MICHIGAN DEPARTMENT OF ENVIRONMENTAL QUALITY

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INTEROFFICE COMMUNICATION

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November 30, 2021

TO: Files for: Kerosene and Kerosene-like Petroleum Distillates

Kerosene	CAS No. 8008-20-6
Deodorized kerosene (Deobase)	CAS No. 8020-83-5
Hydrodesulfurized kerosene	CAS No. 64742-81-0
Acid treated light distillates	CAS No. 64742-14-9
Chemically neutralized light distillates	CAS No. 64742-31-0
Hydrotreated light distillates	CAS No. 64742-47-8
Heavy aliphatic solvent naphtha	CAS No. 64742-96-7
Jet Fuels	CAS No. not available

FROM: Michael Depa, Toxics Unit, Air Quality Division

SUBJECT: Screening Level Derivation

The acute initial threshold screening level (ITSL) for kerosene-like petroleum distillates is 2000 µg/m³, with 8-hour averaging time. The chronic ITSL for kerosene-type petroleum distillates is 200 µg/m³, with annual time.

A footnote is associated with the ITSL for kerosene and kerosene-type petroleum distillates:

> The combined impact of all kerosene-type petroleum distillates cannot exceed the acute ITSL of 2000 µg/m³, with 8-hour averaging time, and the chronic ITSL of 200 µg/m³, with annual averaging time. All kerosene ambient impacts are to be combined with the impacts of all petroleum hydrocarbon materials with footnote No. 1 and the total of these impacts cannot exceed the ITSL of 3500 µg/m³ with 8-hour averaging time.

The previous ITSL of 24 µg/m³ with annual averaging time is being replaced for the following kerosenes: Deodorized Kerosene (CAS No. 8020-83-5), Petroleum Distillates Acid Treated (CAS No. 64742-14-9), Hydrotreated Light Distillate (CAS No. 64742-47-8), Solvent Naphtha (Petroleum) Heavy Aliphatic (CAS No. 64742-96-7). The previous ITSL of 4 µg/m³ with annual averaging time is being replaced for the following kerosene: Hydrodesulfurized Kerosene (CAS No. 64742-81-0).

Background

EPA (2011) describes a kerosene category rather that one specific distillate:

> While kerosenes are similar in composition, the precise composition of a specific kerosene-range refinery stream depends on the grade of crude oil from which the kerosene was derived and on the refinery processes used for its production. Because they are complex petroleum derived hydrocarbons, substances in this category are typically not defined by detailed compositional data but instead by process history, physical properties, and product-use specifications. Regardless of the crude oil source or processing history, the major components of all kerosenes are branched and straight chain paraffins and naphthenes (i.e., cycloparaffins), which normally account for at least 70% by volume. Aromatic hydrocarbons in this boiling range, such as alkylbenzenes (single ring) and alkylnaphthalenes (double ring) do not normally exceed 25% by volume of kerosene streams. Olefins (i.e., alkenes) are usually not present at more than 5% by volume.

The composition of these eight petroleum distillates were examined and deemed to fit the general definition of kerosenes:

Kerosene	CAS No. 8008-20-6
Deodorized Kerosene	CAS No. 8020-83-5
Hydrodesulfurized kerosene	CAS No. 64742-81-0
Acid treated light distillate, light	CAS No. 64742-14-9
Chemically neutralized light distillates	CAS No. 64742-31-0
Hydrotreated light distillates	CAS No. 64742-47-8
Heavy aliphatic solvent naphtha	CAS No. 64742-96-7
Jet Fuels	CAS No. not available

The following references or databases were searched to identify data to determine the screening level: U.S. Environmental Protection Agency (EPA) Integrated Risk Information System (IRIS), Registry for Toxic Effects of Chemical Substances (RTECS), American Conference of Governmental and Industrial Hygienists (ACGIH) Threshold Limit Values (TLVs), National Institute for Occupational Safety and Health (NIOSH) Recommended Exposure Levels (RELs), International Agency for Research on Cancer (IARC) Monographs, Chemical Abstract Service (CAS) SciFinder, CompTox Chemicals Dashboard, Provisional Peer-Reviewed Toxicity Values, ChemID*plus*, and the National Toxicology Program (NTP). The EPA has not established a reference dose (RfD) or a reference concentration (RfC) for kerosene. California's Office of Environmental Health Hazard Assessment has not derived a Recommended Exposure Limit for kerosene.

Summary of Regulatory Standards for Kerosene

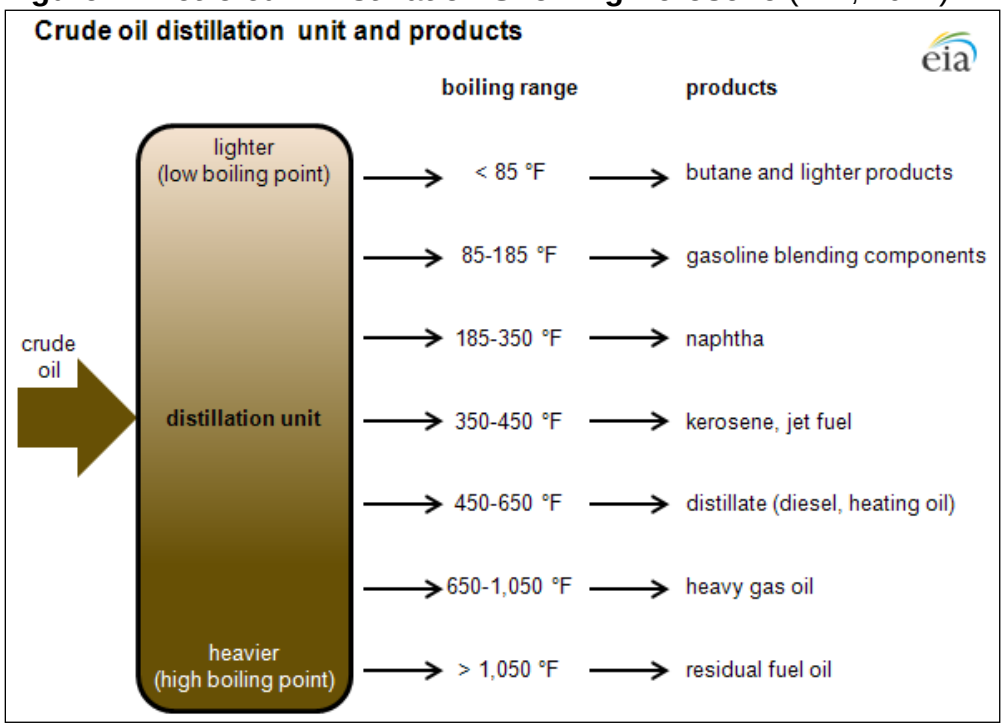
The EPA has derived Acute Exposure Guideline Levels (AEGLs) for kerosene-based jet fuels: the AEGL-1 (nondisabling) for JP-5 and JP-8 is 290 mg/m^3 for all time periods (i.e., 10-minute, 30-minute, 1-hour, 4-hour, and 8-hour) (National Research Council, 2011). The ACGIH (2017) TLV time weighted average (TWA) for kerosene (including jet fuels) is 200 mg/m^3. The NIOSH REL for kerosene is 100 mg/m^3. The Agency of Toxic Substances and Disease Registry (ATSDR, 2017) has derived intermediate duration minimal risk levels (MRLs) for kerosene-based jet fuels, JP-5 and JP-8 at 2 mg/m^3 and 3 mg/m^3, respectively.

Kerosene Description

The substance called “kerosene” is a somewhat general term for a category of petroleum derived distillates that have similar composition and closely related physical properties. EPA (2011) states that, “Kerosenes are the lighter end of a group of petroleum substances known as middle distillates.” ACGIH (2003) reports that the average molecular weight of kerosene is 170g, based on the range of components that contain hydrocarbon lengths of approximately C9 to C16. While kerosenes are similar in composition, the precise composition of a specific kerosene-range refinery stream depends on the crude oil from which the kerosene was derived, and on the refinery processes used for its production (API, 2010).

As shown in Figure 1 (below), kerosene is derived from crude oil using fractional distillation, and can be distinguished from other petroleum distillates by boiling point range. Because kerosenes are complex petroleum derived hydrocarbons, substances in this category are typically not defined by detailed compositional data, but instead by process history, physical properties, and product-use specifications (API, 2010). Kerosene can also be spelled kerosine.

Figure 1. Petroleum Distillation Showing Kerosene (EIA, 2012)



The most common CAS Number for kerosene is 8008-20-6. The American Chemical Society, which creates CAS numbers, defines kerosene (CAS No. 8008-20-6), as:

> Straight Run, Kerosene (petroleum). A complex combination of hydrocarbons produced by the distillation of crude oil. It consists of hydrocarbons having carbon numbers predominantly in the range of C9 through C16 and boiling in the range of approximately 180°C to 300°C (356°F to 572°F).

Hydrodesulfurized kerosene (CAS No 64742-81-0) is defined by CAS as:

> A complex combination of hydrocarbons obtained from a petroleum stock by treating with hydrogen to convert organic sulfur to hydrogen sulfide which is removed. It consists of hydrocarbons having carbon numbers predominantly in the range of C9 through C16 and boiling in the range of approximately 150°C to 290°C (302°F to 554°F).

Two sub-categories of kerosene have been described by the U.S. Energy Information Administration (EIA, 2017), which differ slightly by test methods described by ASTM (American Society for Testing and Materials) Specification:

> Kerosene: A light petroleum distillate that is used in space heaters, cook stoves, and water heaters and is suitable for use as a light source when burned in wick-fed lamps. Kerosene has a maximum distillation temperature of 400 degrees Fahrenheit at the 10-percent recovery point, a final boiling point of 572 degrees Fahrenheit, and a minimum flash point of 100 degrees Fahrenheit. Included are No. 1-K and No. 2-K, the two grades recognized by ASTM Specification D 3699 as well as all other grades of kerosene called range or stove oil, which have properties similar to those of No. 1 fuel oil. Also see Kerosene-type jet fuel.
>
> Kerosene-type jet fuel: A kerosene-based product having a maximum distillation temperature of 400 degrees Fahrenheit at the 10-percent recovery point and a final maximum boiling point of 572 degrees Fahrenheit and meeting ASTM Specification D 1655 and Military Specifications MIL-T-5624P and MIL-T-83133D (Grades JP-5 and JP-8). It is used for commercial and military turbo jet and turbo prop aircraft engines.

The ASTM specifications for kerosene and kerosene-type jet fuel, “ASTM D3699 - 18A Standard Specification for Kerosene” (ASTM, 2019a) and “ASTM D1655 - 18b Standard Specification for Aviation Turbine Fuels” (ASTM, 2019b), respectively, list detailed physical and chemical test results that must be adhered to in order to be formally named these kerosene subtypes. Table 1 provides details on properties of jet fuels.

Table 1. Various Properties of Jet Fuels

Property	JP-4	JP-5	JP-7	JP-8 (Jet A/A-1)
Approximate Formula	$C_{8.5}H_{17}$	$C_{12}H_{22}$	$C_{12}H_{25}$	$C_{11}H_{21}$
Boiling Range (°F)	140-460	360-495	370-480	330-510
Freeze point (°F)	-80	-57	-47	-60 JP-8/Jet A-1; -50 Jet A
Flash Point (°F)	-10	147	140	127
Avg. Composition				
Aromatics, vol%	10	19	3	18
Paraffins	59	45	65	60
Naphthenes	29	34	32	20
Olefins	2	2	-	2
Sulfur, ppm[1]	370	470	60	490

From Edwards and Maurice (2001)

[1] ppm = parts per million

The ACGIH (2003) documentation for kerosene defines kerosene more generally by providing ranges for various chemical and physical values:

> Kerosene and other jet fuels are mixtures of petroleum hydrocarbons with broadly overlapping chemical composition, closely related physical properties, and generally similar toxicological effects. They are mixtures of hydrocarbons consisting of paraffinic[2] (33%-61%) and naphthenic (10%-45%) alkanes, aromatics (12%-25% maximum), and olefins (0.5%-5% maximum) with varying chemical composition depending on their crude oil or blending stock origin.
>
> The maximum final boiling point of kerosene and other middle distillate fuels tends to exclude the presence of high-boiling (3-7 ring) polycyclic aromatic hydrocarbons (PAHs). Diesel No. 1 is for all intents and purposes kerosene. These fuels are naturally brown to straw-colored mobile liquids of rather low volatility. Kerosene can be deodorized and decolorized to a water-white liquid (white gas) by treatment with fuming sulfuric acid and then washing with sodium plumbite and sulfur. The air odor threshold for deodorized kerosene is 0.6 mg/m³ (0.1 ppm). Olfactory fatigue can develop within 15 minutes of inhaling 140 mg/m³ (20 ppm). Chemical and physical properties of kerosene and commercial jet fuels include:
>
> Average molecular weight: 170 (range of components approximately C9-C16)
> Specific gravity: 0.81 to 0.95 g/ml at 15° to 20°C
> Melting point: -34°C
> Boiling point: 175° to 325°C
> Vapor pressure: 2.1 to 2.6 torr at 21 °C
> Flash point: approximately 38° to 52°C (closed cup)
> Autoignition temperature: 177° to 329°C
>
> Commercial Jet A-1 has a slightly lower freeze point than Jet A. The military fuels, JP-5 and JP-8, are kerosene-derived and similar to Jet A-1. The military jet fuels contain additives, including a fuel system icing inhibitor, corrosion inhibitor, and a static dissipater, not found in the commercial Jet A / A-1. A complex mixture of hydrocarbons such as kerosene or jet fuels having a relatively low vapor pressure and variable molecular weight is best characterized by a TLV expressed in mass units. The low volatility means that some exposures could be to aerosol and vapor if considerable spray is generated.

Additional details on the composition of kerosene come from several other organizations that publish occupational exposure limits for protection of public health in the workplace. NIOSH (2019) defines kerosene as:

> A refined petroleum solvent (predominantly C9-C16), which typically is 25% normal paraffins, 11% branched paraffins, 30% monocycloparaffins, 12% dicycloparaffins, 1% tricycloparaffins, 16% mononuclear aromatics and 5% dinuclear aromatics.

For purposes of assessing the toxicity of kerosene, EPA (2011) defines kerosene as a category:

> [A]ll the substances in this [kerosene] category are liquids with low vapor pressures. The substances …share many physical properties that make

[2] Paraffins are straight and branched alkanes, naphthenic compounds are cyclic alkanes, and olefins are straight and branched alkenes.

> them suitable as blending components for finished fuels such as aviation turbine fuels (jet fuels), No. 1-K kerosene (for heating and illumination), and No. 1 grades of fuel oil, diesel fuel, and gas turbine oil.

Justification for grouping various petroleum distillates as "kerosene" is supported by EPA (2011) where EPA states that, "the physical and chemical similarities among the streams in this [kerosene] category allow toxicology data on one material to be extrapolated to the others."

Additives in Commercial Kerosene

Kerosene-based fuels differ from each other in performance specifications (primarily freezing point or sulfur concentration), and minor amounts of performance additives that may be added. The Conservation of Clean Air and Water in Europe (CONCAWE, 1999) describes the kerosene additives in these terms:

> A marketed fuel may contain additives and those that are commonly used are antioxidants, anti-static additives, corrosion inhibitors, fuel system icing inhibitors, metal de-activators and biocides. The total concentration of these additives is generally less than 0.1% (m/m).

The U.S. Department of Defense (DOD, 2011) has fuel standards for jet fuel additives that include a Fuel system icing inhibitor (FSII):

> Diethylene glycol monomethyl ether (DIEGME) with a flash point of 85 °C (185 °F) has been identified as the type of FSII to be purchased for all jet fuels. FSII lowers the freeze point of entrained or free water present in turbine fuels or in fuel systems. The amount of FSII added to turbine fuels in the wholesale system be adjusted to ensure delivery of the fuel with a minimum FSII content of 0.10%, volume, for all grades of turbine fuels.

Carcinogenic Properties of Kerosene

There is no indication in animal studies or workplace exposure studies that kerosene causes cancer via inhalation (Ritchie, 2003). However, there is evidence of carcinogenicity via the dermal route of exposure. The following are excerpts from "Robust Summary of Information on Kerosene/Jet Fuel) (API, 2010):

> Straight-run kerosine (CAS # 8008-20-6) and hydrodesulfurised kerosine (CAS # 64742-81-0) were tested [dermally] in standard 2-year bioassays in C3H/HeJ mice (API, 1989a; API, 1989b). The animals, 50 per group, were treated twice weekly with 50 µl straight-run kerosine or with hydrodesulfurised kerosine. As positive controls, two groups of 50 mice were exposed twice weekly to 50 µl of 0.01% or 0.05% benzo(a)pyrene in toluene, respectively. Two negative control groups received either 50 µl toluene or no treatment. Animals in all groups but the negative control group with no treatment at all showed dermal irritation, but the dermal lesions in the toluene control group were less than in the test group. In all test groups dermal tumors developed. The mean latency time for tumor formation in the straight-run kerosine as well as the hydrodesulfurised kerosine treated group was with 77 and 76 weeks, respectively, significantly longer than the 47 weeks observed in the 0.05% benz(a)pyrene treated group. The number of tumors, 1 benign and 29 malignant, in the 45 surviving animal in the straight-run kerosine treated mice and 1 benign and 19 malignant, in the 41 surviving mice was also significantly less than in the benzo(a)pyrene treated groups (11 benign and 13 malignant tumors in the 0.01% benzo(a)pyrene group and 2 benign and 44 malignant in the 0.05% benzo(a)pyrene group) whilst the toluene group showed no dermal tumors at all (44 mice surviving). It was concluded that both straight-run and hydrodesulfurised kerosine were moderate skin carcinogens.

In the key carcinogenicity study from NTP (Klimisch score=1; NTP, 1986), JP-5 navy fuel in acetone was administered [to the skin] to 50 B6C3F1 mice/sex/dose dermally at dose levels of 0 (vehicle control), 250, or 500 mg/kg bw/day for up to 103 weeks. There was a significant decrease in survival in females at both treatment doses. Remaining high-dose females were sacrificed at week 90. There was no treatment-related effect on survival in male mice. Body weight was reduced in high-dose males and females beginning in week 32. Body weight was reduced by 13 to 25% in females and 14 to 22% in males after week 60. Specifics of the gross pathology were not provided. However, there was an increase in the incidence and severity of chronic dermatitis at the application site. There was an increase in ulcers at both the application site and inguinal sites for both treatments in males and females. There was an increase in the following non-neoplastic lesions in the high-dose males and females: amyloid in the liver, kidney, adrenal cortex (males only), spleen, and multiple organs; granulocytic hyperplasia in the bone marrow; and hyperplasia of the axillary lymph nodes (females only). There was a significant negative trend in the incidence of malignant lymphomas in males (8/50, 3/50, and 1/49 in control, low, and high dose groups). The LOAEL is 250 mg/kg/day, based on dermatitis and decreased survival in females. No NOAEL can be determined. At the doses tested, there was not a treatment-related increase in tumor incidence when compared to controls.

In a key carcinogenicity study (Klimisch score=1; Freeman et al., 1993), 37.5µL of Jet fuel A was administered dermally to the shaved backs of 50 male C3H/HeNCrlBR mice per dose, twice a week for 2 years or intermittently so that application of the jet fuel was suspended when dermal irritation was noted in 20% of the group and was resumed when irritation resolved in all but 20% of the affected animals. Negative and positive controls responded as expected. Dermal irritation occurred at the test site with the incidence and severity lessened in the intermittent group. Skin irritation was minimal in the controls. Histopathology of the skin found the following non-neoplastic lesions: acanthosis (focal and diffuse), epidermal necrosis, necrotic cell debris on epidermal surface, erosion/ulceration, subepidermal inflammatory infiltrate, dermal fibrosis, and oedema. There was a significant increase in tumors at the application site with continuous treatment compared to the control (0% versus 44%), but not with intermittent treatment (0% versus 2%). With continuous treatment, there was a treatment-related increase in dermal tumor incidence compared to controls. However, stopping treatment during dermal irritation nearly eliminated the carcinogenic effect.

In another key carcinogenicity study (Klimisch score=1; Clark et al., 1988), 25 mg of petroleum-derived Jet A was administered dermally to the shaved backs of 25 C3H/HeJ mice/sex/dose, three times a week for 105 weeks. Due to high mortality, Jet A application was discontinued during week 62, but surviving animals were observed until study termination. Negative and positive controls responded as expected. Dermal irritation first appeared at the test site after 10 to 15 days. Inflammation at the test site, characterized by reddening and swelling, progressed from the dermal irritation, generally after 6 months of treatment. Jet A females had inflammation occurring as early as 2 months. Necrosis (occurring by the end of the first year) at the test site was characterized by loss of skin integrity with visible cracking, separation and sloughing of skin, in many cases revealing the underlying mesenchymal tissue. Treated animals had a higher incidence of mortality with less than 10% of the treated mice surviving to study termination (even though treatment was discontinued in the Jet A mice during week 62) compared to approximately 45% in the shaved controls. There was a significant increase in tumors at the application site (0%, 26%, and 26% in the controls, JP-4, and Jet A groups). The majority of the tumors were squamous cell carcinomas or fibrosarcomas. At the doses tested, there was a treatment-related increase in dermal tumor incidence when compared to controls.

In summary, four of five studies reviewed by API (2010) above showed that various kerosenes (straightrun and jet fuels) cause increased incidence of cancer when applied to the skin.

Kerosene Non-Cancer Toxicity Excerpts
Many regulatory organizations have reviewed and summarized the composition and toxicity of kerosene.

NIOSH (1977) described the toxicity of kerosene and derived a recommended exposure limit (REL) of 100 mg/m³:

> Carpenter et al (1976) reported that acute human exposure at 140 mg/m³ (20ppm) of deodorized kerosene was innocuous. None of the subjects expressed discomfort or irritation during or after a 25-minute inhalation period. Fifty percent of the subjects, however, developed olfactory fatigue. Rats and dogs exposed a 100 mg/cu m (14 ppm) of deodorized kerosene for 6 hours/day, 5 days/week, for 67 days showed no toxic effects. In the Carpenter study, deodorized kerosene was used and, as such, may not truly reflect the toxicity of regular kerosene since deodorized kerosene contains less aromatics (3.9% versus 5-20%) than regular kerosene. Volkova et al (1969) showed that aerosol exposure to unpurified kerosene was more toxic than purified kerosene, but there is no indication that any grade of kerosene causes toxicity at concentrations of 100 mg/ cu m or below.
>
> 100 mg/cu m, as a TWA concentration, is recommended for kerosene. While particulates may exist at even lower concentrations, it is believed that kerosene vapor, rather than particulate, will more often be formed at this concentration, while at higher concentrations more particulate formation is likely.

Jet Fuel JP-5: Excerpts from ATSDR (2017)
The ATSDR (2017) derived a minimal risk level (MRL) for intermediate duration (14 to 365 days) for JP-5 (CAS No. 8008-20-6) at 2 mg/m³. The following describes the toxicological basis of ATSDR's MRL for JP-5.

> Groups of approximately 35 female C57BL/6 mice were exposed by inhalation to petroleum-based JP-5 vapor at 0, 150, or 750 mg/m³ continuously for 90 days (Gaworski, et al., 1984). End points evaluated included: clinical signs, body weight (measured monthly), and histopathological examination of major tissues (adrenals, anus, bladder, brain, colon, duodenum, esophagus, gall bladder, heart, ileum, kidneys, larynx, liver, lungs and bronchi, mammary gland, mandibular lymph node, nasal cavity, ovaries, pancreas, parathyroids, pituitary, prostate, salivary gland, sciatic nerve, seminal vesicles, skin, spleen, bone-sternebrae, vertebrae or femur plus marrow, stomach, testes, thigh muscle, thymus, thyroid, trachea, and uterus).

ATSDR (2017) stated that:

> No effect on body weight gain was noted. The only remarkable finding in mice was hepatocellular fatty changes and vacuolization at 150 and 750 mg/m³. The incidences were 8/37 (22%), 29/33 (88%), and 23/34 (68%) in the 0, 150, and 750 mg/m³ groups, respectively.

Additional details on the key study used to derive the MRL for JP-5 (ATSDR, 2017) are as follows:

> The intermediate-duration toxicity of inhaled JP-5 was investigated in rats, mice, and dogs. In male rats, the most sensitive effect is an increase in the occurrence of hyaline droplets in the proximal renal tubules, which was observed in rats continuously exposed to ≥150 mg/m³ JP-5 vapor (Gaworski et al. 1984). This effect, which is only observed in male rats, is due to an accumulation of alpha2u-globulin in hyaline droplets and is not considered relevant to humans (EPA 1991a; Flamm and Lehman-McKeeman 1991; Hard et al. 1993; Swenberg 1993). No adverse effects were observed in female rats exposed to ≤750 mg/m³ JP-5 vapor (Gaworski et al. 1984). Similar to mice, the liver appears to be the most sensitive target of toxicity in dogs; diffuse hepatocellular swelling was observed in male and female dogs continuously exposed to ≥150 mg/m³ JP-5 vapor (Gaworski et al. 1984). The nervous system was the only other target examined in intermediate JP-5 studies. An evaluation of neurobehavioral performance in rats found increased forelimb grip strength in rats similarly exposed to 1,200 mg/m³ JP-5 vapor (Rossi et al. 2001); no alterations in other neurobehavioral tests were found.
>
> Studies with JP-8 have also identified the immune system as a sensitive target of toxicity; based on the similarity between JP-5 and JP-8, it is likely that the immune system will also be a relevant target of JP-5. The lowest reliable LOAEL for immunotoxicity following acute-duration inhalation exposure to JP-8 was 1,000 mg/m³ identified in rats exposed to JP-8 vapor and aerosol 1 hour/day for 7 days (Hilgaertner et al., 2011).

The dose ATSDR (2017) used to derive the MRL for JP-5 was the Lowest-Observed-Adverse-Effect-Level (LOAEL) of 150 mg/m³ from Gaworski, et al. (1984):

> Diffuse hepatocellular swelling was observed in male and female dogs continuously exposed to ≥150 mg/m³ JP-5 vapor.

No duration adjustment was necessary because the mice were exposed for 90 days continuously (Gaworski, et al., 1984).

In order to calculate the MRL for JP-5, ATSDR (2017) took the LOAEL and converted it to a human equivalent concentration (HEC) of 150 mg/m³ by multiplying the LOAEL by the ratio of the blood:gas partition coefficients in humans and animals. Because blood:gas partition coefficients are not measurable for a complex mixture such as JP-5, the default ratio of 1 was used (EPA 1994). The $LOAEL_{HEC}$ = 150 mg/m³ × 1 = 150 mg/m³.

ATSDR (2017) used the following Uncertainty Factors (UFs) to derive the MRL for JP-5:

> UF1 = 3 for use of a minimal LOAEL
> UF2 = 3 for extrapolation from animals to humans with dosimetric adjustments
> UF3 = 10 for human variability

The ATSDR (2017) calculated the intermediate duration MRL for JP-5 as:

> $MRL = LOAEL_{HEC}/(UF1 \times UF2 \times UF3)$
> MRL = 150 mg/m³/(3 × 3 × 10)
> MRL = 1.67 mg/m³

MRL = 2 mg/m³ (rounded to 1 significant figure)

Jet Fuel JP-8

The ATSDR (2017) derived an MRL of 3 mg/m³ for JP-8 vapor with CAS No. 8008-20-6. The MRL for JP-8 vapor is for Intermediate Duration of 15-364 days.

Experimental design: Groups of male Sprague-Dawley rats (16/group) were exposed whole-body to 0, 500, or 1,000 mg/m³ JP-8 fuel vapors 6 hours/day, 5 days/week for 6 weeks (Ritchie et al., 2001). Sixty-five days after exposure termination, the rats underwent simple and difficult operant tasks (lever acquisition, fixed ratio, lever spatial reversal, stimulus reversal, and incremental repeated acquisition in order of increasing difficulty). After the neurobehavioral testing, 4 rats/group were killed and the brains were dissected and processed for determination of neurotransmitters and their metabolites.
Effect noted in study and corresponding doses: Exposure to 1,000 mg/m³ JP-8 fuel vapors induced significant deficits in acquisition or performance of the two most difficult tasks, but not in the simple learning tasks compared to rats in the low-exposure group. Learning/performance of complex tasks in the low-exposure group generally exceeded performance of control rats, while learning by high-exposure rats was almost always inferior to control rats, suggesting possible neurobehavioral hormesis. Neurochemical analyses showed significantly increased levels of dopamine in the cerebral cortex and DOPAC (major dopamine metabolite) in the brainstem for as long as 180 days post-exposure in both exposed groups relative to controls. This could have resulted from solvent-induced reductions in cyclic guanosine monophosphate (GMP) that is involved in signal transduction in specific brain regions.

Other additional studies or pertinent information that lend support to the MRL for JP-8: Two studies have examined the systemic toxicity of JP-8 following intermediate-duration inhalation exposure. Mattie et al. (1991) reported an increase in hyaline nephropathy in male rats continuously exposed to ≥500 mg/m³. JP-8 vapor for 90 days. No other effects were observed in the male rats and no effects were observed in the female rats; the highest concentration tested was 1,000 mg/m³. In contrast, Hanas et al. (2010) reported a number of adverse effects in male rats exposed to JP-8 vapor 6 hours/day for 91 days. At 250 mg/m³ proximal tubular damage was observed in the kidneys. At 500 mg/m³, enlarged alveolar capillaries, myocardial scarring, reduction in fat cells/globules in bone marrow, and dilated sinusoids and fatty hepatocytes were observed (Hanas et al., 2010). Interpretation of the results of the Hanas et al. (2010) study is limited by the small number of animals tested (3/group). The renal effects observed in the Mattie et al. (1991) and Hanas et al. (2010) studies are characteristic of alpha2u-globulin nephropathy, which is not considered a relevant effect in humans (EPA 1991a; Flamm and Lehman-McKeeman 1991; Hard et al. 1993; Swenberg 1993).

A 6-week study conducted by Rossi et al. (2001) also evaluated the neurotoxicity of JP-8 in rats exposed for 6 weeks. An alteration in a novel appetitive stimulus test was observed in rats exposed to 1,000 mg/m³. JP-8 vapor; the investigators suggested that this test quantified dopamine system sensitization in the rat. No other alterations in neurobehavioral tests were found. Studies by Fechter et al. (2012) and Guthrie et al. (2014, 2015) evaluated the potential of JP-8 to damage the auditory system. No significant alterations in auditory function was observed in rats exposed to 1,500 mg/m³ JP-8 vapor for 4 weeks (Fechter et al. 2012); however, if the rats were also exposed to non-damaging noise, there was damage to the auditory function. Central auditory processing dysfunction was observed in rats exposed to 1,000 mg/m³ JP-8 vapor for 4 weeks; however, no damage to peripheral auditory function, including damage to cochlear hair cells, was observed (Guthrie et al. 2014, 2015).

In addition to these studies, three University of Arizona studies have reported edema and inflammation of the terminal bronchioles in rats exposed 1 hour/day for 28 or 56 days to JP-8 aerosols and vapors (Hays et al. 1995; Pfaff et al. 1995, 1996). Hays et al. (1995) also found increased lung epithelial permeability and alveolar permeability. None of the three studies measured the vapor component of the test atmosphere.

ATSDR (2017) identified a No-Observed-Adverse-Effect-Level (NOAEL) of 500 mg/m³ from Ritchie et al. (2001) to derive the MRL for JP-8 vapor. The LOAEL of 1,000 mg/m³ for neurotoxicity was the critical effect identified in Ritchie et al. (2001).

The duration adjusted (ADJ) NOAEL was derived as:

$NOAEL_{ADJ}$ = 500 mg/m³ × 6 hours/24 hours × 5 days/7 days = 89 mg/m³

A $NOAEL_{HEC}$ of 89 mg/m³ was calculated by multiplying the $NOAEL_{ADJ}$ by the ratio of the blood:gas partition coefficients in humans and animals. Because blood:gas partition coefficients are not measurable for a complex mixture such as JP-8, the default ratio of 1 was used (EPA 1994). The $NOAEL_{HEC}$ = 89 mg/m³ × 1 = 89 mg/m³.

Uncertainty Factors used in ATSDR's (2017) MRL derivation for JP-8 vapor:
UF1 = 3 for extrapolation from animals to humans with dosimetric adjustments
UF2 = 10 for human variability

The ATSDR (2017) calculated the intermediate duration MRL for JP-8 as:
MRL = ($NOAEL_{HEC}$)/(UF1 × UF2) =
MRL = (89 mg/m³)/(3 × 10)
MRL = 3 mg/m³

Basis of the ITSLs for Kerosene
Several regulatory agencies have derived exposure standards to protect human health. These standards are summarized in Table 1.

Table 1. Kerosene Inhalation Health Standards

Kerosene Synonyms	Standard Setting Organization (Year Published)	Standard (mg/m³)	Exposure Duration	Potential ITSLs (µg/m³)
JP-5	ATSDR (2017)	2	15 to 364 days	200
JP-8	ATSDR (2017)	3	15 to 364 days	300
Fuel Oil No. 1, Range oil	NIOSH (1977)	100	8 hours	1000
Kerosine, fuel oil no. 1, deodorized kerosene, diesel No. 1, Jet A/Jet A-1, JP-4, JP-5, JP-8	ACGIH (2003)	200	8 hours	2000

As mentioned previously, the AEGL-1 (nondisabling) for JP-5 and JP-8 is 290 mg/m³ for all time periods (i.e., 10-minute, 30-minute, 1-hour, 4-hour, and 8-hour) (National Research Council, 2011). However, since AEGLs are meant to be used for a single

exposure, and not repeated over a lifetime, it was deemed inappropriate to use the AEGL for jet fuels to evaluate air permit emissions which may occur indefinitely.

The ATSDR (2017) minimal risk values (MRLs) for the kerosenes JP-5 and JP-8 are designed to be protective of sensitive individuals; therefore, the safety factor of 10 was not changed in order to derive the ITSL from the MRL. It is important to note that the duration of exposure used for the MRLs for JP-5 and JP-8 are "intermediate," and specifically designed for exposure periods between 15 to 364 days continuously. However, this intermediate exposure duration of the MRL does not conform with the four available averaging times set forth in Air Pollution Control Rules[3] 232(2) and 233(2): 1-hour, 8-hours, 24-hours, and annual.

Both the intermediate MRLs from ATSDR were derived from subchronic inhalation studies (JP-5 Gaworski et al. 1984; JP-8, Ritchie et al., 2001). ATSDR did not apply an UF of 10 to extrapolate from subchronic to chronic exposure. However, when deriving a Reference Concentration (RfC), EPA (1994) guidance specifies an UF of 10 to extrapolate from subchronic to chronic exposure. Using the subchronic to chronic UF of 10, RfCs can be calculated from the intermediate MRLs using the same Point of Departures (PODs; i.e., $LOAEL_{HEC}$ for JP-5, and $LOAEL_{HEC}$ for JP-8) used by the ATSDR (2017).

The RfC for JP-5 is calculated as:

$RfC = LOAEL_{HEC}/(UF1 \times UF2 \times UF3)$
$RfC = 150\ mg/m^3/(3 \times 3 \times 10)$
$RfC = 0.167\ mg/m^3$
$RfC = 0.2\ mg/m^3$ (rounded to 1 significant figure)

Where the uncertainty factors used to derive the RfC for JP-5 vapor are:
UF1 = 3 for extrapolation from animals to humans
UF2 = 10 for human variability
UF3 = 10 for subchronic to chronic study duration

The RfC for JP-8 is calculated as:

$RfC = (NOAEL_{HEC})/(UF1 \times UF2 \times UF3)$
$RfC = (89\ mg/m^3)/(3 \times 10 \times 10)$
$RfC = 0.3\ mg/m^3$

Where the uncertainty factors used to derive the RfC for JP-8 vapor are:
UF1 = 3 for extrapolation from animals to humans
UF2 = 10 for human variability
UF3 = 10 for subchronic to chronic study duration

ITSLs derived from RfCs are given annual averaging times, pursuant to Rule 232(2)(a).

Table 1 also shows two OELs for kerosene that are designed to protect workers exposed for 8-hours a day and a 40-hour workweek, for repeated exposures for a

[3] Part 55. Air Pollution Control, of the Natural Resources and Environmental Protection Act, 1994 PA 451, as amended.

working lifetime. The derivations of the OELs were assessed to determine if these values would be health protective. Both OELs were based on rat inhalation data and experience with work-place exposures to kerosene. The NIOSH (1977) REL of 100 mg/m³ is based on few and older studies; one study (Carpenter et al., 1976) that found no effects in rats exposed at 100 mg/m³. However, the petroleum distillate used by Carpenter et al. (1976) is not normally considered a kerosene[4]. In determining the REL, NIOSH (1977) relied on the fact that kerosene is relatively non-volatile and implies that kerosene lung toxicity is likely due to the aerosol fraction. NIOSH (1997) states:

> Exposure to kerosene in the aerosol form should be minimized, unless proper respiratory equipment is used, to prevent the possible accumulation of kerosene in the lungs which could result in pneumonitis. Because kerosene is less volatile that the other solvents discussed in this criteria document, hydrocarbon mists are more likely at a specific concentration with kerosene that with the other solvents. Thus, an environmental limit lower than those recommended for the other solvents is proposed in the absence of definitive data delineating safe from unsafe concentrations of kerosene.

More recently, in 2003, ACGIH reviewed the toxicological literature and derived a Threshold Limit Value Time Weighted Average (TLV-TWA) of 200 mg/m³ for kerosene and jet fuels. ACGIH (2003) cited many more animal toxicology studies than NIOSH (1977), as well as human epidemiologic studies (Knave et al., 1976; Knave et al., 1978). Because of the limited evaluation of the toxicological data, as well as a non-specific consideration of exposures and effects from "other solvents", it was determined that the NIOSH (1977) REL of 100 mg/m³ was less reliable as an OEL for ITSL derivation purposes than the ACGIH (2003) TLV-TWA for kerosene of 200 mg/m³. Therefore, short-term, repeated exposures should be evaluated using an ITSL derived from the ACGIH TLV-TWA. The acute ITSL for kerosene was derived, pursuant to Rule 232(1)(c), as:

ITSL = OEL/100
Where the OEL is 200 mg/m³.

The acute ITSL kerosene is derived as:

ITSL = (200 mg/m³)/100 × 1000µg/mg
ITSL = 2000 µg/m³

Pursuant to Rule 232(2)(b) the averaging time associated with the OEL-based ITSL is 8-hours. The acute ITSL is 2000 µg/m³ with 8-hour averaging time.

JP-5 vs. JP-8

As shown above, there were two chronic duration ITSLs derived for JP-5 and JP-8. Referring to the variations of kerosene, EPA (2011) stated, "the physical and chemical similarities among the streams in this category allow toxicology data on one material to be extrapolated to the others." It was decided that the chronic ITSL for kerosene should be based on toxicological data for JP-5. The difference in composition of JP-5 and JP-8

[4] In the Carpenter study the distillate used contained less aromatics (3.9%) than regular kerosene (5-20%) (NIOSH, 1977; page 191). American Petroleum Institute (API, 2010) stated that the kerosene that Carpenter (1976) used was, "probably a sweetened kerosene, CAS no. 91770-15-9."

is very small, such that the difference in critical effects observed in the animal studies is very likely to be caused by variation of study design (e.g., rodent species, number of animals, exposure concentrations), rather than an inherent difference in toxicity. Adopting the lower screening level value of 200 µg/m³ (compared to the value for JP-8 screening level value of 300 µg/m³) means that the screening level value of 200 µg/m³ would also be protective of exposures to kerosenes that are more like JP-8.

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