20460. Commenters are requested to submit any references cited in their comments. Commenters also are requested to submit an original and three copies of their written comments and enclosures. Commenters who want receipt of their comments acknowledged should include a self-addressed, stamped envelope. No facsimiles (faxes) will be accepted.

Electronic comments should be addressed to the E-mail address: ow-docket@epamail.epa.gov. Electronic comments must be submitted as an ASCII file and avoid use of special characters and any form of encryption, or may be submitted in WordPerfect 5.1 or 6.1. Electronic comments must be identified as ''EPA Method 1631-Notice of Data Availability.'' Electronic comments on this notice may be filed online at many Federal Depository Libraries. Electronic comments will be transferred into a paper version for the official record. EPA will attempt to clarify electronic comments if there is an apparent error in transmission.

A copy of the supporting documents and data received by the Agency during and pursuant to the comment period for the proposed rule are available for review at EPA's Water Docket, Room EB57, 401 M Street, S.W., Washington, D.C. 20460. For access to the Docket materials, call (202) 260–3027 between 9:00 a.m. and 3:30 p.m. Eastern Time for an appointment.

The complete text of this **Federal Register** notice and EPA Method 1631 may be viewed or downloaded on the Internet at http://www.epa.gov/ost/rules.

FOR FURTHER INFORMATION CONTACT: Dr. Maria Gomez-Taylor,U.S. Environmental Protection Agency, Office of Science and Technology, Engineering and Analysis Division (4303), 401 M Street, S.W., Washington, D.C., 20460, or call (202) 260-1639.

SUPPLEMENTARY INFORMATION:

On May 26, 1998 (63 FR 28867), EPA proposed to add EPA Method 1631: Mercury in Water by Oxidation, Purge and Trap, and Cold Vapor Atomic Fluorescence to 40 CFR Part 136 for National Pollutant Discharge Elimination System (NPDES) data gathering and compliance monitoring under the Clean Water Act (CWA). Mercury is a toxic pollutant as defined in Section 307(a)(1) of the CWA and at 40 CFR 401.16 and is a priority pollutant as listed in 40 CFR Part 423, Appendix A. EPA Method 1631 was proposed under the authority of Sections 301, 304(h), and 501(a) of the CWA. The Agency developed EPA Method 1631 in order to measure mercury reliably at the low levels associated with ambient water quality criteria (WQC) for mercury included in the National Toxics Rule (40 CFR 131.36) and Water Quality Guidance for the Great Lakes System (60 FR 15366). A further description of the development and validation of EPA Method 1631 is provided in the proposed rule.

Following the close of the comment period, the Agency obtained additional analytical data pertinent to EPA Method 1631. The additional data consist of results from laboratory studies and municipal and industrial effluent analyses conducted using EPA Method 1631. This notice makes available for public review and comment these analytical data. Generally, the data supplements existing data by demonstrating the applicability of EPA Method 1631 to a variety of municipal and industrial effluents. The Agency intends to consider these additional data in formulating the final rule for the use of EPA Method 1631.

Today's notice solicits comments only on the new data which confirm or refute the Agency's findings about the acceptability of EPA Method 1631 for the determination of mercury at the low levels associated with Water Quality Criteria. Specifically, the Agency seeks comment on the use of EPA Method 1631 to accurately measure mercury at low levels in a variety of water matrices based on the new data. The Agency does not intend to reopen the comment period on the entire proposed rule. Therefore, there is no need to submit comments on other aspects of the proposal.

The Agency does not interpret the new data as warranting any modification of the proposed rule nor do they indicate a reason to change the Agency's rationale for proposing EPA Method 1631. The Agency believes that these data support the Agency's conclusion that EPA Method 1631 is applicable to a variety of water effluents including municipal and industrial effluents.

Dated: March 1, 1999.

J. Charles Fox,

Assistant Administrator for Water.

[FR Doc. 99–5493 Filed 3–4–99; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 372

[OPPTS–400137; FRL–6054–2]

RIN 2070–AC00

Acetonitrile; Community Right-to-Know Toxic Chemical Release Reporting

AGENCY: Environmental Protection Agency (EPA).

ACTION: Denial of petition.

SUMMARY: EPA is denying a petition to remove acetonitrile from the list of chemicals subject to the reporting requirements under section 313 of the Emergency Planning and Community Right-to-Know Act of 1986 (EPCRA) and section 6607 of the Pollution Prevention Act of 1990 (PPA). EPA has reviewed the available data on this chemical and has determined that acetonitrile does not meet the deletion criterion of EPCRA section 313(d)(3). Specifically, EPA is denying this petition because EPA's review of the petition and available information resulted in the conclusion that acetonitrile meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) due to its potential to cause neurotoxicity and death in humans and its contribution to the formation of ozone in the environment, which causes adverse human health and environmental effects.

FOR FURTHER INFORMATION CONTACT: Daniel R. Bushman, Petitions Coordinator, 202–260–3882 or e-mail: bushman.daniel@epa.gov, for specific information regarding this document or for further information on EPCRA section 313, contact the Emergency Planning and Community Right-to-Know Information Hotline, Environmental Protection Agency, Mail Code 5101, 401 M St., SW., Washington, DC 20460, Toll free: 1–800–535–0202, in Virginia and Alaska: 703–412–9877, or Toll free TDD: 1–800–553–7672.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does This Document Apply To Me?

This document does not make any changes to existing regulations. However, you may be interested in this document if you manufacture, process, or otherwise use acetonitrile. Potentially interested categories and entities may include, but are not limited to the following:

Category	Examples of Potentially Interested Entities
Chemical manufacturers	Chemical manufacturers that manufacture acetonitrile, use acetonitrile as a chemical intermediate, or use acetonitrile in the manufacturing or processing of pharmaceuticals, agriculture chemicals, butadiene, isoprene and specialty chemicals and products (e.g., new high density batteries)
Chemical processors and users	Facilities that use acetonitrile as a process or reaction solvent

This table is not intended to be exhaustive, but rather provides a guide for readers regarding entities likely to be interested in this document. Other types of entities not listed in this table may also be interested in this document. Additional businesses that may be interested in this document are those covered under 40 CFR part 372, subpart B. If you have any questions regarding whether a particular entity is covered by this section of the CFR, consult the technical person listed in the ''FOR FURTHER INFORMATION CONTACT'' section.

B. How Can I Get Additional Information or Copies of This Document or Other Support Documents?

1. *Electronically*. You may obtain electronic copies of this document from the EPA Home Page at http://www.epa.gov/. On the Home Page select ''Laws and Regulations'' and then look up the entry for this document under the ''**Federal Register** – Environmental Documents.'' You can also go directly to the ''**Federal Register**'' listings at http://www.epa.gov/fedrgstr/.

2. *In person or by phone*. If you have any questions or need additional information about this action, please contact the technical person identified in the ''FOR FURTHER INFORMATION CONTACT'' section. In addition, the official record for this document, including the public version, has been established under docket control number OPPTS–400137. This record includes not only the documents physically contained in the docket, but all of the documents included as references in those documents (including the references cited in Unit VII. of this preamble). A public version of this record, which does not include any information claimed as Confidential Business Information (CBI), is available for inspection from 12 noon to 4:00 p.m., Monday through Friday, excluding legal holidays. The official record is located in the TSCA Nonconfidential Information Center, Rm. NE–B607, 401 M St., SW., Washington, DC. The TSCA Nonconfidential Information Center telephone number is 202–260–7099.

II. Introduction

A. Statutory Authority

This action is taken under sections 313(d) and (e)(1) of EPCRA, 42 U.S.C. 11023. EPCRA is also referred to as Title III of the Superfund Amendments and Reauthorization Act of 1986 (SARA) (Pub. L. 99–499).

B. Background

Section 313 of EPCRA requires certain facilities manufacturing, processing, or otherwise using listed toxic chemicals in amounts above reporting threshold levels, to report their environmental releases of such chemicals annually. These facilities must also report pollution prevention and recycling data for such chemicals, pursuant to section 6607 of the PPA, 42 U.S.C. 13106. EPCRA section 313 established an initial list of toxic chemicals that comprised more than 300 chemicals and 20 chemical categories. Acetonitrile was included on the initial list. Section 313(d) authorizes EPA to add or delete chemicals from the list and sets forth criteria for these actions. EPA has added and deleted chemicals from the original statutory list. Under section 313(e)(1), any person may petition EPA to add chemicals to or delete chemicals from the list. Pursuant to EPCRA section 313(e)(1), EPA must respond to petitions within 180 days, either by initiating a rulemaking or by publishing an explanation of why the petition is denied.

EPCRA section 313(d)(2) states that a chemical may be listed if any of the listing criteria are met. Therefore, in order to add a chemical, EPA must demonstrate that at least one criterion is met, but does not need to examine whether all other criteria are also met. Conversely, in order to remove a chemical from the list, EPA must demonstrate that none of the criteria are met.

EPA issued a statement of petition policy and guidance in the **Federal Register** of February 4, 1987 (52 FR 3479) to provide guidance regarding the recommended content and format for submitting petitions. On May 23, 1991 (56 FR 23703), EPA issued guidance regarding the recommended content of petitions to delete individual members of the section 313 metal compounds categories. EPA has also published in the **Federal Register** of November 30, 1994 (59 FR 61432) (FRL–4922–2) a statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals.

III. Description of Petition and Regulatory Status of Acetonitrile

Acetonitrile is on the list of toxic chemicals subject to the annual release reporting requirements of EPCRA section 313 and PPA section 6607. Acetonitrile was among the list of chemicals placed on the EPCRA section 313 list by Congress. Acetonitrile is listed under the Clean Air Act (CAA) as a volatile organic compound (VOC) and a hazardous air pollutant. Acetonitrile is also on the Hazardous Waste Constituents List under the Resource Conservation and Recovery Act (RCRA).

On February 4, 1998, EPA received a petition from BP Chemicals Inc. (BP) and GNI Chemicals Corporation (GNICC) to delete acetonitrile from the list of chemicals reportable under EPCRA section 313 and PPA section 6607. Specifically, BP and GNICC believe that acetonitrile meets all of the criteria for delisting under EPCRA section 313(d)(3) because: (1) ''acetonitrile is not known to cause and cannot be reasonably anticipated to cause significant adverse human health effects at concentrations that are reasonably likely to exist beyond facility boundaries as a result of continuous or frequently recurring releases''; (2) ''at exposures likely to be found at facility fence lines, acetonitrile is not known to cause and cannot be reasonably anticipated to cause cancer or teratogenic effects of serious irreversible reproductive dysfunction, neurological disorders, heritable genetic mutations, or other chronic health effects''; and (3) ''acetonitrile is not known to cause or reasonably likely to cause significant adverse effects to the environment because it is not toxic or persistent and does not readily bioaccumulate.'' In addition, the petitioners believe that EPA's policy requiring that a chemical not be a VOC ''. . . is irrelevant and should not be considered for this delisting petition.'' The petitioners argue for a revised interpretation of the EPCRA section 313 VOC policy, contending that EPA does not have the statutory authority to list chemicals based upon their status as a VOC. EPA has stated in past **Federal Register** documents (54 FR 4072, January 27, 1989; 54 FR 10668, March 15, 1989; 59 FR 49888, September 30, 1994; 60 FR 31643, FRL–4952–7, June 16, 1995; and 63 FR 15195, FRL–5752–6, March 30, 1998) that VOCs meet the criteria for

listing under EPCRA section 313 due to the fact that VOCs contribute to tropospheric ozone. Notwithstanding the petitioners' belief that a chemical's VOC status is irrelevant to EPCRA section 313 listing, the petitioners have submitted a petition to EPA's Office of Air and Radiation (OAR) to add acetonitrile to the list of ''negligibly photoreactive chemicals'' under 40 CFR 51.100(s)(1).

IV. EPA's Technical Review of Acetonitrile

The technical review of the petition to delete acetonitrile from TRI reporting requirements (Ref. 1) included an analysis of the chemistry (Ref. 2), toxicology (including metabolism and absorption, health effects, and ecological effects) (Ref. 3), environmental fate, and exposure (Ref. 4) data known for acetonitrile. A more detailed discussion for each related topic can be found in EPA's technical reports (Refs. 2, 3, 4, 5, and 6) and the studies contained and referenced in the docket.

A. Chemistry and Use

Acetonitrile, also known as cyanomethane and methyl cyanide, is a colorless, volatile, flammable liquid (boiling point = 81.6 °C; flash point = 12.8 °C) with an ether-like odor. It is completely miscible with water and many organic solvents. Its high dielectric constant and dipole moment make it an excellent solvent for both inorganic and organic compounds, including polymers. Acetonitrile forms a low boiling azeotrope with other organic solvents. The impurities present in commercial grade acetonitrile are water, unsaturated nitriles, toluene, aldehydes, and amines. Acetonitrile is a relatively inert material but produces hydrogen cyanide when heated to decomposition or reacted with acids or oxidizing agents.

Acetonitrile is produced commercially as a by-product during the manufacture of acrylonitrile by high temperature catalytic oxidation of propylene in the presence of ammonia (the Sohio process of propylene ammoxidation). Acetonitrile and hydrogen cyanide are principal by-products of the process. The ratio of acetonitrile to acrylonitrile produced is typically 1:35 (Refs. 2, 6, and 7). Reported production of acetonitrile in the United States (US) in 1993 was 17,859,000 kilograms (kg) (Ref. 6).

Acetonitrile is primarily used as: a reaction solvent in the production of pharmaceuticals; an analytical instrumentation/extraction solvent; an extraction solvent in extracting butadiene and isoprene from reaction steams; and a solvent for the manufacture and formulation of agricultural chemicals. Acetonitrile is also used for extracting fatty acids (e.g., from fish liver oils and other animal and vegetable oils) and in refining copper, dyeing textiles, recrystallizing steroids, and other extraction applications. Acetonitrile is also used as a chemical intermediate for many types of organic compounds (Refs. 2, 6, and 7).

B. Metabolism and Absorption

Absorption of acetonitrile occurs after oral, dermal, or inhalation exposure. Although no quantitative absorption data were found for oral exposure, signs of acute toxicity, observed after oral exposure, indicate that absorption occurs. In humans, 74 percent of acetonitrile was absorbed orally from cigarette smoke held in the mouth for 2 seconds; when inhaled into the lungs, absorption increased to 91 percent. Dogs exposed by inhalation to 16,000 parts per million (ppm) of acetonitrile for 4 hours appeared to reach steady-state blood concentrations within 3 to 4 hours (Ref. 3).

Acetonitrile and its metabolites are transported throughout the body in the blood. After oral or inhalation exposures in experimental animals, acetonitrile or its metabolites were found in the brain, heart, liver, kidney, spleen, blood, stomach, and muscle. After a fatal human inhalation exposure, metabolites were found in the brain, heart, liver, kidney, spleen, blood, stomach, and muscle, as well as skin, lungs, intestine, testes, and urine (Ref. 3).

Acetonitrile is metabolized to hydrogen cyanide and thiocyanate, which are responsible for the toxic effects of the chemical. Metabolism is mediated by the cytochrome P-450 system (Refs. 3 and 8).

Acetonitrile is excreted as acetonitrile in expired air and as acetonitrile or its metabolite in urine. Urinary excretion of the thiocyanate metabolite following oral exposure in rats ranged from 11.8 percent to 37 percent of the administered dose. Acetonitrile concentrations of 2.2 to 20 micrograms/100 milliliters (ml) of urine have been found in heavy smokers (Ref. 3).

C. Toxicity Evaluation

1. *Acute effects*. The only available data regarding acute effects of acetonitrile in humans are from reports of accidental poisonings resulting from acute exposures. It is likely that these acute exposures were at concentrations in excess of 500 ppm (Refs. 3 and 8). At these concentrations, acetonitrile affects the central nervous system producing excess salivation, nausea, vomiting, anxiety, confusion, hyperpnea, dyspnea, rapid pulse, unconsciousness, and convulsions, followed by death from respiratory failure. These effects are consistent with those following inorganic cyanide exposure and with effects seen with other aliphatic nitriles, suggesting that the toxic effects of acetonitrile may be correlated with the metabolic release of cyanide. Acute effects of acetonitrile in humans at concentrations less than 500 ppm consist of irritation of the mucous membranes. No other human data were available that allow characterization of acute toxicity at lower concentrations (Ref. 3).

In animal studies, acetonitrile induced acute toxicity at relatively high inhalation exposures. In acute exposure inhalation toxicity studies, the LC_{50} (i.e., the concentration of a chemical that is lethal to 50 percent of the test organisms) ranges from 2,300 to 5,700 ppm in mice and from 7,500 to 16,000 ppm in rats (Refs. 3 and 8). Mice and guinea pigs appear to be more sensitive than rats for acute toxicity by the oral route. The lowest LD_{50} (i.e., the dose of a chemical that is lethal to 50 percent of the test organisms) values in older rats ranged from 1,300 to 6,700 milligrams per kilogram (mg/kg); young rats appeared to be more sensitive with a LD_{50} value of 157 mg/kg (Refs. 3 and 8). A LD_{50} range of 390 to 3,900 mg/kg was reported by the dermal route in rabbits (Ref. 3). Non-lethal effects at 500 ppm in mice include respiratory effects, convulsions, and eye and lung irritation (Refs. 3 and 8).

2. *Chronic effects*—i. *Carcinogenicity*. EPA has identified no human data in the literature on the cancer effects of acetonitrile. The carcinogenicity of acetonitrile has been studied in experimental animals by the National Toxicological Program (NTP) in F344/N rats and B6C3F1 mice in 2-year inhalation studies (Ref. 9). Under the conditions of the 2-year inhalation studies, there was equivocal evidence of carcinogenic activity of acetonitrile in male F344/N rats based on marginally increased incidences of hepatocellular adenoma and carcinoma in the high-dose (400 ppm) group. There was no evidence of carcinogenic activity of acetonitrile in female F344/N rats, or male and female B6C3F1 mice exposed to any concentration of acetonitrile (Refs. 3 and 9).

No evidence of carcinogenicity of structurally related chemicals has been identified. Acrylonitrile is carcinogenic but it is not a good analogue for acetonitrile because acrylonitrile contains a double bond and is

genotoxic. Acetonitrile is biotransformed via a cytochrome P450 monoxygenated system to cyanohydrin, which then decomposes slowly to hydrogen cyanide and formaldehyde and subsequently is detoxified. Based on the results of the NTP studies, there is insufficient evidence to conclude that acetonitrile may or has the potential to cause cancer in humans (Refs. 3 and 9).

ii. *Mutagenicity*. Positive results were obtained in some *in vitro* studies that would present a concern, albeit weak, for mutagenicity. However, due to the lack of evidence for effects in the mammalian gonad *in vivo*, either in mutagenicity studies or in reproductive/teratology studies, there is no basis for concern for potential heritable gene or chromosomal mutagenicity of acetonitrile (Ref. 3).

iii. *Developmental toxicity*. Information in humans reviewed by the Agency regarding the developmental toxicity of acetonitrile is limited to a study of laboratory workers and pregnancy outcomes, in which a slightly elevated, although non-significant, odds ratio was reported for congenital malformations for women exposed to acetonitrile. Seven cases of spontaneous abortion were noted for women exposed to acetonitrile out of a total of 206 cases reported (535 women were involved in the study). This study was confounded by worker exposure to other chemicals (Refs. 3, 10, and 11).

The developmental toxicity of acetonitrile has been evaluated in rats, rabbits, and hamsters. Overall, evidence for developmental toxicity is weak. Oral and inhalation studies in rats and rabbits have shown no signs of developmental toxicity at doses that did not produce excessive maternal mortality. The only data available on hamsters utilized short durations (60 minutes on day 8 of gestation) to high concentrations of acetonitrile vapor or by gavage on day 8 of gestation. There were some signs of developmental toxicity in hamsters by both routes at dose levels that did not produce overt maternal mortality; however, these studies are difficult to interpret for human risk assessment because: (1) Very high doses were used, and (2) no developmental effects have been observed in other species at doses below those which produced extreme maternal toxicity (10 percent mortality or greater).

iv. *Reproductive toxicity*. Since no definitive two-generation reproductive toxicity or fertility studies with acetonitrile have been identified, information in animals is limited to developmental toxicity studies in which only some reproductive parameters were assessed. Moreover, the data appear to be equivocal. For example, there were no changes in pregnancy rates or resorptions in rats exposed to doses as high as 500 milligram/kilogram/day (mg/kg/day) (Ref. 14). However, in another study, significant increases in post-implantation losses and early resorptions in rats exposed to 275 mg/kg/day acetonitrile were observed (Ref. 15). In other studies, acetonitrile was not shown to produce any effects on: The testis, epididymis, and cauda epididymis weights; sperm motility, number, or morphology; or the average estrous cycle length, frequency of estrous stages, or terminal female body weight (Ref. 16). In conclusion, available animal studies do not fully characterize the reproductive toxicity of acetonitrile. Although some reproductive parameters appeared to be unaffected in some studies, none of the studies evaluated the reproductive performance or reproductive system effect of offspring exposed *in utero*. Therefore, there is not sufficient information to fully characterize the potential for reproductive toxicity of acetonitrile (Ref. 3).

v. *Neurotoxicity*. In humans, the nervous system is a major target for acetonitrile toxicity. In reports of accidental poisonings in humans exposed to presumed high concentrations of acetonitrile, signs of salivation, nausea, vomiting, anxiety, confusion, hyperpnea, dyspnea, rapid pulse, unconsciousness, and convulsions followed by death from respiratory failure were observed (Refs. 3 and 8). No information was found on the adverse neurotoxic effects of long-term human exposure to acetonitrile. Brief references appear in the Hazardous Substances Data Bank (HSDB) (Ref. 17) suggesting that chronic exposure to acetonitrile may cause headache, anorexia, dizziness, and weakness, but no additional information on neurotoxicity was provided in support of these statements (Ref. 3).

Neurotoxicity studies indicate that subchronic exposures (subchronic is defined by EPA's Integrated Risk Information System (IRIS) as multiple or continual exposures occurring usually over three months (Ref. 18)) to acetonitrile can cause serious and irreversible health effects in animals. Monkeys appeared to be more sensitive than rats to the neurotoxic effects of acetonitrile with signs of neurotoxicity, such as brain hemorrhages, hyper-excitability, and over-extension reflexes, observed at or near 350 ppm. Subchronic inhalation studies have been conducted on rats, monkeys, and dogs (Ref. 19). Wistar rats (15 per sex per exposure level) were exposed to 0, 166, 330, and 655 ppm of acetonitrile for 7 hours a day for 5 days a week for 90 days. One out of five rat brains examined in the 655 ppm exposure group had focal cerebral hemorrhage. This effect was similar to that reported in Rhesus monkeys that were exposed to acetonitrile at 330, 660, and 2,510 ppm (approximately 28, 55, and 210 mg/kg/day) for 7 hours a day for up to 99 days. The monkey exposed to 2,510 ppm died with severe pulmonary effects after the second day of exposure, and the two monkeys exposed to 660 ppm died after 23 and 51 days, with severe brain hemorrhage and pulmonary abnormalities. The monkey exposed to 330 ppm acetonitrile exhibited unusual reflexes and excitability toward the end of the study. On gross examination, brain hemorrhage was also found in the monkey exposed to 330 ppm. Brain hemorrhages, hyper-excitability, and over-extension reflexes were also observed in three monkeys exposed to 350 ppm (approximately 30 mg/kg/day) of acetonitrile (Ref. 3). There were no signs of neurotoxicity reported for dogs.

In an embryo-fetal toxicity and teratogenicity study of acetonitrile, signs of neurotoxicity were found when acetonitrile was tested in the bred female New Zealand white rabbits receiving 2, 15, or 30 mg/kg/day by oral gavage (Ref. 20). Observations of dams at the high dose level showed neurological signs of ataxia, decreased motor activity, bradypnea, dyspnea, and impaired or lost righting reflex (Refs. 3 and 8).

Other laboratory studies also show that inhalation exposure to acetonitrile can adversely affect the nervous system of animals. In a report on acute exposure inhalation toxicity in rats submitted by E.I. du Pont de Nemours and Company (Refs. 3 and 21), toxicity was evaluated in groups of 10 male Sprague-Dawley rats exposed to acetonitrile for 4 hour periods. Dose levels and number of mortalities were not reported. Mortality was observed up to 24 hours post-exposure and the LC_{50} was determined to be 17,100 ppm. Clinical signs of neurotoxicity during exposure included irregular respiration, hyperemia followed by pale ears, face-pawing, and lack of coordination in all animals and unreactivity in decedents (Ref. 3).

In summary, subchronic exposures to acetonitrile can cause serious and irreversible health effects in animals at concentrations of acetonitrile at or near 350 ppm (approximately 30 mg/kg/day). Developmental studies in animals and acute inhalation studies in animals and exposures to humans provide additional support for the potential for acetonitrile

to cause severe neurological effects and even death in humans.

vi. *Other chronic effects*. Subchronic exposures of acetonitrile at concentrations ranging from 100 to 2,510 ppm (in several species) resulted in lung congestion and edema; increases in liver and kidney weight with swelling of the proximal and convulated tubules; cytoplasmic vacuolation of hepatocytes; brain hemorrhages; decreases in hemoglobin and hematocrit; severe eye irritation; decreases in thymus weight, increases in heart weight; and forestomach hyperplasia (Ref. 3). In addition, immunotoxic effects, such as a dose-dependent significant decrease in hematocrit, hemoglobin, red blood cells (RBC), white blood cells (WBC), and B-lymphocyte function, were observed in mice following inhalation exposure to acetonitrile (Refs. 3 and 22). There is uncertainty regarding the biological significance of the increases in relative liver weight, hepatic vacuolization, and some of the immunological changes observed after subchronic exposure since these effects were not seen following chronic dosing. It is possible that the lack of observed effects could be, however, the result of lower chronic exposure levels (Ref. 3). Chronic effects in rats and mice following chronic exposure to acetonitrile included increases in liver weights and forestomach lesions (Ref. 3). However, there is uncertainty regarding the biological significance of the forestomach lesions observed following inhalation exposure since oral exposure of acetonitrile as a result of the grooming of contaminated fur may also have been a contributing factor. Furthermore, it is difficult to assess the significance of the increases in liver weights without any information on the histopathological or functional changes (Ref. 3).

vii. *Toxicity related to ozone formation*. Acetonitrile is currently considered a VOC and, as such, has the potential to contribute to the formation of ozone in the troposphere (i.e., the lower atmosphere). As EPA has previously stated, ozone can affect structure, function, metabolism, pulmonary defense against bacterial infection, and extrapulmonary effects (Ref. 23). Among these extrapulmonary effects are: (1) Cardiovascular effects; (2) reproductive and teratological effects; (3) central nervous system effects; (4) alterations in red blood cell morphology; (5) enzymatic activity; and (6) cytogenetic effects on circulating lymphocytes. Accordingly, EPA has concluded that acetonitrile, as a VOC, has the potential to cause these effects.

3. *Ecotoxicity*. Acetonitrile is of low concern with respect to direct ecotoxicity based on measured data and Quantitative Structure Activity Relationship (QSAR) analysis. Acute acetonitrile toxicity for 96-hour fish and 48-hour daphnid exposures were 1,100 to 1,640 milligrams per liter (mg/L) (measured concentrations), and 4,900 mg/L, respectively (based on QSAR). Chronic acetonitrile toxicity for 21-day daphnid (reproduction) was greater than 200 mg/L (measured), and 470 mg/L for fish (based on QSAR) (Refs. 3 and 24).

Based on the limited number of laboratory studies conducted to date, the terrestrial toxicity of acetonitrile is low. No published experimental data are available for evaluating its bioaccummulation. Log bioconcentration factors for acetonitrile estimated using Lyman regression equations were −1.81 to 0.6 indicating no potential bioaccumulation (Refs. 3 and 25).

As a VOC, acetonitrile contributes to the formation of ozone in the environment. As EPA has previously stated (Ref. 23), ozone's effects on green plants include injury to foliage, reductions in growth, losses in yield, alterations in reproductive capacity, and alterations in susceptibility to pests and pathogens. Based on known interrelationships of different components of ecosystems, such effects, if of sufficient magnitude, may potentially lead to irreversible changes of sweeping nature to ecosystems.

D. Acute Exposure Assessment

Based on the results of animal studies, there are concerns for acute health effects associated with exposure to acetonitrile. Thus, pursuant to EPCRA section 313(d)(2)(A), EPA performed exposure assessments to determine whether acute health effects from acetonitrile would occur at concentrations reasonably likely to exist beyond the facility site boundaries as a result of continuous, or frequently recurring, releases. EPA's Toxic Release Inventory (TRI) release data were used to estimate acetonitrile exposures to the general population near the release sites. The fugitive emissions to air were the largest contributors to these exposures. Potential exposures due to water releases were also estimated.

1. *Ambient air exposure assessment*. Acetonitrile releases reported to TRI for 1995 and 1996 were used for the exposure assessment. Significant changes occurred between 1995 and 1996 with a greater than 50 percent increase in releases of acetonitrile occurring at the highest air releasing site. Short-term (acute exposure) air concentrations were estimated using the SCREEN3 and ISCST3 models. Among the ten top sites chosen for modeling, a plant in Memphis, Tennessee had the highest air releases for both 1995 and 1996, dominated by fugitive air releases. Using the SCREEN3 model, the estimated air concentrations of acetonitrile beyond facility site boundaries at sites with fugitive air emissions greater than 10,000 kilograms per year (kg/year) for 1995 and 1996 ranged from 4 to 36 milligrams per cubic meter (mg/m^3) (2.4 to 22 ppm) for 1 hour, and 1 to 14 mg/m^3 (0.9 to 8 ppm) for 24 hours, respectively.

Based on the 1995 data and the ISCST3 model, the 1 and 24 hours short-term (acute exposure) acetonitrile concentrations in air, at 100 meters distance from the source center of highest release, in the direction of highest concentration, are 16 and 2.3 mg/m^3 (or 9.52 and 1.37 ppm), respectively. Under the same model scenario, the 1996 data gave an estimated 23 and 3.3 mg/m^3 (or 13.5 and 2.0 ppm) of acetonitrile concentrations in air for the 1 and 24 hour short-term exposure, respectively. Other air concentrations of acetonitrile for ten top facilities were also modeled and the estimated data are summarized in the General Sciences Corporation (GSC) modeling support for exposure assessment of acetonitrile (Ref. 26). The highest estimates were at those facilities with boundaries of approximately ¼ mile (400 meters) from the site center or less (Refs. 4 and 26).

The short-term air modeling was intended to represent acute exposure scenarios for populations spending time in the surroundings of facilities, outside site boundaries, but not necessarily resident. However, the results should be considered ''what-if'' rather than established as high end, because of factors such as variability in meteorology, and uncertainties in release quantities and durations. It is important to recognize that the ambient air concentration estimates use the assumption that releases continue over 365 days per year, 24 hours per day at a constant rate. If annual releases occurred over shorter time periods, the corresponding short-term concentrations would be higher than those presented in the exposure assessment report. For example, if a facility releases approximately 10,000 kilograms of fugitive air releases per year over 30 days per year rather than 365 days per year, then the upper limit of the screening range would exceed 40 mg/m^3, exceeding the value (36 mg/m^3) shown for the highest release of more than 200,000 pounds per year. The

concentrations estimated show a screening range (using SCREEN3 model at a distance of 100 meters from the source center) and provide key results for selected sites. The data also shows maximum results beyond facility boundaries, using distances from site centers indicated by site layouts in the industry report (Refs. 4 and 27). These estimated values of acetonitrile in air are well below those concentration levels that produced acute effects in animal studies.

2. *Drinking water exposure assessment*. Both direct and indirect releases to water were modeled using river reach harmonic mean flows for long-term and low flow data for short term. The REACHSCAN model was used to estimate the contamination of acetonitrile at drinking water utility intakes downstream from facilities releasing to water or making offsite transfers to waste-water treatment facilities. While some locations have low to mid parts per billion (ppb) levels, few intake locations of drinking water utilities have levels above 1 ppb (1 microgram per liter). Based on 1995 TRI water release data, the highest exposure potential with drinking water intakes downstream were found for an indirect discharger in Pennsylvania, with annual concentration of 100 ppb and the short-term concentration of 350 ppb. However, that facility changed reporting from ''transfers to publicly owned treatment works (POTWs)'' to ''other offsite transfers'' for 1996; several other facilities also reduced or ended water releases or transfers to POTWs for 1996. The highest drinking water utility intake level found using 1996 TRI data was approximately 2 ppb for low flow conditions, and 0.7 ppb for typical conditions (downstream from a facility in Rock Hill, South Carolina). Several fresh-water locations without verified drinking water intakes have mid ppb (e.g., 200 ppb) estimated levels (Ref. 4).

Some potential drinking water situations have not been quantified due to lack of data. For example, offsite transfers to POTWs include several sites in Puerto Rico, for which surface water data have not been retrieved. Underground injection wells also may form sources of contamination to drinking water wells in ground water, in the event of containment failure (Ref. 4). Atmospheric deposition of acetonitrile can also contribute to surface water contamination near facilities releasing to air (Ref. 4).

3. *Exposure evaluation*. EPA's exposure assessment attempted to determine whether, as a result of releases from EPCRA section 313 covered facilities, acetonitrile is known to cause or can reasonably be anticipated to cause significant adverse acute human health effects at concentration levels that are reasonably likely to exist beyond facility site boundaries as a result of continuous or frequently recurring releases. The modeling used released data reported under EPCRA section 313 and included both conservative and non-conservative assumptions concerning releases and facility site information. Non-conservative assumptions included the assumption that EPCRA section 313 reported releases are spread over 365 days per year and 24 hours per day. Given a shorter release period, estimated exposures could be significantly higher. Under the conditions modeled here EPA believes it is unlikely that concentrations of acetonitrile sufficient to cause acute toxicity will exist beyond a facility's boundaries as a result of continuous, or frequently reoccurring, releases. This is because the exposure concentrations that resulted from the modeling (9.52 and 1.37 ppm) are below the concentrations that have caused acute toxicity in laboratory animals (500 ppm).

V. Summary of Technical Review

There is sufficient evidence to support a high level of concern for potential neurotoxicity and death following repeated exposure to acetonitrile. This comes from several lines of evidence. In repeated dose (subchronic) inhalation experiments in monkeys, neurological signs of toxicity (brain hemorrhages, hyper-excitability, and over-extension reflexes) and death were observed at concentrations of acetonitrile at or near 350 ppm (approximately 30 mg/kg/day). For effects seen in both the monkey and rabbit studies, the neurotoxicity risk assessment guidelines recommend that these endpoints be included as examples of possible indicators of an adverse neurotoxic effect (Ref. 28). Structural or neuropathological endpoints could include hemorrhage in nerve tissue. Neurological endpoints could include increases or decreases in motor activity and changes in motor coordination. When pregnant rabbits were exposed to the same amount of acetonitrile during gestation, signs of neurotoxicity (including ataxia (muscle incoordination), decreased motor activity, bradypnea (abnormally slow breathing), dyspnea (labored or difficult breathing), and impaired or lost righting reflex) and an increased incidence of maternal mortality were also observed. These effects are consistent with acute inhalation exposures to high concentrations of acetonitrile in humans in which the central nervous system is widely affected (exhibiting signs of salivation, nausea, vomiting, anxiety, confusion, hyperpnea, dyspnea, rapid pulse, unconsciousness, and convulsions followed by death from respiratory failure). The neurological effects seen in the developmental and acute studies provide supplemental support for the determination that acetonitrile can reasonably be anticipated to cause chronic neurotoxicity. These results are also consistent with those effects seen with inorganic cyanide and other aliphatic nitriles exposures, suggesting that the toxic effects of acetonitrile may be correlated with the metabolic release of cyanide.

Acetonitrile is currently considered a VOC and, as such, it contributes to the formation of tropospheric ozone which, as EPA has previously determined, can cause significant adverse effects to human health and the environment (Ref. 23).

The main effects of acetonitrile reported in humans (from accidental poisoning) are likely due to acute inhalation exposures to high concentrations. Based on the results of animal studies, there are concerns for acute health effects associated with exposure to acetonitrile. However, based on EPA's exposure assessment, it is unlikely that concentrations of acetonitrile, sufficient to cause acute toxicity, will exist beyond a facility's boundaries as a result of continuous, or frequently recurring, releases. There is not sufficient information to support a concern for carcinogenicity, mutagenicity, or reproductive toxicity. The case for developmental toxicity is weak. Some studies in rats produced no signs of developmental toxicity even in the presence of maternal toxicity. Other studies exhibited signs of developmental toxicity, however, in the presence of extreme maternal mortality. There is uncertainty regarding the biological significance of the increases in relative liver weight, hepatic vacuolization, and some of the immunological changes observed after subchronic exposure since these effects were not seen following chronic dosing. It is possible that the lack of observed effects could be, however, the result of lower chronic exposure levels. Acetonitrile is of low concern with respect to direct ecotoxicity based on measured data and QSAR analysis.

VI. Rationale for Denial

EPA is denying the petition submitted by BP and GNICC to delete acetonitrile from the EPCRA section 313 list of toxic

chemicals. This denial is based on EPA's conclusion that acetonitrile can reasonably be anticipated to cause serious or irreversible chronic health effects in humans, including neurotoxicity and death. Chronic health effects may result after acute, subchronic, or chronic exposures. EPA determines whether an effect is best considered to be chronic by looking at a number of factors, among which is the length of time it takes for the effect to manifest and the extent to which it persists after exposure to the toxicant ends. Acute or subchronic exposure to acetonitrile can produce serious and irreversible health effects, including brain hemorrhages and death. In addition, acute or subchronic exposure to acetonitrile produce the following serious health effects: Hyper-excitability, over-extension reflexes, ataxia (muscle incoordination), decreased motor activity, bradypnea (abnormally slow breathing), dyspnea (labored or difficult breathing), and impaired or lost righting reflex. Many of these effects (e.g., over-extension reflexes and hyper excitability) manifest toward the end of the exposure period and are thus considered chronic effects. Data from animal studies indicate that neurotoxicity and death can occur at the relatively low dose of approximately 30 mg/kg/day. Based on these data, EPA considers acetonitrile to have moderately high to high chronic toxicity. Therefore, EPA has concluded that acetonitrile meets the listing criteria of EPCRA section 313 (d)(2)(B).

EPA has concluded that acetonitrile meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) due to it contributing to the formation of ozone. EPA has concluded that VOCs, such as acetonitrile, contribute to the formation of tropospheric ozone which is known to cause significant adverse effects to human health and the environment. EPA has previously stated that ozone meets the listing criteria of EPCRA section 313(d)(2)(B) and (d)(2)(C) (59 FR 61432, November 30, 1994). EPA has stated in prior **Federal Register** notices (54 FR 4072, January 27, 1989; 54 FR 10668, March 15, 1989; 59 FR 49888, September 30, 1994; 60 FR 31643, June 16, 1995; and 63 FR 15195, March 30, 1998) that, because VOCs contribute to the formation of tropospheric ozone, they meet the criteria for listing under EPCRA section 313. EPA has also stated (54 FR 4072, January 27, 1989 and 54 FR 10668, March 15, 1989) that while it is not EPA's intention to include all VOC chemicals on the EPCRA section 313 list, those VOCs whose volume of use or emissions are large enough to raise substantial VOC concerns would be retained on the EPCRA section 313 list. Acetonitrile is a VOC with a high production volume, and therefore, EPA has determined that acetonitrile should remain on the EPCRA section 313 list of toxic chemicals. In EPA's most recent petition denial based on VOC concerns (63 FR 15195, March 30, 1998), the Agency provided further explanation concerning its rationale for determining that indirect effects, such as those caused by VOCs, meet the EPCRA section 313 listing criteria.

Because EPA believes that acetonitrile has moderately high to high chronic toxicity, EPA does not believe that an exposure assessment is appropriate for determining whether acetonitrile meets the criteria of EPCRA section 313(d)(2)(B). This determination is consistent with EPA's published statement clarifying its interpretation of the section 313(d)(2) and (d)(3) criteria for modifying the section 313 list of toxic chemicals (59 FR 61432, November 30, 1994).

As mentioned under Unit III. of this preamble, the petitioner's have submitted a petition to EPA's OAR to add acetonitrile to the list of negligibly photoreactive chemicals under 40 CFR 51.100(s)(1). Chemicals that appear on this list are excluded from EPA's definition of a VOC, since they have been determined to have a negligible contribution to tropospheric ozone formation. OAR's initial review of the petition indicates that acetonitrile may be a negligibly photoreactive chemical (Ref. 29). If OAR's initial assessment is confirmed and a rule is issued that adds acetonitrile to the list of negligibly photoreactive chemicals under 40 CFR 51.100(s)(1), then any concerns based solely on acetonitrile being listed as a VOC would no longer be a basis for listing acetonitrile under EPCRA section 313. However, since EPA has also concluded that acetonitrile meets the EPCRA section 313 criteria for listing based on concerns for chronic neurotoxicity, EPA's decision to deny the petition to delete acetonitrile from the EPCRA section 313 list of toxic chemicals would not be affected by a change in acetonitrile's status as a VOC.

VII. References

1. British Petroleum, Inc. (BP) and GNI Chemicals Corporation (GNICC), 1998. Petition to Delist Acetonitrile from the List of Toxic Chemicals under the Authority of Section 313 of the Emergency Planning and Community Right-to-Know Act (EPCRA) of 1986. BP Chemicals Inc. and GNI Chemicals Corp. (February 1998).

2. United States Environmental Protection Agency (U.S. EPA), Office of Pollution Prevention and Toxics (OPPT), Tou, Jenny, 1998. Chemistry Report for Acetonitrile in Response to EPCRA Section 313 Delisting Petition. (June 1998).

3. USEPA, OPPT, Anitole, Katherine, 1998. Hazard Assessment for Acetonitrile in Response to Section 313 Delisting Petition of BP Chemicals Inc. and GNI Chemicals Corporation. (June 1998).

4. USEPA, OPPT, Nold, Annett, 1998. Exposure Assessment for Acetonitrile in Response to Delisting Petition. (July 1998).

5. USEPA, OPPT, Miller, Jim, 1998. Economic Analysis of the Proposed Deletion of Acetonitrile from the EPCRA Section 313 List of Toxic Chemicals. (March 1998).

6. USEPA, OPPT, Blouin, John, 1998. Engineering Analysis of the Proposed Deletion of Acetonitrile from the EPCRA Section 313 List of Toxic Chemicals. (April 1998).

7. The Merck Index. An Encyclopedia of Chemicals, Drugs, and Biologicals, 11th Edition, p.63, 1989, Merck Co., Inc.

8. World Health Organization (WHO), 1993. International Program on Chemical Safety (IPCS), Environmental Health Criteria 154 - Acetonitrile, World Health Organization.

9. National Cancer Institute (NCI)/ National Toxicological Program (NTP), 1996. NTP Technical Report of the Toxicology and Carcinogenesis of Acetonitrile (CAS No. 75–05–8) in F344/N Rats and B6C3F1 Mice (inhalation studies). Carcinogenesis Technical Report Series; National Cancer Institute/National Toxicology Program; U.S. Department of Health and Human Services (U.S. HHS).

10. Taskinen, H., Kyyronen, P., Hemminki, K., Hoikkala, M., Lajunen, K., and Lindbohm, M., 1994. Laboratory Work and Pregnancy Outcome. *JOM* 36:311–319.

11. USEPA, OPPT, 1998. Hazard Assessment Emphasizing Epidemiological Data for the Acetonitrile Delisting Petition. Memorandum dated March 13, 1998, from Andrea Pfahles-Hutchens, Existing Chemicals Assessment Branch, to Katherine Anitole, Existing Chemicals Assessment Branch, RAD. Washington, DC: USEPA.

12. USEPA, OPPT, 1998, Health Effects Test Guidelines OPPTS 870.3700: Prenatal Development Toxicity Study, (EPA 712–C–98–207, August 1998).

13. Willhite, C. 1983. Developmental Toxicity of Acetonitrile in the Syrian

Golden Hamster. *Teratology* 27, 313–325.

14. Smith, M.K., George, E.L., Zenick, H., Manson, J.M., and Stober, J.A. 1987. Developmental Toxicity of Halogenated Acetonitriles: Drinking Water By-Products of Chlorine Disinfection. *Toxicol.* 46, 83–93.

15. Johannsen, F.R., G.L., Levinskas, P.E., Berteau and D.E. Rodwell. 1986. Evaluation of the Teratogenic Potential of Three Aliphatic Nitriles in the Rat. *Fund. Appl. Toxicol.* 7, 33–40.

16. Morrissey, R.E., Schwetz, B.A., Lamb, J.C., Ross, M.Dd., Teague, J.L. and Morris, R.W. 1988. Evaluation of Rodent Sperm, Vaginal Cytology, and Reproductive Organ Weight Data from National Toxicology Program 13-Week Studies. *Fund. Appl. Toxicol.* 11, 343–358.

17. Hazardous Substances Data Bank (HSDB), 1994. MEDLARS Online Information Retrieval System, National Library of Medicine. (Retrieved August, 1994).

18. IRIS. U.S. Environmental Protection Agency's Integrated Risk Information System (IRIS).

19. Pozzani, U.C., Carpenter, C.P., Palm. P.E., Weil, C.S. and Nair, J.H. 1959. An Investigation of the Mammalian Toxicity of Acetonitrile. *J. Occup. Med.* 12, 634–642.

20. Argus Research Laboratories, Inc., 1984. Embryo-fetal Toxicity and Teratogenicity Study of Acetonitrile in New Zealand White Rabbits (Segment II Evaluation). Argus Research Laboratories, Inc. Project No. 419–001, Final Report. EPA Document No. 40–8446070, Fiche No. OTS0507279.

21. E.I. du Pont de Nemours Company, 1968. Acute Inhalation Toxicity in Rats with Cover Letter, Haskell Laboratory for Toxicology and Industrial Medicine. EPA Document No. 878220234, Fiche No. OTS0215023.

22. Immunquest (1984). EPA Docket Office: FYI–OTS–0284–0292.

23. USEPA. ''Addition of Certain Chemicals.'' Proposed rule, (59 FR 1788, January 12, 1994).

24. USEPA, OPPT, 1994. Estimating Toxicity of Industrial Chemicals to Aquatic Organisms using Structure-Activity Relationships. (May 1994)

25. Screening Information Data Set (SIDS)/Organization for Economic Cooperation and Development (OECD), 1997. SIDS Initial Assessment Profile (Draft) on Acetonitrile. (March 1997).

26. General Sciences Corporation (GSC), 1998. ''Modeling Support for Exposure Assessment of Acetonitrile,'' prepared for Exposure Assessment Branch, OPPT. EPA Contract No. 68–W7–0030, Work Assignment No. II–1. (Final Report for Acetonitrile, July 1998).

27. Trinity Consultants (Dallas, Texas), 1998. ''Refined Modeling for Ten Acetonitrile Emitting Facilities,'' prepared for BP Chemicals and GNI Group, Inc., (January 20, 1998), Project 97440.0200.

28. USEPA. ''Proposed Guidelines for Neurotoxicity Risk Assessment.'' Notice, (60 FR 192, October 4, 1995).

29. USEPA. ''Photochemical Reactivity of Acetonitrile.'' Memorandum from G. T. Helms to Maria Doa, (October 1, 1998).

List of Subjects in 40 CFR Part 372

Environmental protection, Chemicals, Community right-to-know, Hazardous substances, Intergovernmental relations, Reporting and recordkeeping requirements, Superfund, Toxic chemicals.

Dated: February 24, 1999.

Susan H. Wayland,

Acting Assistant Administrator for Prevention, Pesticides and Toxic Substances.

[FR Doc. 99–5495 Filed 3–4–99; 8:45 am]

BILLING CODE 6560–50–F

DEPARTMENT OF TRANSPORTATION

National Highway Traffic Safety Administration

49 CFR Part 571

[DOT Docket No. NHTSA–99–5157]

RIN 2127–AH03

Federal Motor Vehicle Safety Standards; Bus Emergency Exits and Window Retention and Release

AGENCY: National Highway Traffic Safety Administration (NHTSA), DOT.

ACTION: Notice of proposed rulemaking.

SUMMARY: In this document, NHTSA proposes to amend the Federal Motor Vehicle Safety Standard on bus emergency exits and window retention and release by regulating the location of the anchorages for wheelchair securement devices. NHTSA is issuing this proposal to ensure that wheelchair securement anchorages and devices cannot be installed, and wheelchairs cannot be secured, in locations where they will block access to any exit needed for school bus evacuation in the event of an emergency. This proposal applies to school buses in which wheelchair positions are provided. Nothing in this rulemaking would require that wheelchair positions be provided.

DATES: You should submit your comments early enough to ensure that Docket Management receives them not later than May 4, 1999.

ADDRESSES: You should mention the docket number of this document in your comments and submit your comments in writing to: Docket Management, Room PL–401, 400 Seventh Street, S.W., Washington, D.C., 20590.

You may call the Docket at 202–366–9324. You may visit the Docket from 10:00 a.m. to 5:00 p.m., Monday through Friday.

FOR FURTHER INFORMATION CONTACT: For non-legal issues, you may call Mr. Charles Hott, Office of Crashworthiness Standards at (202) 366–0247. His FAX number is (202) 493–2739.

For legal issues, you may call Ms. Dorothy Nakama, Office of the Chief Counsel at (202) 366–2992. Her FAX number is (202) 366–3820.

You may send mail to both of these officials at National Highway Traffic Safety Administration, 400 Seventh St., S.W., Washington, D.C., 20590.

SUPPLEMENTARY INFORMATION:

Background

NHTSA has long recognized the safety need for school buses to provide means for readily accessible emergency egress in the event of a crash or other emergency. The agency addressed this safety need by issuing Safety Standard No. 217, *Bus Emergency Exits and Window Retention Release* (49 CFR Section 571.217). Standard No. 217 includes emergency exit requirements for school buses. The standard requires that all new school buses have either (1) one rear emergency door, or (2) one emergency door that is located on the vehicle's left side, in the rear half of the bus passenger compartment, and that is hinged on its forward side and one push-out rear window. (See S5.2.3.1)

As a result of incidents like the 1988 Carrollton, Kentucky, tragedy, in which 27 persons died in a school bus fire following a crash, NHTSA amended Standard No. 217 (November 2, 1992, 57 FR 49413) by revising the minimum requirements for school bus emergency exits, requiring additional emergency exit doors on school buses, and improving access to school bus emergency doors. In the final rule, the agency stated that the preferred method of providing access to side emergency exit doors was through creating a dedicated aisle, and thus, S5.4.2.1(2) and Figures 5B and 5C were added to the standard to require a 30 centimeter (12 inch) wide aisle to provide access to side emergency exit doors.

In a final rule published on January 15, 1993 (58 FR 4586), NHTSA amended