data in several studies. The CTF review does not provide data to justify disregarding the evidence of mutagenicity \textit{in vivo}. Because captan induces duodenal tumors, the CTF review paid attention to the potential genotoxicity of captan in the duodenum. The CTF review concludes that a lack of observed clastogenicity in a 5-day mouse oral study should be considered evidence that there was no genotoxicity in the stem cells of the duodenum.

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15 ORs adjusted for statistically significant medical variables (history of measles, mumps, cancer, allergy desensitization shots, and a positive family history of cancer in a first-degree relative), and with strata for the variables of age and province of residence.


17 EPA Draft Cancer Guidelines. EPA/630/R-03/003 External Review Draft February 2003 “When the data indicate a mutagenic mode of action, the available science (discussed above) indicates that higher cancer risks typically result from a given exposure occurring early in life when compared with the same amount of exposure during adulthood. Consequently, in the absence of early-life studies on a specific agent under consideration, U.S. EPA generally should: Use linear extrapolation to lower doses (see section 3.3.1 of the U.S. EPA cancer guidelines). This choice is based on mode-of-action data indicating that mutagens can give rise to cancers with an apparently low-dose-linear response.” www.epa.gov/ncea/raf/cancer2003.htm
However, lack of clastogencity does not mean that captan did not reach stem cells within crypts. This issue was not addressed at all in the TERA review.

The CTF review that captan is not mutagenic in vivo, and doesn’t bind to DNA. However, Captan does react with DNA (a captan derived DNA adduct was identified) and this may be the mechanism of its mutagenicity. This raises concerns of cancer risk at sites of direct contact. The finding reported in the CTF review of 35S bound to DNA of multiple tissues from animals dosed with 35S-captan was attributed to sulfur exchange with amino acid pool. This makes no sense; there is no S in DNA. The TERA review noted that these studies are limited due to methodological consideration.

Proposed mode of action

It is not clear how the weight of evidence of mutagenicity is used to make conclusions on in vivo mutagenicity. Studies that yield different results must be compared by dose, timing of exposure and collection of tissue. The proposed mechanism of carcinogenicity is cytotoxicity, cell proliferation and fixation of spontaneous mutations. Cell division rates in small intestine are naturally very high, yet spontaneous tumors are rare at this site. Reactivity of captan with DNA is dismissed by the CTF review because in vivo clastogenicity was not reported; that is not an adequate explanation. In Zeneca reports on BrdU labeling of crypt cells (Foster, 1994; Tinston, 1995), no increase was seen in female mice at the carcinogenic dose of 800 ppm (not a very reliable hypotheses of tumor induction). No data or analyses are provided to support the suggestion of a threshold, and abandonment of the EPA default assumption that all mutagens are linear (no threshold) at low doses.

The CTF review claims no evidence that captan is oncogenic in the rat, yet Hasegawa et al (Int J. Cancer 1993) found that captan in the diet increased the number of GST-P positive foci in the liver of rats initiated with a nitrosamine. How did captan cause this effect based on the proposed hypothesis? Why wasn’t this paper included in the analysis on captan carcinogenicity? Other studies that should have been reviewed and included in this analysis: 1) Dermal penetration of captan in rats by Fisher et al. (J Toxicol Environ Health, 1992). 2) Subcutaneous administration [i.e., oral administration is not required] of captan causes gastric mucosal cell proliferation (Wahby et al., Toxicol. Lett, 1990). 3) Dietary administration of captan increased hepatic DNA synthesis and loss of testicular DNA in rats (Decloitre and Martin, Carcinogenesis, 1980). 4) In the presence of S9, captan induced transformation of BALB/c 3T3 cells (Perocco et al., Jpn J. Cancer Res, 1995).

CONCLUSION

In light of the concerns raised in these comments with both the process and the scientific quality of Agency decision to weaken the classification of captan, we therefore request that the Agency conduct and document its own full and independent scientific review of data relevant to captan’s carcinogenicity, and that this review be fully vetted in through its expert Scientific Advisory Panel. At the very least, the review submitted by the Captan Task Force (CTF) must go through an internal Agency scientific review, followed by a public review by its expert Scientific Advisory Panel. Anything less will be unlawful and scientifically unsound.

Respectfully,

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