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riority to place money orders sales. The authority is to help the n its efforts to co-operate nforcement and other government rent the use of postal s a means of money obtained from the ugs.

June 19, 1988.

respondence should be L. Clapp, Secretary of , 1333 H Street, NW., ington, DC 20258 789-6840).

FORMATION CONTACT: General Counsel, 1333 uite 300, Washington. one: 202/789-6820).

INFORMATION: On June 7, 1988, the Governors of the Postal Service approved a decision (Docket No. MC88-1) of the Commission recommending a change in section 8.020 of the Domestic Mail Classification Schedule (DMSC). The Commission issued its recommended Decision on May 18, 1988. DMCS section 8.020 prescribes the maximum dollar value for money orders and states that the Postal Service may place restrictions on the number or dollar value of money order sales. Under the previous DMCS provision, the Postal Service could impose only temporary restrictions on the number of money orders which could be purchased at one time.

The Postal Service intends to use its new authority to set a limit on money order purchases which is lower than the threshold for reporting under the Bank Secrecy Act. The Treasury Department currently requires reporting of transactions over \$10,000. The Postal Service has found that the purchase of money orders in very large dollar amounts for legitimate reasons is rare. The Postal Service believes that the authority to limit the dollar amount of money order purchases will enable it to respond quickly to new methods which money "launderers" may adopt.

The Postal Service filed a request for this regulatory change on December 4, 1987. The Commission invited interested parties to comment and participate in the proceeding. 52 FR 46873-74. On May 2, 1988, the Postal Service filed a motion for acceptance of a unanimous stipulation and agreement. The Postal Service indicated that there was no opposition to the change described in the stipulation and agreement.

The amendment to the DMCS which is published in this order reflect the Governors' June 7, 1988, decision. Consistent with the Commission's

explanation in the rulemaking (Docket No. RM85-1) which led to the publication of the DMCS in the Federal Register, this addition is published as a final rule, since procedural safeguards and ample opportunities to have different viewpoints considered have already been afforded to all interested persons.

List of Subjects in 39 CFR Part 3001

Administrative practice and procedure, Postal Service.

PART 3001—RULES OF PRACTICE AND PROCEDURES

Subpart C—Rules Applicable to Requests for Establishing or Changing the Mail Classification Schedule

1. The authority citation for 39 CFR Part 3001 continues to read as follows:

Authority: 39 U.S.C. 404(b), 3603, 3622-3624, 3661, 3662, 84 Stat. 1303; (5 U.S.C. 553), 80 Stat. 383.

List of Changes

2. The following change in the Domestic Mail Classification Schedule published as Appendix A to Subpart C (39 CFR 3001.61 through 3001.68) of the Commission's rules of practice and procedure is adopted:

Revise 8.020 to read as follows:

8.020 The maximum value for which a domestic postal money order may be purchased is \$700. Other restrictions on the number or dollar value of postal money order sales, or both, may be imposed in accordance with regulations prescribed by the Postal Service.

By the Commission.

Charles L. Clapp,

Secretary.

[FR Doc. 88-13795 Filed 6-17-88; 8:45 am]

BILLING CODE 7715-01-M

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 300

[FRL-3400-5]

National Oil and Hazardous Substances Contingency Plan; National Priorities List Update; Correction

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule; correction.

SUMMARY: EPA is correcting errors in the Final Notice of Deletion of sites from the National Priorities List which appeared in the Federal Register on April 18, 1988 (53 FR 12680).

FOR FURTHER INFORMATION CONTACT: Allen Dotson at (202) 382-5755.

The following corrections are made in FRL-3366-7, the National Oil and Hazardous Substances Contingency Plan; National Priorities List Update published in the Federal Register on April 18, 1988 (53 FR 12680).

1. On page 12680, third column, line 53, change "In Group 14 remove:" to "In Group 15 remove:".

2. On page 12680, third column, line 56, change "In Group II remove:" to "In Group 12 remove:".

Dated: June 2, 1988.

Thaddeus L. Juszczak, Jr.,

Acting Deputy Assistant Administrator for Solid Waste and Emergency Response.

[FR Doc. 88-13811 Filed 6-17-88; 8:45 am]

BILLING CODE 6560-50-M

[OPTS-400010A; FRL-3400-2]

40 CFR Part 372

Toxic Chemical Release Reporting; Community Right-To-Know; Titanium Dioxide

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: EPA is deleting the substance titanium dioxide from the list of toxic chemicals under section 313 of Title III of the Superfund Amendments and Reauthorization Act of 1986 (SARA). EPA is amending the final rule codifying the list of chemicals published on February 18, 1988 (53 FR 4500). EPA is taking this action in response to petitions. Section 313(e) allows any person to petition the Agency to modify the list of toxic chemicals for which toxic chemical release reporting is required.

DATES: June 20, 1988.

FOR FURTHER INFORMATION CONTACT: Renee Rico, Petition Coordinator, Emergency Planning and Community Right-to-Know Hotline, Environmental Protection Agency, 401 M Street SW., (Mail Stop WH-562A), Washington, DC 20460, (800) 535-0202. (In Washington, DC and Alaska, (202) 479-2449).

SUPPLEMENTARY INFORMATION:

I. Introduction

A. Statutory Authority

The response to the petition and deletion are issued under sections 313(d)(3) and 313(e)(1) of Title III of the Superfund Amendments and Reauthorization Act of 1986 (Pub. L. 99-499, "SARA" or "the Act"). Title III of

SARA is also known as the Emergency Planning and Community Right-to-Know Act of 1986.

B. Background

Title III of SARA is intended to encourage and support emergency planning efforts at the State and local level and to provide the public and local governments with information concerning potential chemical hazards present in their communities.

Section 313 of Title III requires owners and operators of certain facilities that manufacture, process, or otherwise use a listed toxic chemical at certain threshold quantities, to report annually their releases of such chemicals to the environment. Only facilities that have manufacturing operations (in Standard Industrial Classification Codes 20 through 39) and have 10 or more employees must report. Such reports are to be sent to both EPA and the State in which the facility is located. The basic purpose of this provision is to make available to the public information about total annual releases of toxic chemicals from industrial facilities in their community. In particular, EPA is required to develop a computer data base containing this toxic chemical release information and to make it accessible by telecommunications on a cost reimbursable basis.

For reporting purposes, section 313 establishes an initial list of "toxic chemicals" that is composed of 328 entries, 20 of which are categories of chemicals. This list is a combination of lists of chemicals used by the States of Maryland and New Jersey for emissions reporting under their individual right-to-know laws. Section 313(d) authorizes EPA to modify by rulemaking the list of chemicals covered either as a result of EPA's self-initiated review or in response to petitions under section 313(e).

Section 313(e)(1) provides that any person may petition the Agency to add chemicals to or delete chemicals from the list of "toxic chemicals." EPA issued a statement of policy and guidance in the *Federal Register* of February 4, 1987 (52 FR 3479). This statement provided guidance to potential petitioners regarding the recommended content and format for submitting petitions. The Agency must respond to petitions within 180 days either by initiating a rulemaking or by using an explanation of why the petition is denied. If EPA fails to respond within 180 days, it is subject to citizen suits. In the event of a petition from a State governor to add a chemical under section 313(e)(2), if EPA fails to act within 180 days, EPA must issue a final rule adding the chemical to

the list. Therefore, EPA is under specific constraints to evaluate petitions and to issue a timely response.

State governors may petition the Agency to add chemicals on the basis of any one of the three toxicity criteria listed in section 313(d) (acute human health effects, chronic human health effects, or environmental toxicity). Other persons may petition to add chemicals only on the basis of acute or chronic human health effects. EPA may delete substances only if they fail to meet any of the criteria contained in section 313(d).

Chemicals are evaluated for inclusion on the list based on the criteria in section 313(d) and using generally accepted scientific principles, the results of properly conducted laboratory tests, or appropriately designed and conducted epidemiological or other population studies, that are available to EPA.

II. Description Of Petitions and Regulatory History

The Agency received three separate petitions to delist titanium dioxide, (TiO₂), CAS No. 13463-67-7, from the list of toxic chemicals. The three petitions, in order of receipt, were from: E.I. du Pont de Nemours and Company (DuPont), SCM Chemicals, Inc. and Didier Taylor Refractories Corporation, and Kemira Oy. EPA received the first petition on August 24, 1987, and under statutory deadline was required to respond by February 20, 1988. DuPont and SCM/Didier Taylor submitted extensive documentation to support their claim that TiO₂ fails to meet any of the statutory criteria in section 313(d).

EPA issued a proposed rule, published in the *Federal Register* of February 19, 1988 (53 FR 5004), announcing its intention to grant the petitions by deleting TiO₂. The Agency received 15 comments on the proposed rule. Fourteen comments were from a combination of individual companies and trade associations. These comments all supported the Agency's proposal to delete TiO₂ from the list of substances subject to reporting under section 313 of the Emergency Planning and Community Right-to-Know-Act. Several commenters agreed with EPA's health assessment and concurred that there is insufficient evidence indicating that TiO₂ may cause adverse effects to human health or the environment. The majority of the 14 commenters expressed particular support for the Agency's proposal to relieve facilities of having to report on TiO₂ releases starting with the 1987 calendar year.

One commenter, the Natural Resources Defense Council (NRDC),

opposed EPA's proposal to delete TiO₂ from the section 313 list. In summary, NRDC "believe(s) that EPA has erred in its health assessment of TiO₂, both in its interpretation of the long-term cancer bioassay studies and in its analysis of human epidemiologic surveys." EPA's response to each concern of NRDC follows.

NRDC expressed a concern with regard to the positive carcinogenicity response seen in a long-term inhalation bioassay study in rats carried out by the Haskell Laboratories of E.I. du Pont de Nemours and Company (Ref. 4). The positive response was seen at the high dose level of 250 mg/m³. Lung fibrosis at all dose levels was marginal. The commenter states that EPA inferred speculative explanations for tumor formation at the high dose level. In response, EPA believes that any proposed mechanism of action for the carcinogenic response (lung tumors) in the high dose rats are hypotheses whether they involve fibrosis, dust-overloading, or a hyperplastic response resulting in tumor formation. In the case of TiO₂, implied mechanisms of action are considered but do not strongly contribute to the weight-of-evidence for or against carcinogenicity. More importantly, regardless of any proposed mechanism of action, a single response in a single study is "limited" evidence according to the EPA "Guidelines for Carcinogenic Risk Assessment" (Ref. 8). All of the additional studies reviewed, six carcinogenicity bioassays and four mutagenicity bioassays, involving TiO₂ were negative (Ref. 1).

NRDC also commented on a National Cancer Institute (NCI) feeding study evaluated for carcinogenic effects (Ref. 10). Rats and mice were orally fed TiO₂ for 103 to 104 weeks at 25,000 or 50,000 ppm dose levels. NRDC commented that this study was not conducted at the maximum tolerated dose (MTD) levels, and that this study does not address the health-effects of airborne TiO₂ dust particles in animals. EPA maintains that TiO₂ was adequately tested in the study described above. The doses selected for rats and mice were accurately based on range-finding studies, and no toxicological effects were noted in the range-finding studies at any dose administered. In cases where the test chemical does not elicit toxicity, the Guidelines of the NCI Bioassay Program (Ref. 14) recommend that the maximum concentration of a test substance given in feed should not exceed 5 percent of the diet. Administration of TiO₂ at 25,000 ppm (2.5 percent) and 50,000 ppm (5.0 percent), the maximum concentrations used in the NCI study on

TiO₂ as described by the guidelines, did not result in significantly increased tumors in rats and mice of either sex. In response to NRDC's comment with regard to whether this NCI feeding study addressed health effects of airborne TiO₂, EPA did not utilize this study to evaluate the health effects of airborne TiO₂. The long-term inhalation study conducted by the Haskell Laboratories of E.I. du Pont de Nemours and Company (Ref. 4) measures the inhalation effects of TiO₂ on rats, and was discussed at length in EPA's *Health Assessment of Titanium Dioxide* (Ref. 15) and related memoranda (Refs. 1 and 2).

NRDC expressed the concern for human exposure to relatively high concentrations of the TiO₂ dust particles in the workplace and through accidental releases. Workplace exposure restrictions are governed by the Occupational Safety & Health Administration (OSHA). EPA based the exposure assessment for off-site persons on worst case values. Exposures likely to occur are well below the no observable adverse effect levels (NOAEL) estimated for lung fibrosis.

NRDC made inquiries about the existence of an inhalation bioassay study in mice mentioned in the health assessment document. EPA has found that the reference to the negative inhalation study in mice was a typographical error and should read "by inhalation to male and female rats."

NRDC commented that the "weight-of-evidence" approach that EPA used to judge the overall carcinogenic potential of TiO₂ is flawed. EPA believes that the commenter fails to provide evidence that would support any other finding using the Agency's Guidelines for Carcinogenic Risk Assessment (Ref. 8). "Limited" animal evidence is based on acceptable animal studies that suggest a carcinogenic effect but are limited because: (1) The data are derived from a single species, strain, or experiment; (2) the experiments are restricted by inadequate dose levels, inadequate duration of exposure to the agent, poor survival, too few animals, or inadequate reporting; or (3) an increase in benign tumors only. Following a review of the available literature on TiO₂, only a single species at the high dose level in one study reported a carcinogenic response (Ref. 8). Therefore, for purposes of section 313, "limited" animal evidence is interpreted as being insufficient evidence to support the determination that a chemical is "known to cause or can reasonably be expected to cause" cancer in humans.

NRDC believes that EPA downplays the human health effects of TiO₂ based

on occupational surveys conducted to date. NRDC agrees with EPA's findings that the recent DuPont epidemiological study on TiO₂/TiCl₄ workers was found to be inconclusive. However, NRDC commented that the Garabrant, et al. (Ref. 9) and the Daum, et al. (Ref. 3) epidemiological studies should be given more weight. EPA believes that these two studies are inadequate for assessing whether TiO₂ causes or can reasonably be anticipated to cause adverse health effects in humans. For example, the spirometry (lung function) results of Garabrant, et al. may be due to chance since the analysis did not reach statistical significance, and the authors did not explicitly state any hypothesis at the start of the study. Second, the absence of a control group or of pre-/post-shift lung function tests in both the studies makes the interpretation of the lung function test results *per se* difficult. Third, EPA questions the biological importance of pleural plaques and thickening, as reported by Garabrant, et al., because lung function decrements were not observed (Ref. 12).

EPA concluded that the Garabrant, et al. and Daum, et al. prevalent studies suffer from other limitations which potentially bias the reported results, making these studies less useful than well-conducted cohort studies of highly exposed long-term workers for examining potential adverse human health effects related to TiO₂ exposure. EPA believes that based on the limitations in the reviewed studies, these reports cannot carry more or less weight in the weight-of-evidence approach (Ref. 12). When these results and those reported by DuPont are fully evaluated, EPA believes that the data are inadequate for assessing the likelihood of chronic respiratory effects from TiO₂ exposure.

In summary, NRDC believes that EPA has erred in its health assessment of TiO₂, both in its interpretation of the long-term cancer bioassay studies and in its analyses of human epidemiological surveys. In accordance with the EPA "Guidelines for Carcinogen Risk Assessment" published in the Federal Register of September 24, 1986 (51 FR 33992), the overall weight-of-evidence determination for the carcinogenicity of TiO₂ is insufficient to reasonably anticipate that this chemical will cause cancer in humans based on inadequate human evidence and on limited animal evidence.

The proposed rule to delete TiO₂, published in the Federal Register of February 19, 1988 (53 FR 5004), contains information on EPA's review of the petitions, including the toxicity evaluation. This background information

will not be repeated here in the final rule. However, based on comments received from NRDC and a reanalysis of available data, EPA is clarifying a number of points with regard to: (1) Acute toxicity, (2) chronic toxicity, (3) oncogenicity, and (4) exposure. All other information concerning the hazard assessment is contained in the proposed rule.

1. *Acute toxicity.* Short-term exposure to high concentrations of inert fine particulates, such as TiO₂, can be associated with an increased incidence of respiratory effects like eye, nose and throat irritation, coughing, and sneezing. In sensitive subgroups such as the elderly or asthmatics, the effects may be more severe (Refs. 5 and 6). The observed effects are attributable to particulate matter in general and are not unique to any specific compound. The observed effects are generally reversible when exposure is reduced or terminated. The National Ambient Air Quality Standard (NAAQS) for particulate matter (50 µg/m³, annual average, 150 µg/m³, 24-hour average) was established, taking sensitive subgroups into consideration, to prevent the health effects described above (Refs. 5 and 6). Modeling studies showed annual and 24-hour average ambient air level concentrations of TiO₂ around the worst case manufacturing plant to be well below the NAAQS's for particulate matter (Ref. 16).

2. *Chronic toxicity.* The few available epidemiological studies are inadequate for determining whether respiratory effects would be expected in humans. Two prevalent studies, Garabrant, et al. (Ref. 9) and Daum, et al. (Ref. 3), reported lung abnormalities in workers exposed in titanium metal or TiO₂ production. Garabrant, et al. reported a statistically significant increase in pleural plaques and thickening; the biological meaning of this observation is questionable since the authors did not observe statistically significant decrements in lung function. Daum, et al. reported a statistically significant decrease in lung functions; however, the authors concluded that exposure associated with TiO₂ production by the sulphate process would not result in serious lung disease. Fibrosis was not observed in either study. A third study by Chen and Fayerweather (Ref. 7) of DuPont workers did not observe any consistent association between TiO₂/TiCl₄ exposure and respiratory morbidity. These three studies contain limitations which make their interpretation difficult. Therefore, EPA is unable to conclude that these studies show that TiO₂ causes or can

reasonably be anticipated to cause serious irreversible chronic health effects in humans. In the absence of adequate epidemiological data, animal data were used to predict expected effects in humans (Ref. 13).

TiO₂ has not been shown to produce significant chronic toxicity in animals. The results of a well-conducted, long-term inhalation study showed the development of minimal lung fibrosis in male and female rats exposed for 2 years to very high concentrations, 50 and 250 mg/m³, of respirable TiO₂ dust. Based on the results of this study, a NOAEL of TiO₂ via inhalation is estimated to be 10 mg/m³ (Ref. 15). Several injection studies showed that TiO₂ did not cause fibrosis of the rat lung or peritoneum following intratracheal instillation and intraperitoneal injection, respectively. TiO₂ appears to be non-toxic upon ingestion. The results of a NCI feeding study in rats and mice showed no chronic toxicity associated with ingestion of TiO₂ in the diet at 25,000 or 50,000 ppm. TiO₂ has been shown to have low *in vitro* biological activity as determined by cytotoxicity assays in cell culture (Ref. 15). Thus, EPA believes that the data are insufficient for showing that TiO₂ causes or can reasonably be expected to cause chronic effects in humans.

3. **Oncogenicity.** Based on a review of the available data, a weight-of-evidence determination concludes that the evidence is insufficient to reasonably anticipate that TiO₂ will cause cancer in humans. This conclusion is based on inadequate evidence in humans and limited evidence in animals. The evidence in animals includes a series of well-conducted laboratory studies performed in multiple species and involving multiple routes of exposure.

Results from a study of DuPont workers (Ref. 7) did not yield consistent associations between lung cancer mortality and morbidity and exposure to TiO₂/TiCl₄. Study limitations include ascertainment bias, inadequate control for confounding, and a cohort potentially composed of low-exposed individuals or individuals whose latency may not be fully expressed. These limitations make this study inadequate for determining potential human oncogenic effects from TiO₂ exposure (Ref. 15).

In long-term animal bioassays, TiO₂ was carcinogenic in male and female rats at a dose level that may have overwhelmed the normal clearance mechanism in the lung. TiO₂ was not carcinogenic at any doses by oral administration to rats and mice (both sexes) or by inhalation to male and

female rats at lower doses. Intraperitoneal injection in mice, subcutaneous injection in dogs, intratracheal instillation in hamsters and intramuscular injection in rats did not result in tumorigenicity. Additionally, TiO₂ does not induce gene mutations in prokaryotes and mammalian cells in culture, or DNA effects or cell transformation in mammalian cells in culture (Ref. 15).

The single positive result in high dose rats along with the multiple negative carcinogenicity results and the negative mutagenicity data leads to an overall weight-of-evidence determination that there is not sufficient evidence to show that TiO₂ will cause or can reasonably be expected to cause cancer in humans (Ref. 2).

4. **Exposure.** Both annual and short-term ambient air level concentrations around manufacturing sites of TiO₂ were estimated. Statistical wind summaries along with a variety of other input parameters, such as emission rate, particle size and density, and stack height, are used to estimate the annual ground level concentrations. Release information estimated for Kemira, the plant with the highest emission rates, was used to estimate the annual ambient air level concentrations at the plant boundaries. The results showed an annual average ambient air level concentration of 1.5 µg/m³ (Ref. 16) which is significantly below the 10,000 µg/m³ NOAEL for fibrosis of the lung inferred by animal studies (Ref. 15). There is a four fold margin between the exposure levels and the NOAEL for lung fibrosis, and concentrations at this exposure level are not expected to cause adverse lung effects. In addition, short-term modeling reveals 24-hour average ambient air level concentrations ranging from 11 to 33 µg/m³. Both the annual and short-term ambient air level concentrations for TiO₂ are well below the annual and 24-hour average NAAQS values of 52 and 150 µg/m³ for particulate matter. (Ref. 16).

III. Environmental Effects

TiO₂ exhibits a very low acute aquatic toxicity in fish with a 96-hour LC₅₀ greater than 1,000 mg/L (Ref. 11). TiO₂ appears to be nontoxic to mammals in acute exposures. This assessment is based on acute oral toxicity testing on guinea pigs and rats which showed LC₅₀s greater than 2,400 mg/kg (Ref. 11). Subchronic feeding studies on mice and rats produced no deaths and no gross or microscopic pathology which could be related to TiO₂ (Ref. 11). Chronic feeding studies also showed no effects related to TiO₂ (Ref. 11). Aquatic species reportedly bioaccumulate TiO₂ to levels

equal to or less than 16 µg/kg in tissues (Ref. 11).

EPA has reviewed the currently available data on the environmental effects of TiO₂ exposure and has found that there is insufficient evidence to establish that TiO₂ causes or can reasonably be anticipated to cause a significant adverse effect on the environment.

IV. Conclusion

EPA has reviewed the readily available data on the health and environmental effects of TiO₂ exposure. The acute toxicity, chronic toxicity, oncogenicity, and mutagenicity data reviewed did not show sufficient evidence to establish that TiO₂ is known to cause or can reasonably be anticipated to cause significant adverse health effects in humans at concentration levels that are reasonably likely to exist beyond facility site boundaries (Ref. 15). The environmental aquatic toxicity, terrestrial toxicity, and bioaccumulation data reviewed did not show sufficient evidence to establish that TiO₂ causes or can reasonably be anticipated to cause significant adverse effects on the environment of sufficient seriousness, in the judgment of the Administrator, to warrant reporting under section 313 (Ref. 11). Therefore, EPA is finalizing its proposal to delete TiO₂ from the list of toxic chemicals subject to release reporting requirements under section 313 of the Emergency Planning and Community Right-to-Know Act.

V. Effective Date

The Agency proposed to make the deletion of TiO₂ from the section 313 list of chemicals effective on or about June 1, 1988. Such an effective date would relieve facilities from their obligation to submit reports on TiO₂ for the 1987 reporting year by July 1, 1988. EPA's basis for this was explained in the preamble to the proposed rule. All comments which EPA received on this issue agreed with the Agency's proposed effective date of June 1, 1988.

In addition, because this rule deleting TiO₂ from the section 313 list of chemicals grants an exemption from a regulatory requirement, EPA is making the rule effective immediately upon publication in the Federal Register rather than 30 days from date of publication. See 5 U.S.C. section 553(d)(1). Thus, facilities are not obligated to file reports on TiO₂ on or before July 1, 1988 for 1987 activities.

VI. References

- (1) Cullen, L. J. *Memorandum to Bill Thomson*. USEPA. 4-14-88.
- (2) Cullen, L. J. *Memorandum to Dennis Leaf*. USEPA. 4-20-88.
- (3) Daum, S., et al. *Proc. R. Soc. Med.* 70:31-32 (1977).
- (4) DuPont Company, Trochimowicz, H. J., et al. *Tox. and Appl. Pharm.* 79:179-192 (1985).
- (5) EPA. *Review of the National Ambient Air Quality Standards for Particulate Matter: Assessment of Scientific and Technical Information*. OAQPS, USEPA. 1982.
- (6) EPA. *Updated Assessment of the Scientific and Technical Information: Addendum to the 1982 OAQPS Staff Paper*. OAQPS, USEPA. 1988.
- (7) Fayerweather, W. E., et al. *DuPont Petition: Attachment III*. 8-21-87.
- (8) *Federal Register* September 24, 1988 (51 FR 33992).
- (9) Garabrant, D. H., et al. *Scand. J. Work Environ. Health*. 13:47-51 (1987).
- (10) NCI. *Natl. Cancer Inst. Carcinog. Tech. Rep. Ser.* 97:114 (1978).
- (11) Morcock, R. E. *Ecologic Hazard Report on TiO₂: Superfund Delisting Petitions*. USEPA. 1987.
- (12) Siegel-Scott, C. L. *Memorandum to Dennis Leaf*. USEPA. 4-12-88.
- (13) Siegel-Scott, C. L. *Memorandum to Dennis Leaf*. USEPA. 4-15-88.
- (14) Sonteg, J. M., et al. "Guidelines for carcinogen bioassay in small rodents". *National Cancer Institute Carcinogenesis Technical Report*. Series. No. 1. 1976.
- (15) Thomson II, W. *Hazard Assessment of Titanium Dioxide*. USEPA. 1987.
- (16) Kinerson, R. *Addendum to Atmospheric Concentration Estimates from Titanium Dioxide Manufacturing Draft Report Dated January 1988*. USEPA. 1988.

VII. Regulatory Assessment Requirements

A. Executive Order 12291

Under Executive Order 12291, EPA must judge whether a rule is "major" and therefore, requires a Regulatory Impact Analysis. EPA has determined that this rule is not a "major rule" because it will not have an effect on the economy of \$100 million or more.

This rule will decrease the impact of the section 313 reporting requirements on covered facilities and will result in cost-savings to industry, EPA, and States. Therefore, this is a minor rule under Executive Order 12291.

This rule was submitted to the Office of Management and Budget (OMB) under Executive Order 12291.

There are four producers of TiO₂. Estimates of the number of processors/users that might be subject to reporting requirements range from 8,125 to 8,940 facilities. The estimated cost savings for industry over a 10-year period range from \$48 million to \$57 million, while the savings for EPA are estimated to be \$1

million (10-year present values using a 10 percent discount rate).

B. Regulatory Flexibility Act

Under the Regulatory Flexibility Act of 1980, the Agency must conduct a small business analysis to determine whether a substantial number of small entities will be significantly affected. Because the rule results in cost savings to facilities, the Agency certifies that small entities will not be significantly affected by this rule.

C. Paperwork Reduction Act

This rule relieves facilities from having to collect information on the use and releases of titanium dioxide. Therefore, there were no information collection requirements for OMB to review under the provisions of the Paperwork Reduction Act of 1980, 44 U.S.C. 15601 *et seq.*

List of Subjects in 40 CFR Part 372

Community Right-to-Know, Environmental protection, Reporting and recordkeeping requirements, Toxic chemicals.

Dated: June 9, 1988.

John A. Moore,
Assistant Administrator, Office of Pesticides and Toxic Substances.

Therefore, 40 CFR Part 372 is amended as follows:

PART 372—[AMENDED]

1. The authority citation continues to read as follows:

Authority: 42 U.S.C. 11013 and 11028.

§ 372.65 [Amended]

2. Section 372.65 (a) and (b) are amended by removing the entire entry for titanium dioxide under paragraph (a) and removing the entire CAS. No. entry for 13463-67-7 under paragraph (b).

[FR Doc. 88-13812 Filed 6-17-88; 8:45 am]
BILLING CODE 6560-50-M

DEPARTMENT OF TRANSPORTATION

Maritime Administration

46 CFR Part 249

[Docket R-101]

Approval of Underwriters for Marine Hull Insurance

AGENCY: Maritime Administration,
Department of Transportation.

ACTION: Final rule.

SUMMARY: The Maritime Administration (MARAD) is issuing this final rule to

govern the placement of marine hull insurance on subsidized and Title XI program vessels. These regulations afford companies participating in MARAD programs wider opportunity to obtain hull insurance coverage from financially sound underwriters with minimal regulatory constraints. Specifically, they eliminate the requirement that 75 percent of the required hull insurance coverage be placed in the American market, provide for the approval, under certain conditions, of additional foreign underwriters to participate in the writing of hull insurance on MARAD program vessels, and modify the limitation of an underwriter's risk on any single vessel.

DATE: This rule is effective July 20, 1988.

FOR FURTHER INFORMATION CONTACT: Edmond J. Fitzgerald, Director, Office of Trade Analysis and Insurance, Maritime Administration, Washington, DC 20590, Telephone: (202) 366-2400.

SUPPLEMENTARY INFORMATION: On October 11, 1985, MARAD published an Advance Notice of Proposed Rulemaking (ANPRM) in the *Federal Register* (50 FR 41531) concerning its existing policies regarding the placement of hull insurance on MARAD program vessels. The purpose of the ANPRM was to elicit opinions and data that would be used in the formulation of a proposed rule. There was general support for the concept from shipowners and previously non-admitted foreign underwriters, and opposition from the American marine insurance industry.

On April 17, 1988, MARAD conducted a public inquiry to give all interested parties an opportunity to provide more information and to support their positions. Based upon the presentations made at the inquiry and all materials submitted in connection with Docket R-101, MARAD prepared and published a Notice of Proposed Rulemaking (NPRM) to define its hull insurance policies for subsidized or Title XI program vessels. That notice was published in the *Federal Register* on October 18, 1987 (52 FR 38481) and elicited 18 comments, plus one request for an extension of time for filing (which was granted).

Comments were filed on behalf of the following carrier interests: Seahawk Management, Lykes Bros. Steamship Co., Inc., American Steamship Co., Hvide Shipping, Sea-Land Corporation, Crowley Maritime Corporation, Energy Transportation Corporation, Waterman Steamship Corporation, American Maritime Transport, Inc., Matson Navigation Company, and the American Institute of Merchant Shipping.