Decane, All Isomers

CAS Registry Number:

n-Decane: 124-18-5

Other 74 Isomers

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Acronyms and Abbreviations

Acronyms and Abbreviations	Definition
AMCV	air monitoring comparison value
^{o}C	degrees Celsius
CNS	central nervous system
d	day(s)
DSD	development support document
DAWS	Dearomatized White Spirit
ESL	effects screening level
$^{acute}ESL$	acute health-based effects screening level for chemicals meeting minimum database requirements
$^{acute}ESL_{odor}$	acute odor-based effects screening level
$^{acute}ESL_{veg}$	acute vegetation-based effects screening level
$^{chronice}ESL_{generic}$	chronic health-based effects screening level for chemicals not meeting minimum database requirements
$^{chronic}ESL_{threshold(c)}$	chronic health-based Effects Screening Level for threshold dose response cancer effect
$^{chronic}ESL_{threshold(nc)}$	chronic health-based Effects Screening Level for threshold dose response noncancer effects
$^{chronic}ESL_{nonthreshold(c)}$	chronic health-based Effects Screening Level for nonthreshold dose response cancer effects
$^{chronic}ESL_{nonthreshold(nc)}$	chronic health-based Effects Screening Level for nonthreshold dose response noncancer effects
$^{chronic}ESL_{veg}$	chronic vegetation-based effects screening level
GLP	good laboratory practice
h	hour(s)
$H_{b/g}$	blood:gas partition coefficient
$(H_{b/g})_A$	blood:gas partition coefficient, animal
$(H_{b/g})_H$	blood:gas partition coefficient, human
mm Hg	millimeters of mercury
HEC	human equivalent concentration

Acronyms and Abbreviations	Definition
HQ	hazard quotient
HSDB	Hazardous Substance Databank
IARC	International Agency for Research on Cancer
IOAEL	inhalation observed adverse effect level
^{acute}IOAEL	acute inhalation observed adverse effect level
^{subacute}IOAEL	subacute inhalation observed adverse effect level
^{chronic}IOAEL$_{(nc)}$	chronic inhalation observed adverse effect level (noncancer effects)
^{chronic}IOAEL$_{(c)}$	chronic inhalation observed adverse effect level (cancer effects)
kg	kilogram
LOAEL	lowest-observed-adverse-effect-level
MW	molecular weight
µg	microgram
µg/m^3	micrograms per cubic meter of air
mg	milligrams
mg/m^3	milligrams per cubic meter of air
min	minute(s)
MOA	mode of action
NOAEL	no-observed-adverse-effect-level
OECD	Organization for Economic Cooperation and Development
POD	point of departure
POD$_{ADJ}$	point of departure adjusted for exposure duration
POD$_{HEC}$	point of departure adjusted for human equivalent concentration
ppb	parts per billion
ppm	parts per million
ReV	reference value
Acute ReV	acute (e.g., 1-hour) health-based reference value for chemicals meeting minimum database requirements

Acronyms and Abbreviations	Definition
Acute ReV-24hr	acute 24-hour health-based reference value for chemicals meeting minimum database requirements
Chronic $ReV_{threshold(nc)}$	chronic health-based reference value for threshold dose response noncancer effects
RGDR	regional gas dose ratio
RPF	relative potency factor
SD	Sprague-Dawley rats
TCEQ	Texas Commission on Environmental Quality
TD	Toxicology Division
UF	uncertainty factor
UF_H	interindividual or intraspecies human uncertainty factor
UF_A	animal to human uncertainty factor
UF_{Sub}	subchronic to chronic exposure uncertainty factor
UF_L	LOAEL to NOAEL uncertainty factor
UF_D	incomplete database uncertainty factor
USEPA	United States Environmental Protection Agency
VCCEP	Voluntary Children's Chemical Evaluation Program
wk	week(s)
yr	year(s)

Chapter 1 Summary Tables

Table 1 and Table 2 provide a summary of health- and welfare-based values from an acute and chronic evaluation of n-decane and all isomers, respectively, for use in air permitting and air monitoring. Please refer to Section 1.6.2 of the *TCEQ Guidelines to Develop Toxicity Factors* (TCEQ 2015a) for an explanation of reference values (ReVs) and effects screening levels (ESLs) used for review of ambient air monitoring data and air permitting. Table 3 provides summary information on physical/chemical data for n-decane.

Table 1 Acute Health and Welfare-Based Screening Values for Decane, All Isomers

Screening Level Type	Duration	Value 1 (µg/m^3)	Value 2 (ppb)	Usage	Flags	Surrogated/ RPF	Critical Effect(s)	Notes
Acute ReV	1 h	5,800	1,000	M	A	--	Increase in sensation of mucous membrane irritation in the eyes in humans.	Applicable to n-decane and 74 isomers.
Acute ReV-24hr	--	--	--	--	--	--	--	Evaluated, not derived.
^{acute}ESL [a]	**1 h**	**1,700**	**300**	**P**	**S,D**	**--**	**Same as above.**	**Applicable to n-decane and 74 isomers.**
^{acute}IOAEL	6 h	580,000	100,000	N	none	--	Same as above.	--
^{subacute}IOAEL	--	--	--	--	--	--	--	--
$^{acute}ESL_{odor}$	--	--	--	--	--	--	--	No odor potential.
$^{acute}ESL_{veg}$	--	--	--	--	--	--	--	No data found.

Bold values used for air permit reviews.

[a] Based on the acute 1-h ReV multiplied by 0.3 (i.e., HQ = 0.3) to account for cumulative and aggregate risk during the air permit review.

Usage:
P = Used in Air Permitting
M = Used to Evaluate Air Monitoring Data
R = Used to Calculate Remediation Cleanup Levels
N = Usage Not Defined

Flags:
A = AMCV report
S = ESL Summary Report
D = ESL Detail Report

Table 2 Chronic Health and Welfare-Based Screening Values for Decane, All Isomers

Screening Level Type	Duration	Value 1 (µg/m³)	Value 2 (ppb)	Usage	Flags	Surrogated/ RPF	Critical Effect(s)	Notes
Chronic $ReV_{threshold(nc)}$	70 yr	1,100	190	M	A	--	Increase in body weight gain and decrease in white blood cell count in rats.	Applicable to n-decane and 74 isomers.
$^{chronic}ESL_{threshold(nc)}$ [a]	**70 yr**	**330**	**57**	**P**	**S,D**	**--**	**Same as above.**	**Applicable to n-decane and 74 isomers.**
$^{chronic}IOAEL_{(nc)}$	70 yr	520,000	90,000	N	none	--	Same as above.	--
$^{chronic}ESL_{threshold(c)}$	--	--	--	--	--	--	--	Inadequate information to assess carcinogenic potential.
$^{chronic}ESL_{nonthreshold(c)}$	--	--	--	--	--	--	--	Inadequate information to assess carcinogenic potential.
$^{chronic}IOAEL_{(c)}$	--	--	--	--	--	--	--	--
$^{chronic}ESL_{veg}$	--	--	--	--	--	--	--	No data found.
$^{chronic}ESL_{animal}$	--	--	--	--	--	--	--	No data found.

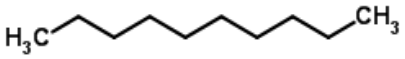
Bold values used for air permit reviews

[a] Based on the chronic ReV multiplied by 0.3 (i.e., HQ = 0.3) to account for cumulative and aggregate risk during the air permit review.

Usage:
P = Used in Air Permitting
M = Used to Evaluate Air Monitoring Data
R = Used to Calculate Remediation Cleanup Levels
N = Usage Not Defined

Flags:
A = AMCV report
S = ESL Summary Report
D = ESL Detail Report

Table 3 Chemical and Physical Data

Parameter	n-Decane	Reference
Molecular Formula	$C_{10}H_{22}$	HSDB
Chemical Structure		ChemSpider
CAS Registry Number	124-18-5	HSDB
Molecular Weight	142.28	HSDB
Structural Formula	CH_3-$(CH_2)_8$-CH_3	HSDB
Physical State	Liquid	HSDB
Color	Colorless	HSDB
Odor	Gasoline-like	HSDB
Synonyms/Trade Names	Decyl hydride; Methyl nonane	HSDB
Solubility in water @25°C	Insoluble (0.052 mg/l)	HSDB
Log K_{ow}	5.01	HSDB
Vapor Pressure @25°C	1.4 mm Hg	HSDB
Vapor density (air = 1)	4.9	HSDB
Density/Specific Gravity (water = 1)	0.73 @ 20°C	HSDB
Melting Point	-29.7°C	HSDB
Boiling Point	171.4°C	HSDB
Lower Explosive Limit	0.8%	HSDB
Conversion Factors	1 ppm = 5.82 mg/m^3 1 mg/m^3 = 0.17 ppm	TD

Chapter 2 Background Information

2.1 Chemical/Physical Properties

While the chemical properties vary slightly from isomer to isomer, every decane isomer has similar structure activities. The main chemical and physical properties of n-decane are summarized in Table 3.

2.2 Major Sources and Uses

Decane is a colorless liquid with gasoline-like odor. It is used as a solvent and in jet fuel. Decane is a constituent in the paraffin fraction of petroleum and natural gas. It composes 1.8% by volume of a crude oil sample. Decane is also found in several widely used petroleum distillates such as Stoddard solvent and jet fuel (ATSDR 1995a,b). n-Decane is used as a solvent and to make other chemicals (HSDB). Decane is listed as a High Production Volume chemical with production exceeding 1 million pounds annually in the United States. Decane is released into the air primarily from evaporative emissions resulting from manufacturing or using products containing decane. Atmospheric concentrations of decane measured in polluted urban atmosphere were 1-2.7 ppb (Cavender 1994, as cited in ATSDR 1995a, b).

In Texas, the highest reported 1-hour (h) concentration (from 1998 through 2015) of n-decane was 35.75 ppb collected from Automated Gas Chromatograph samples at the Clinton Drive ambient air monitoring site in Houston, Texas in 2015. The highest annual average concentration of n-decane was 0.1043 ppb measured at the Clinton Drive monitoring site in 1998. The highest 24-h n-decane value from 1995 to 2015 was 22.43 ppb collected from 24-h summa canister samples at the West Orange monitoring site in Beaumont in 1997. The highest annual average concentration of n-decane collected from canister samples was 0.47 ppb measured at the West Orange monitoring site in Beaumont in 1997.

The isomers of decane are colorless, semi-volatile liquids with a gasoline-like odor and are practically insoluble in water. There are 75 isomers of decane including n-decane (Appendix A).

Chapter 3 Acute Evaluation

3.1 Health-Based Acute 1-Hour ReV and ESL

There are one human and two animal inhalation study available concerning the acute adverse effects of decane. There is another human inhalation study exposure to dearomatized White Spirits (DAWS) (contained >99% C_{10}-C_{12} isoparaffins) available. Like other aliphatic hydrocarbons, n-decane has a low acute toxicity in experimental animals. Respiratory tract irritation or transient central nervous system (CNS) depression was primarily observed at high concentrations (e.g., exposure of n-decane at saturated vapor concentrations). Acute effects in

humans and animals are considered similar to other saturated C_7-C_9 aliphatic hydrocarbons (OECD 2010).

3.1.1 Key Studies

3.1.1.1 Kjærgaard et al. (1989) Human Study

In an inhalation study by Kjærgaard et al. (1989), 63 healthy human subjects (7-9 /sex/group), randomly selected from the general population, were exposed to pure n-decane concentrations of either 0, 10, 35, or 100 ppm (0, 58.2, 204, or 582 mg/m^3) for 6 h in a controlled, double-blind study using a Latin square exposure design. There were no significant differences in distribution of age, sexes, smokers, or education levels in the four exposure groups. The corresponding measured mean ± standard deviation concentrations were 0.01 ± 0.01, 12.1 ± 0.5, 36.6 ± 0.6, and 104 ± 1.2 ppm. The Stinger's test (an indicator of skin sensitivity), odor threshold test, and eye irritation threshold to carbon dioxide test were performed to measure subjective sensitivity. Questionnaires and a linear potentiometer were used to measure subjective reactions. Physiological reactions were measured in the external eye by the dry eye syndromes (measured by tear film stability test as the time from an eye-blink to the breakup of the pre-corneal tear film using a slip lamp microscope), and by the sensation of mucous membrane irritation (graded by linear visual analogue rating scales using a potentiometer) and conjunctival secretions (cytological examination).

The results showed dose-dependent changes (evaluated by stepwise regression analysis, $p < 0.05$) in reduction of the perception of indoor air quality (measured by questionnaire), irritation of mucous membranes of the eyes, decreased tear film stability, increased sensation of odor intensity, and increased conjunctival polymorphonuclear leukocytes. Results for specific endpoints are described below.

Significant reduction of the perception of air quality (changes from pre-exposure baseline), measured immediately after 1 h exposure, was shown in all exposed groups ($p < 0.001$ in the analysis of variance (ANOVA). The reduction of the perception of air quality was dose-dependent ($P < 0.01$). However, no dose-dependent differences for any questions about air quality were found after 3 and 6 h exposure, as the effect decreased in the 35 and 100 ppm exposure groups. The investigators indicated the result was an adaptation effect and thus not considered an adverse effect.

No statistically significant changes (one-way ANOVA) were observed in cell counts in lymphocytes, columnar epithelium, and squamous epithelium. The changes in cell counts in tear fluids were significant for the cuboidal epithelium cells in one-way ANOVA, but only the 35 ppm exposure group showed significantly increased (greater than zero, $p < 0.05$) numbers of cuboidal epithelium cells. The changes in polymorphonuclear leukocytes were significant in a log-linear analysis of trend and showed a dose-response relationship. However, the numbers of

polymorphonuclear leukocytes were statistically significantly less than zero in the control and 10 ppm groups. The decrease was less for the subjects exposed to 10 ppm. There were slight increases (greater than zero) in the 35 ppm group, which were more pronounced in the 100 ppm group. The increases in the 35 and 100 ppm groups, however, were not significant differences greater than zero (Table 4). Therefore, the TCEQ does not consider the changes in numbers of polymorphonuclear leukocytes an adverse effect.

Table 4 Changes in Cell Counts in Conjunctival Secretion During the Day[a]

Cell Type	Control	10 ppm (58.2 mg/m^3)	35 ppm (204 mg/m^3)	100 ppm (582 mg/m^3)
Polymorphonuclear leukocytes[*]	-186[***]	-62[***]	3	27
Cuboidal epithelium[**]	-3	0	5[****]	-12

[a] Source: Kjærgaard et al. 1989
[*] p <0.05 log-linear analysis of trend
[**] p <0.05 one-way analysis of variance
[***] p <0.05 for difference less than zero (Wilcoxon's test)
[****] p <0.05 for difference greater than zero (Wilcoxon's test)

The physiological measurements showed dose-dependent decreased tear film stability in all exposure groups, which decreased more with increasing exposure concentrations in the analysis of variance (ANOVA) ($p<0.035$). The differences of break up time of the tear film before and during exposure were strongly negatively correlated with the baseline values measured before exposure with the highest difference in the 35 ppm exposure group (as opposed to the highest concentration 100 ppm group). The differences were +1, -9, -19, and -9 seconds, respectively, observed in the 0, 10, 35, and 100 ppm groups. The results did not show a dose-response relationship and thus, neither NOAEL nor LOAEL was identified.

The measurements of sensation of mucous membrane irritation (potentiometer test) were significantly related to exposure. The investigators indicated that due to the inhomogeneity of variances neither the ANOVA nor the regression analysis was conclusive in regard to mucous membrane irritation. A nonparametric test, however, showed significantly increased scores for exposed groups compared with the control group ($p <0.05$). The changes from baseline, expressed as percentage of the full scale, were approximately -0.6, 2, 2.5, and 10%, respectively, in the 0, 10, 35, and 100 ppm groups. According to Kjærgaard et al (1992), a 10 % increment of irritation scores is considered as the threshold. Therefore, the level of 100 ppm was considered a lowest-observed-adverse-effect-level (LOAEL) for sensation of mucous membrane irritation. The level of 35 ppm was then considered a no-observed-adverse-effect-level (NOAEL) and was used as a point of departure (POD) to derive a candidate acute ReV.

3.2.1.2 Lammers et al. (2011) Animal Study

In a study by Lammers et al. (2011), neurobehavioral effects of acute exposure to C_5-C_{10} n-paraffins in rats were assessed in accordance with good laboratory practice (GLP) conditions. Male WAG/RijCHBR rats (8 rats/group) were exposed to air (control), 500, 1,500 or 5,000 mg/m^3 (85, 260, 860 ppm, respectively) n-decane (>99% purity, target concentrations) for 8 h/day (d) for 3 d. The mean analytical exposure concentrations were 501, 1,510, and 5,005 mg/m^3. Motor activity and neurobehavioral function (using a standardized functional observational battery (FOB) were evaluated immediately prior to exposure, and 30 minutes (min) after exposure on each of the 3 d and 24 h after the last exposure. The results showed that the only treatment-related effect on FOB measures was a statistically significant reduction in forelimb grip strength in the 5,000 mg/m^3 exposure group after 3 d of exposure. Exposure to n-decane did not induce any dose-related effects on motor activity measures. In another part of this study, separate groups of rats were evaluated for cognitive performance using a discrete-trial, two-choice visual discrimination performance testing. The results showed that the only statistically significant and probably dose-related finding was a minimal and temporal increase in frequency of long latency responses in the 5,000 mg/m^3 but not at the 500 or 1,500 mg/m^3 exposure group. In both parts of the study, no changes in body weights or remarkable clinical signs between the groups were reported. A NOAEL and LOAEL of 1,500 and 5,000 mg/m^3 for neurobehavioral effects were identified from this study. The NOAEL of 1,500 mg/m^3 (260 ppm) was also used as a POD to derive the acute ReV for n-decane.

3.1.2 Supporting Studies

3.1.2.1 Pederson et al. (1984) Human Study

Twelve human volunteers were exposed to a single 6 h exposure to 100 ppm a dearomatized White Spirits (DAWS) contained >99% C_{10}-C_{12} isoparaffin (Shell Sol TS) (Pederson et al. (1984, as cited in Mullin et al. 1990). Answers from self-administered questionnaire showed no difference during and after exposure in sensory parameters (headache, dizziness, dryness of mucous membrane, fatigue, muscular weakness, or nausea). There were no changes in blood chemistry and urine analysis following isoparaffin exposure. The level of 100 ppm was a free-standing NOAEL.

3.1.2.2 Nilsen et al. (1988) Animal Study

In an acute toxicity study by Nilsen et al. (1998), groups of 10 male SD rats were exposed to 1,369 ± 19 ppm (mean measured concentration ± standard deviation, a saturation level) n-decane for 8 h and observed for the following 14 d. Four additional rats were exposed simultaneously to filtered air as the control group. No deaths, differences in body/organs weights, morphological alterations, or CNS depression were observed during or following the 8-h exposure. The level of 1,369 ppm was considered a free-standing NOAEL for this study. The NOAEL is higher than the NOAEL (260 ppm) identified from the key study (Lammers et al. 2011).

3.1.3 Reproductive/Developmental Toxicity Studies

No information on the potential of decane to cause reproductive/developmental toxicity in humans or animals via inhalation is available. OECD guideline oral reproductive toxicity screening studies have been conducted with n-decane and indicate a low potential for reproductive and/or developmental toxicity (Maraschin et al. 1955, as cited in ACC 2004). In the Maraschin et al. (1955) oral study, n-decane was administered to male rats for 28 d (14 d prior to mating and 2 weeks during mating), and to female rats from 14 d prior to mating, through mating and gestation to d 4 of lactation at doses of 0, 25, 150, or 1000 mg/kg/d. No reproductive or developmental toxicity was observed at any administered dose. The NOAEL for parental reproductive effects and effects on the offspring was 1,000 mg/kg/d; equivalent to an inhalation concentration of approximate 7,000 mg/m^3 (1,200 ppm) for 6 h/d (ACC 2004).

3.1.4 Mode of Action (MOA) Analysis and Dose Metric

n-Decane is readily absorbed and distributed throughout the body and metabolized to decanol, decanoic acid and decamethylene glycol. The eye mucous membrane irritation observed in human volunteers exposed to n-decane may be due to n-decane (very lipophilic) dissolved in the membrane of the eyes (Kjærgaard et al. 1989). In addition, n-decane has been observed to distribute and accumulate preferentially into rat brain tissue. In the Lammers et al. (2011) key study, n-decane was rapidly taken up, achieved steady state within 2 h, and eliminated rapidly once exposures were terminated. The brain n-decane levels were 13-22 times higher than the blood levels. The significant distribution of n-decane to the brain would suggest that the CNS is a possible target organ for the toxic effects of the n-decane. Like other C_7-C_{10} n-alkanes, the MOA for n-decane-induced CNS effects is proportional to the n-decane concentration in the brain (Lammers et al. 2010). Data on the exposure concentration of the parent chemical are available, whereas data on more specific dose metrics are not available. Thus, exposure concentration of the parent chemical was used as the dose metric.

3.1.5 Critical Effect and POD

In order to determine the critical effect amongst multiple endpoint PODs, POD_{HEC}s for the LOAELs from both the Kjærgaard et al. (1989) human study and the Lammers et al. (2011) animal study were determined. The lower LOAEL-based POD_{HEC} determines the critical effect for derivation of the acute ReV and ESL (TCEQ 2015a). The acute LOAEL is 100 ppm for Kjærgaard et al. (1989) and 860 ppm for Lammers et al. (2011). After dosimetric adjustments, the POD_{HEC}s for the LOAELs from the Kjærgaard et al. (1989) and Lammers et al. (2011) studies were 181.712 and 285.519 ppm, respectively. Therefore, mucous membrane irritation of the eyes is the critical effect and the NOAEL of 35 ppm identified from the Kjærgaard et al. (1989) was used as the POD to derive acute ReV and ESL. The details of determination of the POD_{HECs} for the LOAELs are described in Appendix B.

3.1.6 Dosimetric Adjustments

3.1.6.1 Exposure Duration Adjustments

The POD of 35 ppm from the Kjærgaard et al. (1989) study was adjusted from 6-h exposure to 1-h exposure concentration using Haber's rule as modified by ten Berge with a default value of "n"=3 (TCEQ 2015a).

$$POD_{ADJ} = C_2 = [(C_1)^3 \times (T_1 / T_2)]^{1/3}$$
$$= [(35 \text{ ppm})^3 \times (6 \text{ h}/1 \text{ h})]^{1/3}$$
$$= 63.599 \text{ ppm}$$

3.1.6.2 Default Dosimetry Adjustments from Animal-to-Human Exposure

The POD_{ADJ} of 63.599 ppm was based on human inhalation exposure and thus, no dosimetry adjustment from animal-to-human exposure. The POD_{HEC} from the POD_{ADJ} is 63.599 ppm.

3.1.7 Adjustments of the POD_{HEC}

The POD_{HEC} of 63.599 ppm was used to derive the acute ReV and ^{acute}ESL for n-decane. The following UFs were applied to the POD_{HEC} (Total UF = 60):

- a UF_H of 10 for intraspecies variability,
- a UF_D of 6 was used for the uncertainty associated with an incomplete database because one human and two animal inhalation studies were available, although only one animal species was used. Additional human inhalation study exposure to DAWS (contained >99% C_{10}-C_{12} isoparaffins) was available. A value of 10 was not used because the endpoints evaluated in animals predominately concerned neurotoxicity, and one oral reproductive/developmental study was reported. Acute effects in humans and animals are considered similar to other saturated C_7-C_9 aliphatic hydrocarbons (OECD 2010). Studies evaluating acute effects as well as reproductive/developmental toxicity conducted in other saturated C_7-C_9 n-alkanes can be applied to database completeness for decane. Consistent with TCEQ (2015a), confidence in the database is considered low to medium. The quality of the key rat study is medium to high.

$$\text{Acute ReV} = POD_{HEC} / (UF_H \times UF_D)$$
$$= 63.599 \text{ ppm} / (10 \times 6)$$
$$= 1.069 \text{ ppm}$$
$$= 1{,}000 \text{ ppb or } 5{,}800 \text{ µg/m}^3 \text{ (rounded to two significant figures)}$$

3.1.8 Health-Based Acute ReV and ^{acute}ESL

In deriving the acute ReV, no numbers were rounded between equations until the ReV was calculated. Once the ReV was calculated, it was rounded to two significant figures. The rounded

ReV was then used to calculate the ESL, and the ESL subsequently rounded. The ^{acute}ESL of 1,700 µg/m^3 (300 ppb) for n-decane is based on the acute ReV of 5,800 µg/m^3 (1,000 ppb) multiplied by a HQ of 0.3 and rounded to two significant figures at the end of all calculations. Table 5 summarizes the derivation of acute toxicity factors for n-decane.

Table 5 Summary of Acute ReV and ^{acute}ESL for n-Decane

Parameter	Values and Descriptions
Study	Kjærgaard et al. (1989)
Study Quality	Medium to high
Study Population	63 healthy human subjects (7-9 /sex/group)
Exposure Method	Air (control), 10, 35, or 100 ppm (0, 58.2, 204, or 582 mg/m^3, respectively) (target concentrations)
Exposure Duration	6 h/d
Critical Effects	Increase in sensation of mucous membrane irritation in the eyes at 100 ppm
POD	35 ppm (NOAEL)
POD_{ADJ} to 1h	63.599 ppm
POD_{HEC}	63.599 ppm
Total UFs	60
Intraspecies UF	10
Incomplete Database UF *Database Confidence*	6 Low to Medium
Acute ReV [1 h] (HQ = 1)	**5,800 µg/m^3 (1,000 ppb)**
^{acute}ESL [1 h] (HQ = 0.3)	**1,700 µg/m^3 (300 ppb)**

3.2 Health-Based Acute 24-Hour ReV

Consistent with TCEQ Guidelines (TCEQ 2015a), the potential need for a 24-h ReV was evaluated. However, the highest monitored 24-h concentrations (22.43 ppb) of n-decane across Texas (1995-2015) was ≥ 8.5 times below the chronic ReV of 190 ppb. Therefore, a 24-h ReV is not needed and is not derived in this DSD.

3.3 Welfare-Based Acute ESLs

3.3.1 Odor Perception

n-Decane has a gasoline-like odor. An odor detection threshold of 620 ppb (3,600 µg/m^3) has been reported by Nagata (2003). Since decane and isomers do not have a pungent or disagreeable odor, an $^{acute}ESL_{odor}$ was not developed (TCEQ 2015b).

3.3.2 Vegetation Effects

No information was found to indicate that special consideration should be given to possible vegetation effects from decanes.

3.4 Short-Term ESLs and Values for Air Monitoring Data Evaluations

The acute evaluation resulted in the derivation of the following values for n-decane:

- Acute ReV (1-h) = 1,000 ppb (5,800 µg/m^3)
- ^{acute}ESL = 300 ppb (1,700 µg/m^3)

For the evaluation of ambient air monitoring data, the 1-h ReV of 5,800 µg/m^3 (1,000 ppb) is used (Table 1). The short-term ESL for air permit reviews is the health-based ^{acute}ESL of 1,700 µg/m^3 (300 ppb) (Table 1). The ^{acute}ESL (HQ = 0.3) is not used to evaluate ambient air monitoring data.

3.4.1 Other Decane Isomers

No acute toxicity data were available for the 74 other decane isomers. For the purpose of health effects evaluations for air permit applications and/or ambient air monitoring data, the 1-h ReV of 2,800 µg/m^3 and short-term ESL of 840 µg/m^3 values for n-decane will be used as surrogates.

3.5 Acute Inhalation Observed Adverse Effect Levels (IOAELs)

The acute inhalation observed adverse effect level (^{acute}IOAEL) of 100 ppm for n-decane was conservatively based on the 6-h $LOAEL_{HEC}$ of 100 ppm for increase in sensation of mucous membrane irritation in human eyes (Kjærgaard et al. 1989). No duration adjustments were made. Effects occurred in the ^{acute}IOAEL represent a concentration at which it is possible that similar effects could occur in some individuals exposed to this level over the same duration or longer as used in the study (i.e., ≥ 6 h). Importantly, effects are not a certainty due to potential interspecies differences in sensitivity. The ^{acute}IOAEL level is provided for informational purposes only (TCEQ 2015a). The ^{acute}IOAEL for n-decane is:

- n-Decane ^{acute}IOAEL = 580 mg/m^3 (100 ppm) (rounded to 2 significant figures)

The margin of exposure between the ^{acute}IOAEL (100 ppm) and the acute ReV (1 ppm) for n-decane is approximately a factor of 100.

Chapter 4 Chronic Evaluation

4.1 Noncarcinogenic Potential

4.1.1 Key and Supporting Studies

n-Decane is not sufficiently volatile (i.e., saturated vapor concentration at 1,369 ppm) to cause specific toxic effects from repeated inhalation exposures. There are no chronic inhalation studies on n-decane available and only one subchronic inhalation study (Nau et al. 1966). The results of the Nau et al. (1966) subchronic study did not indicate any effects in animals at concentration ≤ 540 ppm.

4.1.2 Key Animal Study (Nau et al. 1966)

Nau et al. (1966) exposed 43 rats (strain/age not documented) to 3.1 mg/L (540 ppm) n-decane by inhalation for 18 h/d, 7 d/week for 13 weeks with 32 d recovery. The results showed a significant positive effects on the expected body weight gain (approximately a 17% increase after 91 d of exposure) and a significant decrease in the total white blood cell count (WBC) (approximate 14.3% and 2.5% decrease after 57 and 84 d of exposure, respectively) compared to the controls. However, an increase of total WBC count (approximately 5% increase) was observed after 91 f of exposure. After 123 d (91 d of exposure with 32 d recovery), both body weight gain and total WBC count were increased. No changes in the gross or microscopic organ, bone marrow, polymorphonuclear leukocytes or lymphocytes were observed. The American Chemistry Council n-Alkane Voluntary Children's Chemical Evaluation Program (VCCEP) Consortium (ACC 2004) indicated that no relevant adverse effects were noted in this study. The level of 540 ppm was considered a free-standing NOAEL by VCCEP. Nevertheless, the TCEQ conservatively considered the level of 540 ppm a minimal LOAEL for increase in body weight gain and a significant decrease in white blood cell count and was used as the POD to derive chronic ReV and ESL.

4.1.3 Supporting Animal Studies

Several subchronic/chronic inhalation studies on isoparaffinic solvents, dearomatized mineral spirits or White spirit (Stoddard Solvent) containing <2% aromatics were available. In a review by Amoruso et al. (2008), toxicity data on selected chemical constituents of mineral spirits (e.g., n-nonane, n-decane, n-undecane) indicate that these chemicals have similar toxicological properties as dearomatized mineral spirits. For the purpose of comparison, some of these studies were included as supporting studies in the chronic evaluation of decanes.

4.1.3.1 Lund et al. (1996)

In a chronic inhalation study by Lund et al. (1996), groups of 3 month old Wistar rats (36 rats/group) were exposed to 0, 400, or 800 ppm (0, 2,339 or 4,679 mg/m^3) DAWS (Exxon D40 Fluid, CAS# 64742-48-9, C_{10}-C_{13} n-, iso- and cyclo-alkanes containing < 0.4% aromatics, mean molecular weight 143 g/mol) for 6 h/d, 5 d/week for 6 months. The body weights of the exposed animals did not differ from the controls throughout the experiment. After an exposure-free period of 2-6 months duration, neurophysiological, neurobehavioral, and macroscopic pathologic examinations were performed. The study revealed exposure-related changes in sensory-evoked potentials and a decrease in motor activity during dark (no light) periods but no white spirit-induced changes in learning and memory functions. The measurements of the flash evoked potential (FEP), somatosensory evoked potential (SEP), and auditory brain stem response (ABR) all demonstrated dose-dependent increases of the amplitudes of the early latency peaks of the sensory evoked potentials (EPs). Furthermore, an increase of the dose showed that the measurements of FEP and SEP revealed changes in the later-latency peaks, which reflect the more associative aspects of sensory processing. The investigators concluded that 6 months of exposure to DAWS at 400 or 800 ppm could induce long-lasting and possible irreversible effects in the nervous system of the rat. The level of 400 ppm was considered a LOAEL. Since the DAWS is a mixture of C_{10}-C_{13} n-, iso- and cyclo-alkanes and the weight % of decane was not indicated, the identified LOAEL was not used as a POD to derive the chronic toxicity factors for n-decane.

4.1.3.2 Phillips and Egan (1984)

In a subchronic study by Phillips and Egan (1984), male and female rats (35 rats/sex/group) were exposed by inhalation 6 h/d, 5 d/week for 12 weeks to target concentrations of either 1.89 or 5.67 g/m^3 (mean measured concentrations of 1.97 ± 0.15 g/m^3 or 5.61 ± 0.21 g/m^3 (312 or 890 ppm) C_9-C_{13} multiconstituent alkanes (dearomatized White Spirit (DAWS), containing <0.5% aromatics, 58% n- and iso-alkanes and 42% cycloalkanes; or C_{10}-C_{11} isoparaffinic hydrocarbon (IPH) vapor at target concentrations of either 1.83 or 5.48 g/m^3 (mean measured concentrations standard deviation of 1.91 ± 0.16 and 5.62 ± 0.3 g/m^3 (314 or 922 ppm). Following weeks 4, 8, and 12 of exposure, a total of 10, 10, and 15 rats, respectively, from each group were sacrificed. Clinical chemistry, hematology parameters, and organ weights were measured, and histopathological examinations were performed at study termination. There were no deaths during the course of this study related to either DAWS or IPH. Mean body weights were slightly but significantly lower than controls (< 10%) in male rats following exposure to 5.61 g/m^3 DAWS, and 5.62 or 1.91 g/m^3 IPH. Body weights were not affected in females. These minor decreases in body weights, however, were not accompanied by hematological or clinical signs of toxicity. There were increased liver weight and liver/body weight ratios in both male and female rats exposed to either DAWS or IPH in the high exposure groups. The liver weight significantly increased 10.3 and 6.9 % in females ($p < 0.05$), but not in males, at 1.97 and 5.61 g/m^3 DAWS exposure group, respectively. The significant increases in liver/body weight ratio were observed in males (10 %) and females (9.5 %) exposed to 5.61

g/m^3 DAWS (P <0.01). However, no associated histopathology was measured, indicating the liver weight increase was an adaptive effect. Kidney weights were increased only in males, and histological effects are consistent with chronic progressive nephritis and/or α2μ-globulin induced nephropathy, effects in male rats that have been determined to have no relevance to humans. The finding is not considered to be of biological significance to humans (ACC 2004). The investigators concluded that, with the exception of the mild male rat tubular nephrotoxicity, the results of this subchronic study did not suggest a significant difference in other toxic effects of the two light hydrocarbon solvents studied.

4.1.3.3 Carrillo et al. (2013)

In a similar subchronic inhalation study of isoparaffinic solvents by Carrillo et al. (2013), 13 week old male and female SD rats (18 rats/sex/group) were exposed to C_{10}-C_{12} isoalkanes (<2% aromatics) at nominal concentrations of 2,600, 5,200, or 10,400 mg/m^3 (368, 737, or 1,474 ppm, respectively) for 6 h/d, 5 d/week for 13 weeks. The respective measured concentrations ± standard deviations were 2,529 ± 116, 5,200 ± 207, or 10,186 ± 327 mg/m^3 (359, 737, or 1,444 ppm, respectively). Body and organ weights, clinical chemistry, and hematology parameters were measured, and histopathological examinations were performed at study termination. No clinical signs of toxicity were observed in any of the exposed groups. The average body weights of male and female exposed groups were not significantly different than the controls. Treatment-related nephropathy was found in male rats exposed to all concentrations. Increases in liver weights of 10, 14 and 32% were observed in male rats in the low, medium and high exposure groups, respectively. The nephropathy observed was attributed to the occurrence of α2μ-globulin specific to the male rat (Section 4.1.2.1). In females, the liver weights were increased in the medium (10%) and high (37%) exposure groups. The authors, however, indicated that the increased liver weights were not associated with any histological findings and were considered an adaptive effect. Among hematological parameters, a slight but statistically significant decrease in the percent of neutrophils and leukocytes was observed in females exposed to high dose (10,400 mg/m^3). Small but statistically significant decreases in hemoglobin, packed cell volumes, and red blood cell counts (approximately 3-5%) were observed in all male exposed groups. The investigators suggested that the reductions in hematological endpoints observed only in male rats might be due to normal variation or a secondary consequence of the nephropathy. The key findings in this study were consistent with the Phillips and Egan (1984) study. The investigators concluded that the NOAEL was 10,400 mg/m^3 (1,474 ppm).

4.1.3.4 Reproductive/Developmental Toxicity Studies

No information on the potential of n-decane to cause reproductive/developmental toxicity in humans or animals via inhalation is available. OECD guideline oral reproductive toxicity screening studies have been conducted with n-decane and indicate a low potential for reproductive and/or developmental toxicity (Maraschin et al. 1955, as cited in VCCEP 2004). In this oral study, n-decane was administered to male rats for 28 d (14 d prior to mating and 2

weeks during mating), and to female rats from 14 d prior to mating, through mating and gestation to d 4 of lactation at doses of 0, 25, 150, or 1000 mg/kg/d. No reproductive or developmental toxicity was observed at all administered doses. The NOAEL for parental reproductive effects and effects on the offspring was 1,000 mg/kg/d; equivalent to an inhalation concentration of approximate 7,000 mg/m^3 (1,200 ppm) for 6 h/d (ACC 2004). In addition, no treatment-related reproductive/developmental effects were found in rats exposed to 300 or 900 ppm C_9-C_{13} mixed alkanes containing 0.4% aromatics (petroleum distillate, CAS# 64742-47-8) for 6 h/d on gestation day 6-15 (EMBSI 1978b, as cited in Amoruso et al. 2008). The NOAEL for developmental/reproductive effects was ≥ 900 ppm (approximately ≥ 5,000 mg/m^3). Accordingly, reproductive/developmental effects will be protective by using the NOAEL of 540 ppm (Nau et al. 1966) as the POD to derive chronic toxicity factor.

4.1.4 MOA Analysis and Dose Metric

The MOA for n-decane-induced chronic effects was assumed to be similar to other C_7-C_{10} n-alkanes (see Section 3.1.4). Data on the exposure concentration of the parent chemical are available, whereas data on more specific dose metrics are not available. Thus, exposure concentration of the parent chemical was used as the dose metric.

4.1.5 POD and Critical Effect

The subchronic NOAEL of 3,100 mg/m^3 (540 ppm) based on a 13-week inhalation rat study (Nau et al. 1966) was used as the POD to develop the chronic ReV. The critical effects were increase in body weight gain and decrease in white blood cell count.

4.1.6 Dosimetric Adjustments

4.1.7.1 Exposure Duration Adjustments

The POD of 3,100 mg/m^3 (540 ppm) was adjusted from a discontinuous exposure (18 h/d 7 d/week) to continuous exposure concentration.

$$POD_{ADJ} = POD \times (D/24\ h) \times (F/7\ d)$$

where:

D = Exposure duration, h per day

F = Exposure frequency, days per week:

$$POD_{ADJ} = 3{,}100\ mg/m^3 \times (18/24) \times (7/7) = 2{,}325\ mg/m^3$$

4.1.6.2 Default Dosimetry Adjustments from Animal-to-Human Exposure

n-Decane is practically water insoluble. The endpoints studied by Nau et al. (1966) were for systemic rather than POE effects. n-Decane was considered a Category 3 gas. As described in Section 3.1.6.2, the ratio of $((H_{b/g})_A$ /$((H_{b/g})_H)$ is 0.166. The POD_{HEC} from the POD_{ADJ} of 105.357 ppm in the Sung et al. (2010) rat study is:

$POD_{HEC} = POD_{ADJ} \times [(H_{b/g})_A / (H_{b/g})_H]$
$= 2{,}325 \text{ mg/m}^3 \times [17.3/104]$
$= 2{,}325 \text{ mg/m}^3 \times 0.166$
$= 385.95 \text{ mg/m}^3$

4.1.6.3 Adjustments of the POD_{HEC}

The POD_{HEC} of 385.95 mg/m^3 obtained from the default dosimetric adjustment for Category 3 gases was used to derive the chronic ReV and ^{chronic}ESL for n-decane. The following UFs were applied to the POD_{HEC} (Total UF = 360):

- a UF_H of 10 for intraspecies variability,
- a UF_A of 3 for interspecies variability because a default dosimetric adjustment was conducted to account for toxicokinetic differences between animals and humans but not toxicodynamic differences,
- a UF_L of 2 was used to account for extrapolation from a conservative minimal LOAEL to NOAEL,
- a UF_{Sub} of 1 was considered appropriate to account for the use of a subchronic study. A higher value was not used because the exposure duration (18 h/d, 7 d/week for 13 weeks) is considered chronic, and
- a UF_D of 6 was used because only one subchronic inhalation animal study in one species was available and used to evaluate toxicity. A higher value was not used because additional information including subchronic/chronic neurotoxic and reproductive/developmental toxicity is available for similar C_7-C_9 alkanes and C_9-C_{13} mixed alkanes. Confidence in the database is considered low to medium, consistent with TCEQ (2015a). The quality of the key rat study, however, is low to medium.

$\text{Chronic ReV} = POD_{HEC} / (UF_H \times UF_A \times UF_L \times UF_{Sub} \times UF_D)$

$= 385.95 \text{ mg/m}^3 / (10 \times 3 \times 2 \times 1 \times 6)$
$= 385.95 \text{ mg/m}^3 / 360$
$= 1.0721 \text{ mg/m}^3$
$= 1{,}100 \text{ µg/m}^3$ or 190 ppb (rounded to two significant figures)

4.1.7 Summary of the Health-Based Chronic ReV and $^{chronic}ESL_{nonlinear(nc)}$

In deriving the chronic ReV, no numbers were rounded between equations until the ReV was calculated. Once the ReV was calculated, it was rounded to two significant figures. The rounded ReV was then used to calculate the ESL, and the ESL subsequently rounded.

The $^{chronic}ESL_{threshold(nc)}$ of 330 µg/m^3 (57 ppb) for n-decane is based on the acute ReV of 1,100 µg/m^3 (190 ppb) multiplied by a HQ of 0.3 and rounded to two significant figures at the end of all calculations (Table 6).

Table 6 Summary of Chronic ReV and $^{chronic}ESL_{threshold(nc)}$ for n-Decane

Parameter	Values and Description
Study	Nau et al. 1966
Study Quality	Low to medium
Study Population	Rats (43/group)
Exposure Method	0, and 540 ppm
Exposure Duration	18 h/d, 7 d/week for 13 weeks
Critical Effects	Increase in body weight gain and decrease in white blood cell count
POD	3,100 mg/m^3 (540 ppm) (minimal LOAEL)
POD_{ADJ}	2,325 mg/m^3
POD_{HEC}	385.95 mg/m^3
Total UFs	360
Intraspecies UF	10
Interspecies UF	3
LOAEL to NOAEL UF	2
Subchronic to chronic UF	1
Incomplete Database UF *Database Quality*	6 Medium
Chronic ReV (HQ = 1)	**1,100 µg/m^3 (190 ppb)**
$^{chronic}ESL_{threshold(nc)}$ (HQ = 0.3)	**330 µg/m^3 (57 ppb)**

4.2 Carcinogenic Potential

The International Agency for Research on Cancer (IARC) has not evaluated the carcinogenicity of decane. Additionally, no data were found on long-term the carcinogenicity of n-decane. Thus, a $^{chronic}ESL_{nonthreshold(c)}$ cannot be developed.

4.3 Welfare-Based Chronic ESL

No information was found to indicate that special consideration should be given to possible chronic vegetation effects from decane.

4.4 Chronic ReV and $^{chronic}ESL_{threshold(nc)}$

The chronic evaluation resulted in the derivation of the following values for n-decane:

- Chronic ReV = 1,100 µg/m^3 (190 ppb)
- $^{chronic}ESL_{threshold(nc)}$ = 330 µg/m^3 (57 ppb)

For the evaluation of ambient air monitoring data, the chronic ReV of 1,100 µg/m^3 (190 ppb) is used (Table 2). The long-term ESL for air permit reviews is the health-based $^{chronic}ESL_{threshold(nc)}$ of 330 µg/m^3 (57 ppb) (Table 2). The $^{chronic}ESL_{nonlinear(nc)}$ (HQ = 0.3) is not used to evaluate ambient air monitoring data.

4.4.1 Other Decane Isomers

No chronic toxicity data were available describing the potential chronic toxicity of 74 other decane isomers. For the purpose of health effects evaluations for air permit applications and/or ambient air monitoring data, the chronic ReV and $^{chronic}ESL_{threshold(nc)}$ values of 1,100 and 330 µg/m^3, respectively, for n-decane will be used as surrogates.

4.5 Chronic Inhalation Observed Adverse Effect Levels (IOAELs)

The chronic inhalation observed adverse effect level (^{chronic}IOAEL) of 90 ppm for n-decane was based on the $LOAEL_{HEC}$ of 90 ppm (LOAEL x RGDR= 540 ppm x 0.166) for increase in body weight gains and decrease in white blood cell count from the subchronic rat study (Nau et al. 1966). No duration adjustments were made although animal-to-human dosimetric adjustments were performed. Effects occurred in some animals and the ^{chronic}IOAEL represent a concentration at which it is possible that similar effects could occur in some individuals exposed to this level over the same duration as used in the study or longer. Importantly, effects are not a certainty due to potential interspecies and intraspecies differences in sensitivity. The ^{chronic}IOAEL level is provided for informational purposes only (TCEQ 2015a). The ^{chronic}IOAEL for n-decane is:

- n-Decane ^{chronic}IOAEL = 520 mg/m^3 (90 ppm) (rounded to 2 significant figures)

The margin of exposure between the ^{chronic}IOAEL (90 ppm) and the chronic ReV (0.19 ppm) for n-decane is approximately a factor of 474.

Chapter 5 References

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Appendix A. Decane Isomers

Table 7 List of Decane Isomers and CAS Numbers

Name	CAS No.
n-decane	124-18-5
2-methylnonane	871-83-0
3-methylnonane	5911-04-6
4-methylnonane	17301-94-9
5-methylnonane	15869-85-9
3-ethyloctane	5881-17-4
4-ethyloctane	15869-86-0
2,2-dimethyloctane	15869-87-1
2,3-dimethyloctane	7146-60-3
2,4-dimethyloctane	4032-94-4
2,5-dimethyloctane	15869-89-3
2,6-dimethyloctane	2051-30-1
2,7-dimethyloctane	1072-16-8
3,3-dimethyloctane	4110-44-5
3,4-dimethyloctane	15869-92-8
3,5-dimethyloctane	15869-93-9
3,6-dimethyloctane	15869-94-0
4,4-dimethyloctane	15869-95-1
4,5-dimethyloctane	15869-96-2
4-propylheptane	3178-29-8
4-isopropylheptane	52896-87-4
3-ethyl-2-methylheptane	14676-29-0
4-ethyl-2-methylheptane	52896-88-5
5-ethyl-2-methylheptane	13475-78-0
3-ethyl-3-methylheptane	17302-01-1
4-ethyl-3-methylheptane	52896-89-6
3-ethyl-4-methylheptane	52896-91-0
4-ethyl-4-methylheptane	17302-04-4
3-ethyl-5-methylheptane	52896-90-9
2,2,3-trimethylheptane	52896-92-1
2,2,4-trimethylheptane	14720-74-2
2,2,5-trimethylheptane	20291-95-6
2,2,6-trimethylheptane	1190-83-6
2,3,3-trimethylheptane	52896-93-2
2,3,4-trimethylheptane	52896-95-4
2,3,5-trimethylheptane	20278-85-7
2,3,6-trimethylheptane	4032-93-3
2,4,4-trimethylheptane	4032-92-2
2,4,5-trimethylheptane	20278-84-6
2,4,6-trimethylheptane	2613-61-8
2,5,5-trimethylheptane	1189-99-7
3,3,4-trimethylheptane	20278-87-9
3,3,5-trimethylheptane	7154-80-5
3,4,4-trimethylheptane	20278-88-0
3,4,5-trimethylheptane	20278-89-1
3-isopropyl-2-methylhexane	62016-13-1
3,3-diethylhexane	17302-02-2
3,4-diethylhexane	19398-77-7
3-ethyl-2,2-dimethylhexane	20291-91-2
4-ethyl-2,2-dimethylhexane	52896-99-8
3-ethyl-2,3-dimethylhexane	52897-00-4
4-ethyl-2,3-dimethylhexane	52897-01-5
3-ethyl-2,4-dimethylhexane	7220-26-0
4-ethyl-2,4-dimethylhexane	52897-03-7
3-ethyl-2,5-dimethylhexane	52897-04-8
4-ethyl-3,3-dimethylhexane	52897-05-9
3-ethyl-3,4-dimethylhexane	52897-06-0
2,2,3,3-tetramethylhexane	13475-81-5
2,2,3,4-tetramethylhexane	52897-08-2
2,2,3,5-tetramethylhexane	52897-09-3
2,2,4,4-tetramethylhexane	51750-65-3
2,2,4,5-tetramethylhexane	16747-42-5
2,2,5,5-tetramethylhexane	1071-81-4
2,3,3,4-tetramethylhexane	52897-10-6
2,3,3,5-tetramethylhexane	52897-11-7
2,3,4,4-tetramethylhexane	52897-12-8
2,3,4,5-tetramethylhexane	52897-15-1
3,3,4,4-tetramethylhexane	13475-81-5
3-isopropyl-2,4-dimethylpentane	13475-79-1
3,3-diethyl-2-methylpentane	52897-16-2
3-ethyl-2,2,3-trimethylpentane	52897-17-3
3-ethyl2,2,4-trimethylpentane	52897-18-4
3-ethyl-2,3,4-trimethylpentane	52897-19-5
2,2,3,3,4-pentamethylpentane	16747-44-7
2,2,3,4,4-pentamethylpentane	16747-45-8

Appendix B. Determination of POD_{HEC} for LOAELs

B.1 POD for LOAEL from the Kjærgaard et al. (1989) Study

B.1.1 Exposure Duration Adjustments

The POD of 100 ppm for LOAEL from the Kjærgaard et al. (1989) study was adjusted from 6-h exposure to 1-h exposure concentration using Haber's rule as modified by ten Berge with a default value of "n"=3 (TCEQ 2015a).

$$POD_{ADJ} = C_2 = [(C_1)^3 \times (T_1 / T_2)]^{1/3}$$
$$= [(100\ ppm)^3 \times (6\ h/1\ h)]^{1/3}$$
$$= 181.712\ ppm$$

B.1.2 Default Dosimetry Adjustments from Animal-to-Human Exposure

The POD_{ADJ} of 181.712 ppm was based on human inhalation exposure and thus, no dosimetry adjustment from animal-to-human exposure. The POD_{HEC} for the LOAEL-based POD_{ADJ} is 181.712 ppm.

B.2. POD for LOAEL from the Lammers et al. (2011) Study

B.2.1 Exposure Duration Adjustments

The POD of 860 ppm (5,000 mg/m^3 from the Lammers et al. (2011) study was adjusted from 8-h exposure to 1-h exposure concentration using Haber's rule as modified by ten Berge with a default value of "n"=3 (TCEQ 2015a).

$$POD_{ADJ} = C_2 = [(C1)^3 \times (T1 / T2)]^{1/3}$$
$$= [(860\ ppm)^3 \times (8\ h/1\ h)]^{1/3}$$
$$= 1{,}719.994\ ppm$$

B.2.2 Default Dosimetry Adjustments from Animal-to-Human Exposure

n-Decane is practically water insoluble. Acute exposures to n-decane cause neurobehavioral effects which are systemic effects and thus, n-decane was considered a Category 3 gas. For Category 3 gases, the default dosimetric adjustment from an animal concentration to a POD_{HEC} is conducted using the following equation:

$$POD_{HEC} = POD_{ADJ} \times [(H_{b/g})_A / (H_{b/g})_H]$$

where: $H_{b/g}$ = ratio of the blood:gas partition coefficient
A = animal

H = human

The measured blood/air partition coefficients for n-decane in rats ($(H_{b/g})_A$) and in humans ($(H_{b/g})_H$) (RGDR) are 17.3 and 104, respectively (Meulenberg and Vijverberg 2000). The resulting POD_{HEC} for the LOAEL-based POD_{ADJ} of 520 ppm in the Lammers et al. (2011) rat study is:

POD_{HEC} for LOAEL = POD_{ADJ} x $[(H_{b/g})_A / (H_{b/g})_H]$
= 1,719.994 ppm x [17.3/104]
= 1,719.994 ppm x 0.166
= 285.519 ppm