Propionaldehyde; CASRN 123-38-6

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR PROPIONALDEHYDE CASRN 123-38-6

File First On-Line 09/30/2008

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	qualitative discussion	09/30/2008
Inhalation RfC (I.B.)	yes	09/30/2008
Carcinogenicity Assessment (II.)	yes	09/30/2008

I. HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

No current IRIS health assessment for propionaldehyde is available. No data were available for derivation of an RfD. An RfC of 8 x 10^{-3} mg/m^3 was derived from a $BMCL_{10HEC}$ of 8 mg/m^3 based on an increased incidence of atrophy of the olfactory epithelium in male rats, and application of the total UF of 1,000 (Union Carbide, 1993). Confidence in the RfC is judged to be low to medium.

I.A. REFERENCE DOSE (RfD) FOR ORAL EXPOSURE

Substance Name — Propionaldehyde
CASRN — 123-38-6
Section I.A. Last Revised — 09/30/2008

The RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily

oral exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfD is intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action. It is expressed in units of mg/kg-day. Please refer to the guidance documents at http://www.epa.gov/iris/backgrd.html for an elaboration of these concepts. Because RfDs can be derived for the noncarcinogenic health effects of substances that are also carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.A.1. ORAL RfD SUMMARY

No human or animal, subchronic or chronic oral studies were identified for propionaldehyde.

Critical Effect	Point of Departure	UF	RfD
No oral studies available.	--	--	Not derived.

I.A.2. PRINCIPAL AND SUPPORTING STUDIES

Not applicable.

I.A.3. UNCERTAINTY FACTORS

Not applicable.

I.A.4. ADDITIONAL STUDIES/COMMENTS

Not applicable.

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.A.5. CONFIDENCE IN THE ORAL RfD

Not applicable.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.A.6. EPA DOCUMENTATION AND REVIEW OF THE ORAL RfD

Source Document — U.S. EPA (2008)

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Propionaldehyde; CASRN 123-38-06* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF)***.

Agency Completion Date -- 09/30/2008

I.A.7. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

I.B. REFERENCE CONCENTRATION (RfC) FOR INHALATION EXPOSURE

Substance Name — Propionaldehyde
CASRN — 128-38-6
Section I.B. Last Revised — 09/30/2008

The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a continuous inhalation exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC considers toxic effects for both the respiratory system (portal of entry) and for effects peripheral to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in units of mg/m^3) is analogous to the oral RfD and is similarly intended for use in risk assessments for health effects known or assumed to be produced through a nonlinear (presumed threshold) mode of action.

Inhalation RfCs are derived according to *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). Because RfCs can also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is essential to refer to other sources of information concerning the carcinogenicity of this chemical substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file.

I.B.1. INHALATION RfC SUMMARY

Critical Effect	Point of Departure	UF	RfC
Atrophy of olfactory epithelium **Subchronic inhalation study in rats** **Union Carbide, 1993**	$BMCL_{10HEC}$: 8 mg/m^3	1000	8×10^{-3} mg/m^3

I.B.2. PRINCIPAL AND SUPPORTING STUDIES

The Union Carbide (1993) study was selected as the principal study for derivation of the RfC. In this study, young adult male and female CD rats (15/sex/group) were exposed to 0, 150, 750, or 1500 ppm (0, 357, 1,785, or 3,570 mg/m^3) propionaldehyde for 6 hours/day, 7 days/week via whole-body inhalation, during a 2-week premating period and a 14-day (maximum) mating phase. The mated females were exposed daily through GD 20 only for a minimum of 35 days and a maximum of 48 days depending upon when they mated (average exposure period ~ 38 days). The females were then allowed to deliver their litters naturally and raise their offspring until postnatal day (PND) 4 both free of exposure to propionaldehyde. The males continued to be exposed for a total of 52 exposures until sacrifice in week 7. Clinical observations were made daily, following exposure, and body weight and food consumption were measured at regular intervals throughout the study. Offspring body weight, viability, and disposition were monitored from birth until PND 4. Following the last exposure, males were fasted and blood samples were obtained for clinical pathology analyses prior to necropsy. On PND 4, necropsies were performed on adult females, and a number of organs and tissues, including at least two sections of the nasal cavity (sectioning details not provided), were examined histologically. The offspring were examined externally and sacrificed without pathologic evaluation.

No exposure-related clinical signs were noted in the adult females. During the first week of exposure to 750 and 1,500 ppm, body weight gains were decreased to approximately 60 and

71% ($p < 0.01$), respectively, of controls, and food consumption was decreased by approximately 7% ($p < 0.05$) of controls at both concentrations. No differences were observed during the second week of exposure. During gestation, body weight (over GDs 0-14) and food consumption (over GDs 0-21) were decreased in the high exposure group compared with controls, but no significant differences in body weight gain were observed. At sacrifice, no gross lesions attributable to propionaldehyde exposure were found. However, microscopic examination of the nasal cavity revealed propionaldehyde-induced vacuolization of the olfactory epithelium in the 150 and 750 ppm exposure groups and atrophy of the olfactory epithelium in the 750 and 1,500 ppm exposure groups. These effects were noted to be localized to the dorsal anterior two sections of the nasal cavity. The incidence of atrophy was 0/15, 0/15, 2/15, and 15/15 at 0, 150, 750, and 1500 ppm, respectively (see Table 4-1 of the toxicology review). The severity of this nasal lesion increased with exposure concentration being minimal to mild at 750 ppm and moderate to marked at 1500 ppm. No evidence of squamous metaplasia was found in olfactory or respiratory epithelium. Low incidences of minimal to mild rhinitis involving the respiratory epithelium were also noted at 150, 750, and 1,500 ppm. No significant effects of exposure on any of the reproductive parameters assessed were found. Litter size and viability were similar among the groups. Pup body weights on the day of birth and PND 4 were not affected by exposure, although at the high concentration only body weight gain for that period was significantly depressed ($p < 0.05$, -0.8 g) compared with controls. The biological significance of this finding is difficult to assess since changes in absolute body weight were not demonstrated and the time period of observation was relatively short.

The adult males did not display any overt signs of toxicity at any time during the study. Body weight, weight gain, clinical observation, and food consumption were similar among all exposure groups and controls. Hematology and clinical chemistry analyses revealed elevated erythrocyte counts, with a corresponding increase in hemoglobin and hematocrit values and an increase in monocytes in the males exposed to 1,500 ppm. These effects were considered to be consistent with and indicative of dehydration. At necropsy (examination performed as per the adult females), no gross lesions were found that could be attributable to propionaldehyde exposure. However, similar to effects in the females, microscopic examination revealed exposure-related effects in the olfactory epithelium of the nasal cavity that consisted of vacuolization and atrophy in the low, intermediate, and high exposure groups. These effects were noted to be localized to the dorsal anterior two sections of the nasal cavity. The incidence of atrophy was 0/15, 2/15, 10/15, and 15/15 at 0, 150, 750, and 1,500 ppm, respectively.

The severity of this nasal lesion increased with exposure concentration being minimal at 150 ppm, minimal to moderate at 750 ppm, and mild to marked at 1,500 ppm. Squamous metaplasia of the respiratory epithelium was reported in one male from the 750 ppm group and two males from the 1,500 ppm group. An increased incidence of minimal to moderate rhinitis

involving the respiratory epithelium was also noted at 750 and 1,500 ppm. The results of this study indicate a lowest-observed-adverse-effect level (LOAEL) for portal-of-entry toxicity of 150 ppm as a result of olfactory atrophy graded by Union Carbide (1993) as being of minimal severity by the study authors and supported by the presence of vacuolization.

The critical endpoint chosen for analysis from this study was the incidence of atrophy of the olfactory epithelium in male rats. Furthermore, the critical effect in male rats was chosen as it is biologically relevant effect, exhibited a concentration-response relationship, and was observed at the lowest exposure concentration tested (150 ppm). The atrophy observed at the lowest exposure concentration was of minimal severity and not noted in females, possibly as a result of the greater exposure duration of the male rats compared to the female rats in this study. Similarly, the atrophy observed at the middle exposure concentration (750 ppm) was characterized as being of minimal to moderate severity. The induction of nasal lesions by propionaldehyde is consistent with the irritant properties and the portal-of-entry effects observed in studies conducted for other aldehydes (e.g., acetaldehyde, isobutyraldehyde and formaldehyde).

I.B.3. UNCERTAINTY FACTORS

UF = 1,000

A default UF_A of 3 ($10^{1/2}$) was applied to account for interspecies (animal-to-human extrapolation). This factor incorporates two areas of uncertainty given equal weight: pharmacokinetics and pharmacodynamics. Because the pharmacokinetic component was addressed in this assessment by the calculation of the HEC by applying the RGDR in extrapolating from animals to humans according to the procedures in the RfC methodology (U.S. EPA, 1994), only the pharmacodynamic component of this factor of uncertainty remains.

A default UF_H of 10 was applied for intraspecies uncertainty to account for human variability and sensitive subpopulations as there was very limited information available to definitively address the variability in the severity or range of response from propionaldehyde exposure among individuals, and available data suggest there are differences among humans in metabolism of propionaldehyde. Recent PBPK modeling investigating the impact of ALDH2 polymorphisms on rat and human nasal tissue dosimetry demonstrated a negligible impact on olfactory tissue dose (Teeguarden et al., 2008). Additionally, application of this UF considers the potential sensitivity of children and individuals with conditions such as asthma.

A default UF_S of 10 was applied to account for adjustment from subchronic to chronic duration. A subchronic study was used to derive the RfC, as no other supportive studies of similar or longer durations were available for propionaldehyde.

A UF_D of 3 ($10^{1/2}$) was applied to account for database deficiencies. The database for propionaldehyde consists of several short-term inhalation animal studies, ranging from 6 days to 7 weeks in duration, and two reproductive/developmental toxicity studies. The database is lacking a multigeneration reproductive toxicity study. Although the principal study used for the RfC derivation was a reproductive/developmental study (Union Carbide, 1993), this study provided limited reproductive and developmental information, since the pups were sacrificed on PND 4 and pathology in the pups was not evaluated; only an external examination for the presence of malformations was performed. Although limited nasal sectioning (i.e. 2-3 sections compared to typical 4-6) was performed at necropsy, the critical effect identified was atrophy of the olfactory epithelium in adult male rats (also observed in females), which is concordant with the portal-of-entry effects attributable to the aldehydes acrolein, formaldehyde, acetaldehyde, and isobutyraldehyde as well as other irritant gases. In addition, none of these aldehydes appear to induce direct systemic effects, as measured by clinical chemistry and pathology, at exposure concentrations that produce initial portal-of-entry effects. Similarly, propionaldehyde would not be anticipated to have significant systemic distribution based on its deposition, solubility, and reactivity in the respiratory tract. The uptake of propionaldehyde in the upper respiratory tract measured in dogs is approximately 70-80% (Egle, 1972a). In the same study, moderate to high respiratory tract uptake was observed for both acrolein (~80%) and formaldehyde (near 100%). In the rat, acetaldehyde uptake in the upper respiratory tract averaged from 76 to 26% over a concentration range of 1-1,000 ppm (Stanek and Morris, 1999; Morris and Blanchard, 1992). In general, the toxicological information and limited kinetic information available for propionaldehyde is consistent with other structurally related aldehydes and provides support for the critical effect chosen. However, the lack of a multigeneration reproductive toxicity study warrants the application of a UF_D of 3.

No LOAEL to NOAEL UF was applied since BMC analysis was used to determine the POD, and this factor was addressed as one of the considerations in selecting the BMR. Based on the data, a BMR of 10% change in the incidence of minimal olfactory atrophy was selected under an assumption that it represents a minimal biologically significant change.

I.B.4. ADDITIONAL STUDIES/COMMENTS

The pattern of nasal lesions observed after exposure to propionaldehyde is very similar to that observed after exposure to structurally-related aldehydes. For example, inhalation exposure to acetaldehyde over a period for up to 28 months produced olfactory degeneration/atrophy with and without hyperplasia/metaplasia at 4 weeks, followed by progression to focal basal cell

hyperplasia of the olfactory epithelium and squamous metaplasia of the respiratory epithelium at 12-15 months and finally by squamous cell carcinomas and adenocarcinomas at 16-28 months (Woutersen et al., 1986, 1984; Appelman et al., 1982). The severity and incidence of these nasal effects were dependent on exposure concentration and duration. A similar pattern and progression of nasal olfactory lesions were observed in rats exposed to acetaldehyde for up to 65 exposure days (Dorman et al., 2008). Olfactory epithelial degeneration, noted as the most sensitive endpoint observed, increased in incidence and severity with both exposure concentration and duration. The presence of vacuolization was also noted, but the severity was not graded. In rats exposed chronically to isobutyraldehyde, nonneoplastic lesions in the nose consisted of squamous metaplasia of the respiratory epithelium at concentrations ≥500 ppm, degeneration of the olfactory epithelium at 2000 ppm, and suppurative inflammation at 2,000 ppm (NTP, 1999). No increases in neoplastic nasal lesions were observed in this study. Exposure to formaldehyde for 13 weeks also produces similar effects in the nasal respiratory epithelium, consisting of epithelial hyperplasia, squamous metaplasia, and increases in cell proliferation at concentrations as low as 3 ppm (Zwart et al., 1988). Formaldehyde-induced nasal tumors are reported at concentrations ≥6 ppm after chronic exposure (Monticello et al., 1996).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

I.B.5. CONFIDENCE IN THE INHALATION RfC

Study — Low to Medium
Data Base — Low
RfC — Low to Medium

Confidence in the principal study (Union Carbide, 1993) is judged to be low to medium because few details were provided specific to the study results. In addition, the key study provided limited developmental information as the pups were sacrificed on PND 4 and pathology was not evaluated; only an external examination for the presence of malformations was performed. However, the critical effect identified was atrophy of the olfactory epithelium in adult male rats (also observed in females), which is concordant with the portal-of-entry effects attributable to irritant gases and other aldehydes. Thus, this endpoint is supported by the aldehyde inhalation exposure-effects database as a whole. Confidence in the critical effect identified in the principal study is medium. Confidence in the overall database specific to propionaldehyde is low because there are no additional and/or supporting subchronic or chronic animal studies available to evaluate and support the concentration-response effect of propionaldehyde on multiple endpoints. Therefore, confidence in the RfC is judged to be low to medium.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document - U.S. EPA (2008)

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Propionaldehyde* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF).***

Agency Completion Date -- 09/30/2008

I.B.7. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name — Propionaldehyde
CASRN — 123-38-6
Section II. Last Revised — 09/30/2008

This section provides information on three aspects of the carcinogenic assessment for the substance in question: the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral and inhalation exposure. Users are referred to Section I of this file for information on long-term toxic effects other than carcinogenicity.

The rationale and methods used to develop the carcinogenicity information in IRIS are described in the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a) and the *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*

(U.S. EPA, 2005b). The quantitative risk estimates are derived from the application of a low-dose extrapolation procedure, and are presented in two ways to better facilitate their use. First, route-specific risk values are presented. The "oral slope factor" is a plausible upper bound on the estimate of risk per mg/kg-day of oral exposure. Similarly, a "unit risk" is a plausible upper bound on the estimate of risk per unit of concentration, either per µg/L drinking water (see Section II.B.1.) or per $\mu g/m^3$ air breathed (see Section II.C.1.). Second, the estimated concentration of the chemical substance in drinking water or air when associated with cancer risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000 is also provided.

II.A. EVIDENCE FOR HUMAN CARCINOGENICITY

II.A.1. WEIGHT-OF-EVIDENCE CHARACTERIZATION

In accordance with the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a), there is "inadequate information to assess the carcinogenic potential" for propionaldehyde. No human health effects data or chronic animal bioassay studies are available that assess the carcinogenic effects of propionaldehyde.

The genotoxicity of propionaldehyde has been studied in bacteria and a number of mammalian cells in vitro. Propionaldehyde was found to be mutagenic in *Salmonella typhimurium* strain TA1534 (Sampson and Bobik, 2008) and nonmutagenic in all other strains tested (Dillon et al., 1998; Aeschbacher et al., 1989; Mortelmans et al., 1986), but produced concentration-related increases in HGPRT and ouabain mutants in V79 hamster cells (Brambilla et al., 1989). These effects, however, were associated with decreases in cell viability in these test systems. Smith et al. (1990) determined that propionaldehyde was not mutagenic at the HGPRT locus in V79 hamster cells exposed to lower, noncytotoxic concentrations. Propionaldehyde produced a concentration-related increase in chromosome aberrations in Chinese hamster embryonic cells (Furnus et al., 1990) and chromosome breaks in CHO cells (Seoane and Dulout, 1994). In addition, propionaldehyde induced a concentration-related increase in UDS in rat, but not human, hepatocytes (Martelli, 1997; Martelli et al., 1994) and a weak, concentration-related increase in DPXs in cultured human lymphoma cells (Costa et al., 1997). For formaldehyde, increases in DPXs serve as an important aspect in describing the dosimetry and the carcinogenic mode of action for nasal tumors (Conolly, et al., 2000; Casanova et al., 1994). Although the information provided in the in vitro studies suggests that propionaldehyde is DNA reactive, information from in vivo animal bioassay studies is unavailable. This overall lack of information represents a data gap and does not allow for either a quantitative or a qualitative assessment of the carcinogenic potential of propionaldehyde or a definitive statement concerning its mutagenic potential.

It is important to note that inhalation exposure to propionaldehyde produced a low incidence of respiratory epithelium squamous metaplasia in male rats in the intermediate and high exposure groups (Union Carbide, 1993). Although this alteration may be viewed as an adaptive response typical of nasal epithelial tissues in response to continued irritant insult, the lesion may become part of a progression from nasal tissue injury and toxicity (e.g., epithelial degeneration and atrophy) to hyperplasia to increased cell proliferation and lastly to nasal tumorigenesis (Renne et al., 2007; Boorman et al., 1990). Squamous metaplasia is also noted in studies examining the nasal effects of both acetaldehyde and formaldehyde in which marked to severe metaplasia and/or hyperplasia and increases in cell proliferation are observed prior to nasal tumor formation during chronic exposure (Monticello et al., 1996; Zwart et al., 1988; Woutersen et al., 1986, 1984; Appelman et al., 1982). A similar pattern of nasal lesions were also noted in a 2- year chronic study evaluating the toxicology and carcinogenesis of isobutyraldehyde, but no increases in neoplastic lesions were observed (NTP, 1999). In contrast, the exposure concentrations required to induce similar nasal effects were higher compared to formaldehyde and acetaldehyde. Thus, the pattern of nasal tissue effects and the carcinogenicity of related aldehydes raise concern. However, the more specific alterations observed for related aldehydes, such as squamous metaplasia with atypia and disorganization, concurrent hyperplasia, changes in cell proliferation, and tumor formation in nasal tissues, were not observed after exposure to propionaldehyde (Union Carbide, 1993). Therefore, as it relates to the effects observed after exposure to propionaldehyde, the presence of squamous metaplasia alone is considered to be a nonneoplastic lesion in nasal tissue and is of limited quantitative use in assessing cancer risk.

For more detail on Characterization of Hazard and Dose Response, exit to the toxicological review, Section 6 (PDF).

For more detail on Susceptible Populations, exit to the toxicological review, Section 4.7 (PDF).

II.A.2. HUMAN CARCINOGENICITY DATA

No information is available on the carcinogenicity of propionaldehyde in humans.

II.A.3. ANIMAL CARCINOGENICITY DATA

No information is available on the carcinogenicity of propionaldehyde in animals.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Not applicable.

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

II.B.1. SUMMARY OF RISK ESTIMATES

Not applicable.

II.B.2. DOSE-RESPONSE DATA

Not applicable.

II.B.3. ADDITIONAL COMMENTS

Not applicable.

II.B.4. DISCUSSION OF CONFIDENCE

Not applicable.

II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

II.C.1. SUMMARY OF RISK ESTIMATES

Not applicable.

II.C.2. DOSE-RESPONSE DATA

Not applicable.

II.C.3. ADDITIONAL COMMENTS

Not applicable.

II.C.4. DISCUSSION OF CONFIDENCE

Not applicable.

II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA (2008)

This document has been reviewed by EPA scientists, interagency reviewers from other federal agencies, and the public, and peer reviewed by independent scientists external to EPA. A summary and EPA's disposition of the comments received from the independent external peer reviewers and from the public is included in Appendix A of the *Toxicological Review of Propionaldehyde* (U.S. EPA, 2008). ***To review this appendix, exit to the toxicological review, Appendix A, Summary Of External Peer Review And Public Comments And Disposition (PDF)***.

II.D.2. EPA REVIEW

Agency Completion Date -- 09/30/2008

II.D.3. EPA CONTACTS

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Propionaldehyde
CASRN — 123-38-6

VI.A. ORAL RfD REFERENCES

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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VII. REVISION HISTORY

Substance Name — Propionaldehyde
CASRN — 123-38-6
File First On-Line - 09/30/2008

Date	Section	Description
09/30/2008	I., II.	RfD, RfC, and cancer assessment first on line

VIII. SYNONYMS

Substance Name — Propionaldehyde
CASRN — 123-38-6
Section VIII. Last Revised — 09/30/2008

- Propanal
- Propionic Aldehyde
- Methylacetaldehyde
- Propyl Aldehyde
- Propaldehyde
- Propylic Aldehyde