

This document is a **Final Agency/Interagency Review draft**. It has not been formally released by the U.S. Environmental Protection Agency and should not at this stage be construed to represent Agency position on this chemical. It is being circulated for review of its technical accuracy and science policy implications.

Substance code
2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD); CASRN 1746-01-6
00/00/0000

Human health assessment information on a chemical substance is included in IRIS only after a comprehensive review of toxicity data by U.S. EPA health scientists from several program offices, regional offices, and the Office of Research and Development. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the positions that were reached during the review 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 at <http://www.epa.gov/iris/backgrd.htm>.

STATUS OF DATA FOR 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

File First On-Line __/__/__

<u>Category (section)</u>	<u>Status</u>	<u>Last Revised</u>
Chronic Oral RfD Assessment (I.A.)	on-line	00/00/0000
Chronic Inhalation RfC Assessment (I.B.)	not available	00/00/0000
Carcinogenicity Assessment (II.)	message	00/00/0000

I. HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

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

Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
CASRN — 1746-01-6
Section I.A. Last Revised—

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](#) for an elaboration of these concepts. Because RfDs can be derived for the

1 noncarcinogenic health effects of substances that are also carcinogens, it is essential to refer to
2 other sources of information concerning the carcinogenicity of this chemical substance. If the
3 U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that
4 evaluation will be contained in Section II of this file.

5
6 There was no previous RfD for TCDD on the IRIS database.

7
8 For the assessment of human health risks posed by exposure to mixtures of TCDD and dioxin-
9 like compounds (DLCs), including polychlorinated dibenzo-*p*-dioxins, polychlorinated
10 dibenzofurans, and dioxin-like polychlorinated biphenyls, and when data on a whole mixture or a
11 sufficiently similar mixture are not available, EPA recommends use of the consensus mammalian
12 TEF values developed by the World Health Organization ([U.S. EPA, 2010](#); [Van den Berg et al.,
13 2006](#)).

14 15 I.A.1. CHRONIC ORAL RfD SUMMARY

<u>Co-Critical Effects</u>	<u>Point of Departure</u>	<u>UF</u>	<u>Chronic RfD</u>
Decreased sperm count and motility in men exposed to TCDD as boys	LOAEL[adjusted]: 0.020 ng/kg-day ^a (2.0 x 10 ⁻⁸ mg/kg-day)	30	7 x 10 ⁻¹⁰ mg/kg-day

Epidemiologic cohort
study

Mocarelli et al., ([2008](#))

Increased TSH in neonates LOAEL[adjusted]: 0.020 ng/kg-day^a
(2.0 x 10⁻⁸ mg/kg-day)

Epidemiologic cohort
study

Baccarelli et al., ([2008](#))

17 ^aConversion Factors and Assumptions—for both studies, PBPK modeling was used to estimate oral intakes from
18 TCDD exposures reported as serum concentrations. The details are presented in Methods of Analysis below. ^bData
19 were not amenable to Benchmark Dose Modeling.

20 21 I.A.2. PRINCIPAL AND SUPPORTING STUDIES (ORAL RfD)

22
23 *EPA's Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments,
24 Volume 1 (Reanalysis Volume 1)* reviews and summarizes the available data on noncancer effects
25 caused by TCDD (for summary of the noncancer effects, see U.S. EPA (201X), Section 4).

26 Adverse noncancer effects associated with oral TCDD exposure include hepatic, neurological,
27 immunological, reproductive, endocrine, and developmental effects. As recommended by A
28 *Review of the Reference Dose and Reference Concentration Process* ([U.S. EPA, 2002](#)), the RfD
29 was developed based on consideration of all relevant and appropriate endpoints carried through
30 to the derivation of “candidate” RfDs. Candidate RfDs were developed for all endpoints on the
31 basis of physiologically based pharmacokinetic (PBPK) model-derived internal dose (U.S. EPA
32 (201X), Sections 3 and 4).

1
2 Two human epidemiologic studies (Baccarelli et al., (2008); Mocarelli et al., (2008)) were
3 chosen as the basis for the RfD. Both of these studies are of a human population that was
4 exposed to TCDD through an industrial accident in Seveso, Italy in 1976. Baccarelli et al.
5 (2008) reported increased levels of TSH in newborns exposed to TCDD in utero, indicating a
6 possible dysregulation of thyroid hormone metabolism. The study authors related TCDD
7 concentrations in maternal plasma to neonatal TSH levels using a multivariate linear regression
8 model adjusting for a number of covariates including gender, birth weight, birth order, maternal
9 age, hospital, and type of delivery. Based on this regression modeling, EPA defined the LOAEL
10 for Baccarelli et al.(2008) to be the maternal TCDD LASC of 235 ppt (at delivery)
11 corresponding to a neonatal TSH level of 5 μ U/mL. Using the Emond human PBPK model
12 (Emond et al., 2005), the corresponding daily oral intake at the LOAEL is estimated to be
13 0.020 ng/kg day (see *Reanalysis Volume 1*, Section 4.2.3.1.). The World Health Organization
14 (WHO/UNICEF/ICCIDD, 1994) established the 5 μ U/mL standard as an indicator of potential
15 iodine deficiency and potential thyroid problems in neonates. The 5 μ U/mL limit for TSH
16 measurements in neonates was recommended by WHO (1994) for use in population surveillance
17 programs as an indicator of iodine deficiency disease (IDD). For TCDD, the toxicological
18 concern is not likely to be iodine uptake inhibition, but rather increased metabolism and
19 clearance of the thyroid hormone, thyroxine (T4). An increased TSH level is an indicator of a
20 potential decrease in circulating T4 levels, which could eventually lead to neurological
21 deficiencies. Adequate levels of thyroid hormone are essential in the newborn and young infant
22 as this is a period of active brain development(Zoeller and Rovet, 2004; Glinoyer and Delange,
23 2000).

24
25 Mocarelli et al. (2008) reported decreased sperm concentrations and decreased motile sperm
26 counts in men who were 1–9 years of age at the time of the Seveso accident (initial TCDD
27 exposure event) in 1976. These results identify the first 10 years of life as a critical window of
28 susceptibility for TCDD-induced sperm effects. Serum TCDD levels (as LASC) were measured
29 in samples collected within one year of the initial exposure. Serum TCDD levels and
30 corresponding responses were reported by quartile, with a reference group of individuals
31 assigned a TCDD LASC value of 15 ppt (the mean of the TCDD LASC reported in individuals
32 outside the contaminated area). In the reference group, mean sperm concentrations and motile
33 sperm counts were approximately 74 million sperm/mL and 41%, respectively. The lowest
34 exposed group (1st-quartile) TCDD LASC mean was 68 ppt. In the 1st quartile, mean sperm
35 concentrations of approximately 55 million sperm/mL and motile sperm counts of approximately
36 36% were reduced about 25 and 12%, respectively, from the reference group. Further decreases
37 in these measures in the groups exposed to more than 68 ppt was minimal, with maximum
38 reductions of 33% for sperm concentration and 25% for progressive sperm motility in the 4th and
39 3rd quartiles, respectively.

40
41 The lowest exposure group (TCDD LASC = 8 ppt) in Mocarelli et al. (2008) is designated as a
42 LOAEL. Using the Emond PBPK model (Emond et al., 2005), EPA estimated both the peak
43 initial exposure and the average exposure over the critical window. Because of the uncertainty in
44 the influence of the peak exposure relative to the average exposure over the entire 10-year
45 window, the LOAEL of 0.020 ng/kg-day was calculated as the average of the peak exposure
46 (0.032 ng/kg-day) and the average exposure across the critical exposure window
47 (0.008 ng/kg-day) (see *Reanalysis Volume 1*, Section 4.2.3.2).

48

Supporting Studies

Mocarelli et al. (2011), also reported reductions in sperm concentrations for men who were exposed to TCDD in utero and by lactation; the effects were very similar to those in men exposed as boys from the coprincipal study by Mocarelli et al. (2008). The maternal TCDD exposures associated with the effects reported by Mocarelli et al. (2011) are somewhat lower than the exposures in the LOAEL group in Mocarelli et al. (2008), but quantifying either a NOAEL or LOAEL is complicated by large uncertainties arising from relatively-high background exposures to dioxin-like compounds (DLCs) in this study population. Reduced sperm concentration associated with TCDD exposure has been reported in rodent studies (Simanainen et al., 2004; Latchoumycandane et al., 2002) and overt male reproductive effects have also been shown for rodents (Ikeda et al., 2005; Ohsako et al., 2001) (see *Reanalysis Volume 1*, Section 4.3.6.2.). In addition to the effects of TCDD on neonatal TSH levels, TCDD has been associated with reductions in thyroid hormone (thyroxine, T4) levels in a number of animal studies (Crofton et al., 2005; Seo et al., 1995; Sewall et al., 1995; Van Birgelen et al., 1995a; Van Birgelen et al., 1995b) NTP (2006) (see *Reanalysis Volume 1*, Section 4.3.6.1.)

Methods of Analysis

For both coprincipal studies, PBPK modeling was used to estimate continuous daily oral intakes from TCDD (lipid-adjusted) serum concentrations (LASC) (see *Reanalysis Volume 1*, Section 4.2.3). Additional PBPK modeling was conducted for Mocarelli et al. (2008) to adjust for the changing LASC time profile, characterized by a high initial exposure followed by slow elimination of TCDD over an average 3-year period. For Mocarelli et al. (2008), the lowest exposure group (TCDD LASC = 68 ppt) was designated as a LOAEL and the Emond PBPK model (Emond et al., 2005) was used to determine a LOAEL[adjusted] of 0.020 ng/kg-day. The LOAEL[adjusted] was calculated as the average of the peak-exposure intake and the average intake over a critical exposure window of susceptibility of 10 years (the first 10 years of life). For Baccarelli et al. (2008), the maternal TCDD LASC of 235 ppt at delivery associated with a neonatal TSH level of 5 µU/ml, as modeled by the study authors, was determined to be a LOAEL; a corresponding 30-year continuous oral intake of 0.020 ng/kg-day was estimated using the Emond PBPK model (Emond et al., 2005) and assigned as the LOAEL[adjusted]. TCDD LASC was assumed to be constant during pregnancy.

___ I.A.3. UNCERTAINTY FACTORS

UF = 30

An uncertainty factor (UF) of 10 was selected for LOAEL-to-NOAEL extrapolation as a NOAEL could not be identified for either co-principal study.

An UF of 3 was selected for interindividual variability to account for human-to-human variability in susceptibility. The individuals evaluated in the two principal studies included infants (exposed in utero) and adults who were exposed when they were less than 10 years of age, groups that are considered to represent sensitive lifestages.. These studies considered together associate TCDD exposures with health effects in potentially vulnerable lifestage subgroups. An UF of 1 was not applied because the sample sizes for the lifestages studied were relatively small, which, combined with uncertainty in exposure estimation, may not fully capture

1 the range of interindividual variability. In addition, potential chronic effects were not fully
2 elucidated for humans and could possibly be more sensitive.

3
4 An UF for interspecies extrapolation was not applied because human data were utilized for
5 derivation of the RfD.

6
7 An UF for study duration was not applied because, although chronic effect levels are not well
8 defined for humans, animal bioassays indicate that duration of exposure is not likely to be a
9 determining factor in toxicological outcomes. Developmental effects and other short-term
10 effects occur at doses similar to effects noted in chronic studies.

11
12 An UF for database deficiencies was not applied because the database for TCDD contains an
13 extensive range of human and animal studies that examine a comprehensive set of endpoints.
14 There is no evidence to suggest that additional data would result in a lower RfD.

15 16 I.A.4. ADDITIONAL STUDIES/COMMENTS

17
18 Other outcomes reported in humans include female reproductive effects ([Eskenazi et al., 2007](#);
19 [Warner et al., 2007](#); [Eskenazi et al., 2005](#); [Warner et al., 2004](#); [Eskenazi et al., 2002](#)), subtle
20 changes in immune system components ([Baccarelli et al., 2004](#); [Baccarelli et al., 2002](#)),
21 development dental defects ([Alaluusua et al., 2004](#)), and diabetes ([Michalek and Pavuk, 2008](#)).
22 Most of these effects occur at higher doses than the cocritical effects of semen quality and
23 neonatal TSH; also, for many of these studies, the quantification of an adverse response level is
24 too uncertain to define meaningful NOAELs or LOAELs. Diabetes may be a sensitive outcome
25 as a result of TCDD exposure in humans, but the relevant exposures could not be quantified for
26 the purposes of RfD development (see *Reanalysis Volume 1*, Appendix C for discussion).

27
28 Although human data are preferred, Table 4-5 in *Reanalysis Volume 1* (U.S. EPA, 201X)
29 presents candidate RfDs for a number of animal bioassays, many of which are lower than the
30 human RfD. Adverse noncancer effects reported in these studies include hepatic, neurological,
31 immunological, reproductive, endocrine, and developmental effects. PBPK modeling ([Emond et
32 al., 2005](#)) was used for interspecies toxicokinetic extrapolation of all rodent bioassays, assuming
33 that whole-blood TCDD concentration was the relevant dose metric; a 1st-order body burden
34 model was used for other species for which no PBPK model was available. Two of the rodent
35 bioassays among this group of studies—Bell et al. ([2007](#)) (candidate RfD = 1.4E-9 mg/kg-day
36 based on delay in the onset of puberty) and NTP ([2006](#)) (candidate RfD = 4.6E-10 mg/kg-day
37 based on liver and lung lesions)—are of particular note. Both studies were well designed and
38 conducted, using 30 or more animals per dose group. Bell et al. ([2007](#)) evaluated several
39 reproductive and developmental endpoints, initiating TCDD exposures well before mating and
40 continuing through gestation. NTP ([2006](#)) is the most comprehensive evaluation of TCDD
41 chronic toxicity in rodents to date, evaluating dozens of endpoints at several time points in all
42 major tissues.

43 44 I.A.5. CONFIDENCE IN THE CHRONIC ORAL RfD

45
46 Study – High
47 Data Base -- High
48 RfD -- High

1
2 The overall confidence in this RfD assessment is high. Confidence in the coprincipal studies is
3 high. The two principal studies were well conducted and show health effects in humans.
4 Exposure assessment is a limitation of Mocarelli et al. (2008) because of the atypical exposure
5 profile. However, the use of PBPK modeling to estimate internal exposures over the critical
6 window and to account for the potential influence of the initial high peak exposure is considered
7 to mitigate this limitation. The maternal exposures reported in Baccarelli et al. (2008) were not
8 subject to large fluctuations, since the maternal blood measurements occurred several years
9 following the accident and the newborns were exposed over a much narrower critical window
10 (i.e., during pregnancy); there is, however, some uncertainty in the extrapolation of serum
11 TCDD concentrations from the time of measurement to the time of pregnancy.

12
13 Confidence in the database is high. The TCDD database is extensive, covering a myriad of
14 endpoints and exposure durations in both sexes of many species of animals and in humans.
15 Analogous effects have been observed in animal bioassays and in human epidemiological
16 studies, increasing the overall confidence in the relevance to humans of the effects reported in
17 rodents and the association of TCDD exposure with the effects reported in humans.

18 19 **___I.A.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC ORAL RfD**

20
21 Source Document -- *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS*
22 *Comments, Volume 1* (U.S. EPA, 201X)

23
24 This document has provided for review to EPA scientists, interagency reviewers from
25 other federal agencies and White House offices, and the public, and peer reviewed by
26 independent scientists external to EPA. A summary and EPA's disposition of the comments
27 received from the independent external peer reviewers and from the public is included in
28 Appendix A of *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS*
29 *Comments, Volume 1* (U.S. EPA, 201X).

30 Agency Completion Date -- __/__/__

31 32 **___I.A.7. EPA CONTACTS**

33
34 Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in
35 general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email
36 address).

37 38 **___I.B. REFERENCE CONCENTRATION (RfC) FOR CHRONIC INHALATION** 39 **EXPOSURE**

40
41 Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

42 CASRN — 1746-01-6

43 Section I.B. Last Revised—

44
45 The RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a
46 continuous inhalation exposure to the human population (including sensitive subgroups) that is
47 likely to be without an appreciable risk of deleterious effects during a lifetime. The RfC
48 considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral

1 to the respiratory system (extrarespiratory effects). The inhalation RfC (generally expressed in
2 units of mg/m³) is analogous to the oral RfD and is similarly intended for use in risk assessments
3 for health effects known or assumed to be produced through a nonlinear (presumed threshold)
4 mode of action.

5
6 Inhalation RfCs are derived according to *Methods for Derivation of Inhalation Reference*
7 *Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994). Because RfCs can
8 also be derived for the noncarcinogenic health effects of substances that are carcinogens, it is
9 essential to refer to other sources of information concerning the carcinogenicity of this chemical
10 substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a
11 summary of that evaluation will be contained in Section II of this file.

12
13 There was no previous inhalation RfC for TCDD on the IRIS database.

14
15 **___ I.B.1. CHRONIC INHALATION RfC SUMMARY**

16
17 Not available. An RfC was not derived for TCDD.

18
19 **___ I.B.2. PRINCIPAL AND SUPPORTING STUDIES**

20
21 Not applicable

22
23 **___ I.B.3. UNCERTAINTY FACTORS**

24
25 Not applicable

26
27 **___ I.B.4. ADDITIONAL STUDIES/COMMENTS**

28
29 Not applicable

30
31 **___ I.B.5. CONFIDENCE IN THE CHRONIC INHALATION RfC**

32
33 Not applicable

34
35 **___ I.B.6. EPA DOCUMENTATION AND REVIEW OF THE CHRONIC INHALATION**
36 **RfC**

37
38 Source Document -- *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS*
39 *Comments, Volume 1* (U.S. EPA, 201X)

40
41 This document has been provided for review to EPA scientists, interagency reviewers from other
42 federal agencies and White House offices, and the public, and peer reviewed by independent
43 scientists external to EPA. A summary and EPA's disposition of the comments received from
44 the independent external peer reviewers and from the public is included in Appendix A of
45 *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments, Volume 1*
46 (U.S. EPA, 201X).

1 Agency Completion Date -- __/__/__

2
3 **__I.B.7. EPA CONTACTS**

4
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6 general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email
7 address).

8
9
10
11 **__II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE**

12
13 Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)

14 CASRN — 1746-01-6

15 Section I.A. Last Revised—

16
17 This section provides information on three aspects of the carcinogenic assessment for the
18 substance in question: the weight-of-evidence judgment of the likelihood that the substance is a
19 human carcinogen, and quantitative estimates of risk from oral and inhalation exposure. Users
20 are referred to Section I of this file for information on long-term toxic effects other than
21 carcinogenicity.

22
23 The rationale and methods used to develop the carcinogenicity information in IRIS are
24 described in the *Guidelines for Carcinogen Risk Assessment* (U.S. EPA, 2005a) and the
25 *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*
26 (U.S. EPA, 2005b). The quantitative risk estimates are derived from the application of a low-
27 dose extrapolation procedure, and are presented in two ways to better facilitate their use. First,
28 route-specific risk values are presented. The “oral slope factor” is a plausible upper bound on
29 the estimate of risk per mg/kg-day of oral exposure. Similarly, a “unit risk” is a plausible upper
30 bound on the estimate of risk per unit of concentration, either per µg/L drinking water (see
31 Section II.B.1.) or per µg/m³ air breathed (see Section II.C.1.). Second, the estimated
32 concentration of the chemical substance in drinking water or air when associated with cancer
33 risks of 1 in 10,000, 1 in 100,000, or 1 in 1,000,000 is also provided.

34
35 There was no previous cancer assessment for TCDD on the IRIS database.

36
37 **MESSAGE:** On August 29, 2011 EPA announced a plan to separate the *Reanalysis of Key*
38 *Issues Related to Dioxin Toxicity and Response to NAS Comments* into two volumes: Volume 1
39 (noncancer assessment) and Volume 2 (cancer assessment and uncertainty analysis). The
40 noncancer assessment and TCDD RfD are provided in this document. EPA will finalize Volume
41 2 as expeditiously as possible.

42
43 **__II.A. EVIDENCE FOR HUMAN CARCINOGENICITY**

44
45 Not applicable

46
47 **__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL**
48 **EXPOSURE**

1
2 Not applicable

3
4 **__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM**
5 **INHALATION EXPOSURE**

6
7 **__** Not applicable

8
9 **__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY**
10 **ASSESSMENT)**

11
12 **__II.D.1. EPA DOCUMENTATION**

13
14 Source Document -- *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS*
15 *Comments, Volume 1* (U.S. EPA, 201X)

16
17 This document has been provided for review to EPA scientists, interagency reviewers from other
18 federal agencies and White house offices, and the public, and peer reviewed by independent
19 scientists external to EPA. A summary and EPA’s disposition of the comments received from
20 the independent external peer reviewers and from the public is included in Appendix A of
21 *Reanalysis of Key Issues Related to Dioxin Toxicity and Response to NAS Comments, Volume 1*
22 (U.S. EPA, 201X).

23
24 **__II.D.2. EPA REVIEW**

25
26 Agency Completion Date -- __/__/__

27
28 **__II.D.3. EPA CONTACTS**

29
30 Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in
31 general, at (202) 566-1676 (phone), (202) 566-1749 (fax), or hotline.iris@epa.gov (email
32 address).

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35
36 **_III.** [reserved]
37 **_IV.** [reserved]
38 **_V.** [reserved]

39
40 _____

41
42 **_VI. BIBLIOGRAPHY**

43
44 Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
45 CASRN — 1746-01-6
46 Section VI. Last Revised—

47
48 **__VI.A. ORAL RfD REFERENCES**

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27
28 **__ VI.B. INHALATION RfC REFERENCES**

31
32 **__ VI.C. CARCINOGENICITY ASSESSMENT REFERENCES**

35
36 **__ VII. REVISION HISTORY**

37
38 Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
39 CASRN — 1746-01-6
40 File First On-Line ___/___/___

Date	Section	Description
___/___/___	I.A.	RfD added

46
47 **__ VIII. SYNONYMS**

1 Substance Name—2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD)
2 CASRN — 1746-01-6
3 Section VIII. Last Revised—
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