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Health Effects Support Document for Terbacil

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U.S. Environmental Protection Agency
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Health and Ecological Criteria Division
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FOREWORD

The Safe Drinking Water Act (SDWA), as amended in 1996, requires the Administrator of the Environmental Protection Agency (EPA) to establish a list of contaminants to aid the Agency in regulatory priority setting for the drinking water program. In addition, the SDWA requires EPA to make regulatory determinations for no fewer than five contaminants by August 2001 and every five years thereafter. The criteria used to determine whether or not to regulate a chemical on the Contaminant Candidate List (CCL) are the following:

- The contaminant may have an adverse effect on the health of persons.
- The contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern.
- In the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

The Agency's findings for all three criteria are used in making a determination to regulate a contaminant. The Agency may determine that there is no need for regulation when a contaminant fails to meet one of the criteria. The decision not to regulate is considered a final Agency action and is subject to judicial review.

This document provides the health effects basis for the regulatory determination for terbacil. In arriving at the regulatory determination, The Office of Water used the Re-registration Eligibility Document (RED) for terbacil published by the Office of Pesticides Programs (OPP)as well as any OPP health assessment documents that supported the RED. The following publications from OPP were used in development of this document.

U.S. EPA (United States Environmental Protection Agency). 1998a. Reregistration eligibility decision. Terbacil. EPA738-R-97-011. Washington, DC: U.S. EPA Office of Prevention, Pesticides and Toxic Substances. Available from: <http://www.epa.gov/oppsrrd1/REDs/0039red.pdf>.

Information from the OPP risk assessment was supplemented with information from the primary references for key studies where they have been published and recent studies of terbacil identified in a literature search conducted 2004 with a focused update in 2008.

A Reference Dose (RfD) is provided as the assessment of long-term toxic effects other than carcinogenicity. RfD determination assumes that thresholds exist for certain toxic effects, such as cellular necrosis, significant body or organ weight changes, blood disorders, etc. It is expressed in terms of milligrams per kilogram per day (mg/kg-day). In general, 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 carcinogenicity assessment for terbacil includes a formal hazard identification and an estimate of tumorigenic potency when available. Hazard identification is a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen via the oral route and of the conditions under which the carcinogenic effects may be expressed.

Development of these hazard identification and dose-response assessments for terbacil has followed the general guidelines for risk assessment as set forth by the National Research Council (1983). EPA guidelines that were used in the development of this assessment may include the following: *Guidelines for the Health Risk Assessment of Chemical Mixtures* (U.S. EPA, 1986a), *Guidelines for Mutagenicity Risk Assessment* (U.S. EPA, 1986b), *Guidelines for Developmental Toxicity Risk Assessment* (U.S. EPA, 1991), *Guidelines for Reproductive Toxicity Risk Assessment* (U.S. EPA, 1996), *Guidelines for Neurotoxicity Risk Assessment* (U.S. EPA, 1998b), *Guidelines for Carcinogen Assessment* (U.S. EPA, 2005a), *Recommendations for and Documentation of Biological Values for Use in Risk Assessment* (U.S. EPA, 1988), (proposed) *Interim Policy for Particle Size and Limit Concentration Issues in Inhalation Toxicity* (U.S. EPA, 1994a), *Methods for Derivation of Inhalation Reference Concentrations and Application of Inhalation Dosimetry* (U.S. EPA, 1994b), *Use of the Benchmark Dose Approach in Health Risk Assessment* (U.S. EPA, 1995), *Science Policy Council Handbook: Peer Review* (U.S. EPA, 1998c, 2000a), *Science Policy Council Handbook: Risk Characterization* (U.S. EPA, 2000b), *Benchmark Dose Technical Guidance Document* (U.S. EPA, 2000c), *Supplementary Guidance for Conducting Health Risk Assessment of Chemical Mixtures* (U.S. EPA, 2000d), and *A Review of the Reference Dose and Reference Concentration Processes* (U.S. EPA, 2002a).

The chapter on occurrence and exposure to terbacil through potable water was developed by the Office of Ground Water and Drinking Water. It is based primarily on first Unregulated Contaminant Monitoring Regulation 1 (UCMR1) data collected under the SDWA. The UCMR1 data are supplemented with ambient water data, as well as data from the States, and published papers on occurrence in drinking water.

ACKNOWLEDGMENT

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1.0 EXECUTIVE SUMMARY

The U.S. Environmental Protection Agency (EPA) has prepared this Health Effects Support Document for Terbacil to support a determination regarding whether to regulate terbacil with a National Primary Drinking Water Regulation (NPDWR). The available data on occurrence, exposure, and other risk considerations suggest that, because terbacil does not occur in public water systems at frequencies and levels of public health concern, regulating terbacil will not present a meaningful opportunity to reduce health risk. EPA will present a determination and further analysis in the Federal Register Notice covering the CCL proposals.

Terbacil (Chemical Abstracts Services Registry Number 759-94-4) is an herbicide used to selectively control many annual and some perennial weeds in crops, forestry, and feed crops. It is an odorless, colorless to white crystalline powder. Terbacil is released primarily into the environment through aircraft and ground equipment such as band treatment and broadcast sprays. Terbacil is listed as a Toxic Release Inventory (TRI) chemical, with on-site releases to surface water constituting the majority of releases.

Terbacil is applied to fields where crops are grown for weed control, and its residues can be expected to persist and dissipate in soil by photolysis. Terbacil has not been detected in groundwater, however, depending on the use of the herbicide and the amount of rain, this highly mobile herbicide may reach groundwater sources. Additionally, the available data for terbacil production, use, and environmental releases all show an increasing trend.

Data on the occurrence of terbacil in drinking water were obtained from the first Unregulated Contaminant Monitoring Regulation (UCMR1) program. Although UCMR 1 monitoring was conducted primarily between 2001 and 2003, some results were not collected and reported until as late as 2006. As a List 1 contaminant, terbacil was scheduled to be monitored by all large CWSs and NTNCWSs and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR1 analytical samples submitted and quality-checked under the regulation as of March 2006. Terbacil data were collected and submitted by 797 small public water systems and 3,076 large public water systems. Terbacil data were analyzed at the level of simple detections (at or above the minimum reporting level, ≥MRL, or ≥2 µg/L), exceedances of the health reference level (>HRL, or >90 µg/L), and exceedances of one-half the value of the HRL (>½HRL, or >45 µg/L). No detections of terbacil were found in any samples, and thus there were also no exceedances of the HRL or one-half the HRL.

There are no current studies that examine the human health effects due to terbacil exposure. Terbacil is acutely toxic to rodents and rabbits when exposure occurs orally, dermally, and by inhalation at high concentrations. According to oral subchronic and chronic studies, the liver appears to be the target organ in dogs and rats. Observed hepatotoxic effects include increased liver weights (absolute and/or relative); increased incidence of vacuolization and hypertrophy of hepatocytes; and increased incidence in centrilobular hepatocyte hypertrophy, biliary hyperplasia, and eosinophilic foci of cellular alteration in the liver.

Hepatotoxic effects also were included as critical effects observed in the principal study for determining the RfD, which is 0.013 mg/kg/day. This principal study is a chronic toxicity study, in which beagle dogs (4/sex/group) were administered terbacil via diet for 2 years at concentrations of 50, 250, or 2500/10,000 ppm (equivalent to 1.25, 6.25, 62.5/250 mg/kg/day, respectively). The no-observed-adverse-effect-level (NOAEL) was determined to be 1.25 mg/kg/day, and the lowest-observed-adverse-effect-level (LOAEL) was determined to be 6.25 mg/kg/day, based on increased thyroid to body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels. An uncertainty factor of 100 was used in calculating the RfD to ensure the protection of infants and children from exposure to terbacil. A factor of 10 was used to account for interspecies differences, while another factor of 10 was used to account for intraspecies differences.

Additionally, developmental and reproductive effects have been observed in rats and rabbits. Developmental effects included significantly decreased number of live fetuses per litter apparently due to fetal loss occurring before or near the time of implantation in rats (and decreased live fetal weights in rabbits. Additionally, decreased body weight gains in 250-ppm male offspring were observed when terbacil was administered orally over 3 generations at doses of 0, 50, or 250 ppm (equivalent to 0, 2.5, and 12.5 mg/kg/day, respectively). These results, however, do not suggest that offspring exhibited an increase in pre- or post-natal sensitivity to terbacil exposure because developmental NOAELs were the same as those for maternal toxicity. Additionally, the NOAEL for systemic (parental) toxicity was set at a lower concentration than the NOAEL for reproductive toxicity, indicating that the reproductive system is less sensitive than other organ systems to the effects of terbacil.

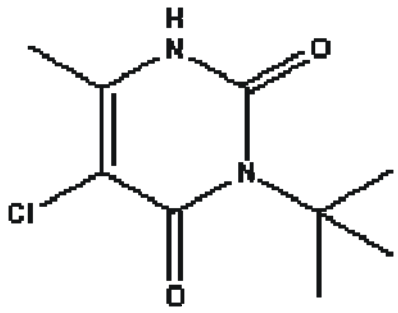
Two oral studies examined the carcinogenic effects of terbacil. Both studies conclude that oral administration of terbacil did not show evidence of increased tumor incidence in the treated animals when compared to the controls. Consequently, terbacil is classified as *not likely to be carcinogenic to humans* because animal evidence failed to demonstrate a carcinogenic effect in at least two well-designed and well-conducted studies in two appropriate animal species (U.S. EPA, 2005a). Additionally, terbacil is not mutagenic. Terbacil was tested and found negative in a chromosomal aberration study in rat bone marrow cells, found negative in a gene mutation assay (with and without S9 activation), and found negative for DNA synthesis when tested up to cytotoxic levels in rats.

When considered in its totality, the data on the occurrence of terbacil in public potable water systems indicate that a positive regulatory determination to regulate this compound in drinking water is not justified at this time. Although terbacil's physicochemical properties and increasing use causes some concern, it does not occur widely in drinking water systems. Terbacil does not occur in potable water systems at levels of concern, and regulation would not provide a meaningful opportunity to reduce risk for the population.

2.0 IDENTITY: CHEMICAL AND PHYSICAL PROPERTIES

Terbacil is an odorless, colorless to white crystalline powder, herbicide (HSDB, 2004). Terbacil is noncorrosive and stable to hydrolysis (Ahrens, 1994). It is stable at its melting point temperature and in aqueous alkaline media at room temperature (Tomlin, 1997).

Figure 2-1 Chemical Structure of Terbacil



Source: Chemfinder (2004)

The chemical structure of terbacil is shown above (Figure 2-1). Its physical and chemical properties, and other reference information are listed in Table 2-1.

The chemical name for terbacil is 3-tert-butyl-5-chloro-6-methyluracil or 5-chloro-3-(1, 1-dimethylethyl)-6-methyl-2,4(1H, 3H)-pyrimidinedione (U.S. EPA, 1998a). Terbacil also is referred to as Sinbar®, Geonter, Herbicide 732, Compound 732, Dupont 732, and experimental herbicide 732 (U.S. EPA, 1998a, 2000c). Ninety-five percent of technical grade terbacil is the pure active compound. Terbacil is available in wettable powder form consisting of 80% active ingredient; Krovar, a combination of terbacil and Hyvar X (Spencer, 1982); and in mixtures with diuron (Farm Chemicals Handbook 1993), MPCA, liuron, diuron, and linuron, or with linuron and monolinuron (Tomlin, 1997).

Table 2-1 Chemical and Physical Properties of Terbacil

Property	Information
Chemical Abstracts Registry (CAS) No.	5902-51-2
EPA Pesticide Chemical Code	OPP Code: 012701
Synonyms	Geonter, Dupont Herbicide 732
Registered Trade Name(s)	Sinbar®
Chemical Formula	$C_9H_{13}ClN_2O_2$
Molecular Weight	216.65
Physical State (25°C)	White crystals
Boiling Point	--
Melting Point	175-177°C
Density (25°C)	1.34 g/mL
Vapor Pressure (29.5°C)	4.8×10^{-7} mmHg
Partition Coefficients:	
Log K_{ow}	1.89
Log K_{oc}	9.00
Solubility in:	
Water (25°C)	710 mg/L
Other Solvents	dimethylformamide (337 g/kg at 25°C), cyclohexanone (220 g/kg at 25°C), methyl isobutyl ketone (121 g/kg at 25°C), butyl acetate (88 g/kg at 25°C), xylene (65 g/kg at 25°C) and is very soluble in strong aqueous alkalis; it is sparingly soluble in mineral oils aliphatic hydrocarbons

Source(s): U.S. EPA (1989a); Hansch et al. (1995); Wauchope et al. (1991); U.S. EPA (1998a); Tomlin (1997)

3.0 USES AND ENVIRONMENTAL FATE

This section summarizes information derived from cited secondary references pertaining to the production, use, environmental release, and environmental fate of terbacil.

3.1 Production and Use

Terbacil is a herbicide used to selectively control many annual and some perennial weeds (Tomlin, 1997) in crops (e.g., apples, mint, sugarcane, asparagus, blackberries, boysenberries, dewberries, loganberries, raspberries, youngberries, strawberries, and peaches), forestry (e.g., cottonwood), and feed crops (e.g., alfalfa, sainfoin, and forage) (U.S. EPA, 1998a). The chemical in the form of 80% (20% a.i.) wettable powder (WP; EPA Reg. No. 352-317) is manufactured from an unregistered Technical Grade Active Ingredient (TGAI) (95% a.i) by E.I. du Pont de Nemours and Company, Inc. (U.S. EPA, 1998a). The estimated use of terbacil during 1992 was recorded as 285,000 lbs (USGS, 1992). Terbacil can be applied by aircraft and ground equipment such as band treatment and broadcast sprays (U.S. EPA, 1998a). The maximum application rates range from 0.120 to 1.45 kg a.i./acre (U.S. EPA, 1998a). Lower rates generally are used on coarse textured soils and higher rates on fine textured soils (U.S. EPA, 1998a).

Annual usage data between 1987 and 1995 show that terbacil was used to treat approximately 401,000 acres of crop land and 4,000 acres of non-crop land (i.e., fallow, forest trees, and ornamentals). These uses accounted for an annual application of approximately 100,000 and 4,000 kg of terbacil a.i., respectfully (U.S. EPA, 1998a).

3.2 Environmental Release

Terbacil is listed as a Toxic Release Inventory (TRI) chemical. TRI data for terbacil (see Table 3-1) are reported for the years 1995 to 1997. During that three-year period, all reported releases were on-site releases to surface water. These releases were all in Texas (U.S. EPA, 2004a) and showed an increase in surface water discharge over the years.

Table 3-1 Environmental Releases (in pounds) of Terbacil in the United States, 1995-1997

Year	On-Site Releases				Off-Site Releases	Total On- & Off-site Releases
	Air Emissions	Surface Water Discharges	Underground Injection	Releases to Land		
1997	0	10,318	0	0	0	10,318
1996	0	3,835	0	0	0	3,835
1995	0	4,608	0	0	0	4,608

Source: U.S. EPA (2004a)

3.3 Environmental Fate

Terbacil dissipation in soil appears to depend on microbial-mediated degradation, photodegradation in water, and movement into ground and surface waters. Terbacil has a low sorption affinity to soil (K_{ad}=0.39 to 1.3 mL/g; K_{oc}=44 to 61 mL/g) and relatively high solubility

in water (710 mg/L); therefore, it is expected to be mobile in soil. The current data on terbacil and its degradation products indicate that it is persistent and potentially mobile in terrestrial environments (U.S. EPA, 1998a). Terbacil has a low vapor pressure (4.8×10^{-7} mmHg at 29.5°C) and Henry's Law constant (1.9×10^{-9} atm m^3/mole); therefore, is not expected to occur in large concentrations in the air.

Degradation

Terbacil is resistant to abiotic hydrolysis and slowly degrades through photolysis in water. Terbacil was stable for up to 6 weeks at pH of 5, 7, and 9 in the dark and in buffered solutions at 25°C at pH of 4-10 (U.S. EPA, 1998a). Photosensitizers such as riboflavin, rose bengal, and methylene blue enhance the process of photodegradation. Under natural sunlight, radiolabeled terbacil had a first-order photodegradation half-life ($t_{1/2}$) of 29 days in standard reference water (no further descriptions of water quality were presented), 37 days in river water (Brandywine River), 54 days in river water (Brandywine River) with suspended sediment, and 3.25 days in reference water with riboflavin (Rhodes, 1975). In the same laboratory, when exposed to ultraviolet (UV) rays, the radiolabeled terbacil displayed photodegradation half-lives of 44 days in standard reference water, 82.91 days in river water, and 4.8 days in reference water with a riboflavin sensitizer (U.S. EPA, 1998a). After 4 weeks of irradiation of terbacil at 5 ppm in distilled water with a pH of 6.2 with UV light of 300 to 400 nm, approximately16% was photodegraded (Davidson et al., 1978). Major photodegradation products were 5-chloro-6-methyluracil, 3-tert-butyl-6-methyluracil, and 6-chloro-2,3-dihydro-3,3,7-trimethyl-5H oxazolo (3,2-a)-pyrimidine-5-one.

In non-buffered aqueous solutions of pH 3.4 to 9.2, radiolabeled terbacil at 700 g/mL under natural sunlight with photosensitizers, a first-order photodegradation reaction was observed with the half-life being less than 2 hours (Acher, 1981). However, at 250 ppm, terbacil was photolytically stable in non-buffered aqueous solutions irradiated with a mercury vapor lamp at 25°C. The dyes, rose bengal and methylene blue, were effective photosensitizers in alkaline non-buffered aqueous solutions with a pH above 6.6. Humic acid was not an effective sensitizer in aqueous solutions. Major photodegradation products were 3-tert-butyl-5-hydroxyhydantoin (Compound II), 3-tert-butyl-5-hydroxyhydantoin (Compound III), and 5-chloro-6-methyl-(3',5')-5'-chloro-6'-methyl-5',6'-dihydro-6',2-anhydro-3'-tert-butyluracil (Compound VI), and an unidentified product (Compound V). Compound II does not appear to be a photodegradate because it was detected in dark controls. In the dark, radiolabeled terbacil was stable in buffer solutions of pH between 4 and 10 for greater than 6 weeks (U.S. EPA, 1998a).

Degradation of terbacil in aerobic and anaerobic soil is slow. Terbacil degradation was dependant on first-order degradation kinetics (U.S. EPA, 1998a). In soil, terbacil is stable to abiotic hydrolysis and if it is not in contact with direct sunlight; photolysis is the only degradation pathway with a half-life of 122 days (U.S. EPA, 1998a). Irradiated and dark treatment of terbacil yielded the degradation product 5-chloro-6-methyluracil.

Terbacil in silt loam continuously irradiated with fluorescent sun lamps and black lights for an 8 week period, displayed a first-order degradation half-life of 46 days (Rhodes, 1975). The photodegradation rate appears to be dramatically enhanced by the presence of the photosensitizers, riboflavin and methylene blue. The major transformation product was CO_2 and minor transformation products were t-butylurea and 3-tert-butyl-6-methyluracil (U.S. EPA, 1998a). In nonsterile soils incubated in a greenhouse, terbacil was found to have a half-life of 2 to 3 months (Rhodes, 1975). Furthermore, 90 percent of 2 ppm terbacil remained after a 90-day incubation period in sterile and nonsterile soil, and 0.8 to 1.5 percent of the applied ^{14}C was later found in CO_2 molecules from nonsterile soil, while 0.01 percent was in the CO_2 from sterile soil (U.S. EPA, 1982). Only trace amounts of radiolabeled terbacil applied at 2.88 ppm were degraded to radiolabeled carbon dioxide after 145 days when metabolized by microbes in a dark anaerobic environment (Rhodes, 1975).

Radiolabeled terbacil at 9.3 µg/g in sandy loam soil was found to have a first-order half-life of 653 days after 12 months in the dark (U.S. EPA, 1998a). Another study found that terbacil at 100 g had a half-life of 720 days in sandy loam soils (Marsh and Davis, 1978). When 2.1 ppm terbacil is applied to anaerobic silt loam and sandy soil, after 60 days at 20°C less than 5 percent of the chemical degraded (U.S. EPA, 1982). A study performed in 1970 illustrated that 8 ppm of terbacil had a half-life of approximately 5 months in aerobic loam soil (Zimdahl et al., 1970). Eight ppm of terbacil had a 5-month half-life in aerobic loam soil at 32°C, and 2 ppm of terbacil had a 2 to 3 month half-life in aerobic silt loam and sandy loam soils (U.S. EPA, 1982). In terms of biodegradation, 20% of 100 ppm terbacil biodegraded after 32 weeks in aerobic sandy loam soil at 23°C (U.S. EPA, 1982). Terbacil, at 4.5 lbs. a.i./A, had a half-life of 32.5 days when incubated in silt loam soil irradiated with UV light for 12 hr/d for 6.5 weeks (Rhodes et al., 1969). When 2.88 ppm radiolabeled terbacil was applied on sandy loam soil 28% degraded to $^{14}CO_2$ (Wolf, 1973; Wolf, 1974; Wolf and Martin, 1974).

In the atmosphere, photochemically-produced hydroxyl radicals degrade vapor-phase terbacil with a half-life for this degradation of 51 hrs. Wet and dry deposition removes particulate-phase terbacil from the atmosphere and redistributes it into terrestrial or aquatic systems (HSDB, 2004).

Environmental Media Transport and Distribution

A model of gas/particle partitioning of semivolatile organic compounds in the atmosphere (Bidleman, 1988) portrays that terbacil may exist in the vapor and particulate phases in the ambient atmosphere due to its 4.7×10^{-7} mm Hg vapor pressure. Volatilization is not expected to be a major route of dissipation for terbacil because of its low vapor pressure and low Henry's Law constant of 1.9×10^{-9} atm m^3/mole (U.S. EPA, 1998a).

Terbacil is usually applied by aircraft and orchard airblasts, which is a potential risk to nontarget aquatic organisms. The Spray Drift Task Force (SDTF) completed and submitted a series of studies intended to characterize spray droplet drift potential to the EPA's Office of Pesticide Prevention Agency, which had not yet been evaluated by time the RED document was published in 1998. Previous data indicate, however, that off-target drift rates are 1% of the

applied spray volume from ground applications and 5% from aerial and orchard airblast applications at 100 feet downwind (U.S. EPA, 1998a).

With regard to terbacil in soil, studies show that terbacil does not adsorb to suspended solids and sediment and has a high/very high mobility due to its K_{oc} values that range from 41 to 85 (U.S. EPA, 1998a; Kenaga, 1980; Rao and Davidson, 1982). Terbacil was negligibly adsorbed to soils ranging in texture from sand to clay (Davidson et al., 1978; Liu et al., 1971; Rao and Davidson, 1979). Studies indicated that 54% of terbacil was adsorbed to muck soil, which is 36% organic matter (Liu et al., 1977).

Field dissipation studies indicate that terbacil, at 5 lbs a.i./A, is persistent and mobile under actual use conditions. Field dissipation half-lives in Delaware, Illinois, and California ranged from 204 to 252 days (U.S. EPA, 1998a). These field dissipation studies showed that terbacil's persistence in the soil is dependant on application rate, soil type, and rainfall. In the field, terbacil residues persisted in soil for up to 16 months following a single application. Residues were found at the maximum depths sampled, i.e., 3 to 43 inches (Gardiner, No date a,b; Gardiner et al., 1969; Isom et al., 1969; Isom et al., 1970; Liu et al., 1977; Mansell et al., 1977; Mansell et al., 1979; Morrow and McCarty, 1976; Rahman, 1977; Rhodes, 1975). Multiple applications of terbacil demonstrated persistence for 1 to more than 2 years following the final application of the herbicide (Skroch et al., 1971; Tucker and Phillips, 1970; Benson, 1973; Doughty, 1978).

Laboratory soil mobility studies demonstrated that terbacil and its minor transformation products can leach through a 30 cm column of silt loam and sand loam soil when eluted with 20 inches of 0.01 M $CaCl_2$ (U.S. EPA, 1998a). Terbacil was predominately detected in the elution samples. In another study, 4 to 64% of radioactive terbacil (2 lbs/A), as well as an unidentified radioactive product was leached through 18 inches of packed soil columns of sandy loam and silt loam when eluted with 20 inches of water (Rhodes, 1975). Terbacil residues were eluted with 10 to 20 cm of water to a depth of 27.5 to 30 cm in packed 30 cm soil columns of sandy orchard soil (Marriage et al., 1977). A fourth study utilized fine sand (30 cm column) and approximately 73 to 90% of applied radioactivity was leached when eluted with 15.5 to 20 inches of 0.01 M $CaSO_4$.

Terbacil applied as spray at 5 lbs a.i./A had a first order half-life of 212 days on silt loam, 204 days on silty clay soil, and 252 days on a sandy loam soil (U.S. EPA, 1998a). Terbacil residues of less than 0.09 µg of terbacil/g of soil were detected at a maximum soil depth of 45 to 50 cm (U.S. EPA, 1998a). In some field studies, the transformation product, 3-t-butyl-5-chloro-6-hydroxymethyluracil, had a maximum concentration of 0.14 µg of terbacil/g of soil at 15 days. The transformation product, 6-chloro-2,3-dihydro-7-(hydroxymethyl)3,3-dimethyl-5H-oxazolo[3,2-a]pyrimidin-5-one, had a maximum concentration of 0.07 µg of terbacil/g of soil at 60 days post-treatment (U.S. EPA, 1998a).

In a microplot field dissipation study, radiolabeled terbacil at 2 lbs a.i./A had a half-life of 1 to 2 months when incubated in the field for 4 months with a cumulative rainfall of 18.33

inches (Rhodes, 1975). Terbacil was detected in the 12 to 15 inch soil segment. Terbacil applied at 4 lbs a.i./A had an estimated half-life of 131 days when incubated for 52 weeks in silt loam (Gardiner et al., 1969). Residues were detected through the lysimeter at 5 weeks post-treatment.

Terbacil applied at 1.6 lbs a.i./A on orchard soil was detected 1 year post-treatment using oats, beans, and cucumbers as phytotoxicity indicators at a maximum depth of 18 to 24 inches (Benson, 1973). Terbacil applied at 2.24 kg/ha/year for 4 consecutive years caused phytotoxic effects on oats and beans 2 year post-treatment on acidic clay loam soil (Doughty, 1978). A phytotoxic amount of terbacil residues were not detected on a high organic matter, acidic, silt loam on which 4.48 kg/ha of terbacil was applied for 4 consecutive years (U.S. EPA, 1998a). Other studies indicate that phytotoxic levels of terbacil residues were detected 7 to 13 months post-treatment in clay loam soil (Liu et al., 1977; Isom et al., 1969; Isom et al., 1970). Terbacil applied on the soil surface or incorporated at rates of 2.24 and 4.48 kg/ha for 3 consecutive years degraded with a half-life of approximately 157 days on peach orchard sandy soil (Skroch et al., 1971). Terbacil was detected at maximum soil depths of 15 to 30 cm soil following the second and third year applications (U.S. EPA, 1998a).

Terbacil is persistent and mobile in soils, which are conditions favorable for it to dissipate into groundwater (U.S. EPA, 1989a). Based on the environmental fate data, terbacil exceeds the mobility and persistence triggers for the proposed Restricted Use Classification for ground water concerns. The Groundwater Ubiquity Score (GUS) for terbacil is 5.32 in the best case scenario (scores above 2.8 indicate relatively high leaching potential) (U.S. EPA, 1998a). Currently, there are insufficient terbacil detections in ground water to warrant a Restricted Use classification. The lack of detections of terbacil in ground water may be associated with its limited geographical use. Terbacil has relatively low total environmental loading relative to other herbicides. Testing for terbacil has been conducted in areas where the herbicide was not used. In addition to this, the limit of detection for terbacil is 5 to 100 times higher than those of other herbicides (U.S. EPA, 1998a).

PATRIOT (Pesticide Assessment Tool for Rating Investigations of Transport) modeling, can do a comparative leaching assessment of terbacil, relative to a conservative tracer, and can predict the amount of potential leaching in soils. The modeling predicted that approximately 40 to 75% of the terbacil mass applied could leach to shallow ground water (4.5 feet) (U.S. EPA, 1998a). Annual leaching may be highly variable depending on the rainfall. Since terbacil is persistent in soil, it will most likely accumulate in soil and the total mass reaching ground water in a particular year may exceed the total mass applied in a given year. The mass of terbacil estimated to leach to ground water for each year ranged from 0 to 125% of the annual application (U.S. EPA, 1998a). Terbacil is only used on minor crops; therefore, the impact on groundwater is expected to be limited to very localized, site specific soil/hydrological conditions. According to the PATRIOT model, based on the use pattern and site conditions, mint and sugarcane production areas are predicted to be vulnerable groundwater areas for terbacil. Terbacil could reach the shallow groundwater at high concentrations (U.S. EPA, 1998a).

A groundwater screening model, Screening Concentrations in Ground Water (SCI-GRO) predicted that terbacil could potentially contaminate shallow groundwater near specific use sites at low levels. This is due to its use on limited areas of minor crops with relatively low environmental loading (U.S. EPA, 1998a).

Tier 1 GENEEC (Generic Expected Environmental Concentration) modeling indicates that terbacil may reach surface waters at concentrations between 19 and 154 g/L. Another surface water simulation device, PRZM-EXAMS EECs, suggests that terbacil may accumulate in static surface waters from long-term use, and that surface water runoff may be an important route of dissipation, while the STORET database suggests that terbacil does not accumulate in surface waters (U.S. EPA, 1998a). More research is required in this area.

Bioaccumulation

The BCF (the bioconcentration factor) of terbacil is estimated to be 16, which indicates that bioconcentration of terbacil in aquatic organisms is unlikely (HSDB, 2004). Its log K_{ow} (Hansch, 1995) and regression-derived equation (Meylan, 1999) also point to a low bioconcentration of terbacil in aquatic organisms. In a study with a 4-week exposure period, 0.01 and 1.00 µg/mL of radiolabeled terbacil was accumulated, respectively, at concentrations of 0.11 and 7.9 µg of terbacil/g of tissue in viscera, 0.02 and 1.8 µg of terbacil/g of tissue in head, 0.07 and 4.4 µg of terbacil/g of tissue in the livers, and 0.02 and 1.7 µg of terbacil/g of tissue in edible tissue of bluegill sunfish (U.S. EPA, 1998a). After 3 days of depuration, radioactive terbacil residues were below the detection limit of 0.01 µg of terbacil/g of tissue in all fish tissues (U.S. EPA, 1998a). A metabolism study, which used 65x feeding level of terbacil, indicated that there is no likelihood of finite residues in poultry (U.S. EPA, 1998a). Terbacil residues in ruminant commodities showed no likelihood of finite residues (U.S. EPA, 1998a).

3.4 Summary

In summary, terbacil is applied to fields where crops are grown for weed control, and its residues can be expected to persist and dissipate in soil by photolysis. Terbacil has not been detected in groundwater, however, depending on the use of the herbicide and the amount of rain, this highly mobile herbicide may reach groundwater sources. Finally, terbacil is applied to fields but is not expected to remain in the atmosphere.

4.0 EXPOSURE FROM DRINKING WATER

4.1 Introduction

EPA used data from several sources to evaluate the potential for occurrence of terbacil in Public Water Systems (PWSs). The primary source of drinking water occurrence data for terbacil was the first Unregulated Contaminant Monitoring Regulation (UCMR1) program. The Agency also evaluated ambient water quality data from the United States Geological Survey (USGS).

4.2 Ambient Occurrence

4.2.1 Data Sources and Methods

USGS instituted the National Water Quality Assessment (NAWQA) program in 1991 to examine ambient water quality status and trends in the United States. NAWQA is designed to apply nationally consistent methods to provide a consistent basis for comparisons among study basins across the country and over time. These occurrence assessments serve to facilitate interpretation of natural and anthropogenic factors affecting national water quality. For more detailed information on the NAWQA program design and implementation, please refer to Leahy and Thompson (1994) and Hamilton and colleagues (2004).

Study Unit Monitoring

The NAWQA program conducts monitoring and water quality assessments in significant watersheds and aquifers referred to as "study units." NAWQA's sampling approach is not "statistically" designed (i.e., it does not involve random sampling), but it provides a representative view of the nation's waters in its coverage and scope. Together, the 51 study units monitored between 1991 and 2001 include the aquifers and watersheds that supply more than 60% of the nation's drinking water and water used for agriculture and industry (NRC, 2002). NAWQA monitors the occurrence of chemicals such as pesticides, nutrients, volatile organic compounds (VOCs), trace elements, and radionuclides, and the condition of aquatic habitats and fish, insects, and algal communities (Hamilton et al., 2004).

Monitoring of study units occurs in stages. Between 1991 and 2001, approximately one-third of the study units at a time were studied intensively for a period of three to five years, alternating with a period of less intensive research and monitoring that lasted between five and seven years. Thus, all participating study units rotated through intensive assessment in a ten-year cycle (Leahy and Thompson, 1994). The first ten-year cycle was called "Cycle 1." Summary reports are available for the 51 study units that underwent intensive monitoring in Cycle 1 (USGS, 2001). Cycle 2 monitoring is scheduled to proceed in 42 study units from 2002 to 2012 (Hamilton et al., 2004).

Pesticide National Synthesis

Through a series of National Synthesis efforts, the USGS NAWQA program is preparing comprehensive analyses of data on topics of particular concern. These data are aggregated from the individual study units and other sources to provide a national overview.

The Pesticide National Synthesis began in 1991. Results from the most recent USGS Pesticide National Synthesis analysis, based on complete Cycle 1 (1991-2001) data from NAWQA study units, are posted on the NAWQA Pesticide National Synthesis website (Martin et al., 2003; Kolpin and Martin, 2003; Nowell, 2003; Nowell and Capel, 2003). USGS considers these results to be provisional. Data for surface water, ground water, bed sediment, and biota are presented separately, and results in each category are subdivided by land use category. Land use categories include agricultural, urban, mixed (deeper aquifers of regional extent in the case of ground water), and undeveloped. The National Synthesis analysis for pesticides is a first step toward the USGS goal of describing the occurrence of pesticides in relation to different land use and land management patterns, and developing a deeper understanding of the relationship between spatial occurrence of contaminants and their fate, transport, persistence, and mobility characteristics.

The surface water summary data presented by USGS in the Pesticide National Synthesis (Martin et al., 2003) only include stream data. Sampling data from a single one-year period, generally the year with the most complete data, were used to represent each stream site. Sites with few data or significant gaps were excluded from the analysis. NAWQA stream sites were sampled repeatedly throughout the year to capture and characterize seasonal and hydrologic variability. In the National Synthesis analysis, the data were time-weighted to provide an estimate of the annual frequency of detection and occurrence at a given concentration.

The USGS Pesticide National Synthesis only analyzed ground water data from wells; data from springs and agricultural tile drains were not included. The sampling regimen used for wells was different than that for surface water. In the National Synthesis analysis (Kolpin and Martin, 2003), USGS uses a single sample to represent each well, generally the earliest sample with complete data for the full suite of analytes.

NAWQA monitored bed sediment and fish tissue at sites considered likely to be contaminated and sites that represent various land uses within each Study Unit. Most sites were sampled once in each medium. In the case of sites sampled more than once, a single sample was chosen to represent the site in the Pesticide National Synthesis analysis (Nowell, 2003). In the case of multiple bed sediment samples, the earliest one with complete data for key analytes was used to represent the site. In the case of multiple tissue samples, the earliest sample from the first year of sampling that came from the most commonly sampled type of fish in the Study Unit was selected.

As part of the National Pesticide Synthesis, USGS also analyzed the occurrence of select semivolatile organic compounds (SVOCs) in bed sediment at sites considered likely to be contaminated and sites that represent various land uses within each Study Unit (Nowell and

Capel, 2003). Most sites were sampled only once. When multiple samples were taken, the earliest one was used to represent the site in the analysis.

Over the course of Cycle 1 (1991-2001), NAWQA analytical methods may have been improved or changed. Hence, reporting levels (RLs) varied over time for some compounds. In the summary tables, the highest RL for each analyte is presented for general perspective. In the ground water, bed sediment, and tissue data analyses, the method of calculating concentration percentiles sometimes varied depending on how much of the data was censored at particular levels by the laboratory (i.e., because of the relatively large number of non-detections in these media).

4.2.2 Results

Under the NAWQA program, USGS monitored terbacil between 1992 and 2001 in representative watersheds and aquifers across the country. Reporting limits varied but did not exceed 0.034 µg/L. All concentrations determined for terbacil are estimated concentrations. Results for surface water and ground water are presented in Tables 4-1 and 4-2. Terbacil was not monitored in bed sediment or biota.

Table 4-1 USGS National Synthesis Summary of NAWQA Monitoring of Terbacil in Ambient Surface Water, 1992-2001

Land Use Type	No. of Samples (and No. of Sites)	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,858 (77)	4.52%	<RL	<RL	0.540 µg/L
Mixed	996 (46)	1.82%	<RL	<RL	0.341 µg/L
Undeveloped	60 (4)	1.40%	<RL	<RL	0.092 µg/L
Urban	896 (33)	1.98%	<RL	<RL	0.035 µg/L

Source: Martin et al. (2003)
RL = Reporting limit. Reporting limits for terbacil varied, but did not exceed 0.034 µg/L.
All terbacil concentrations are estimated concentrations.
The USGS Pesticide National Synthesis used one year of data, generally the year with the most sampling results, to represent each site in this analysis. The sampling results were time-weighted, to eliminate bias from more frequent sampling at certain times of year. Detection Frequencies and Percentile Concentrations can be interpreted as representing annual occurrence. For instance, the detection frequency can be thought of as the percent of the year in which detections are found at a typical site in this land use category, and the 95th percentile concentration can be thought of as a concentration that is not exceeded for 95% of the year at a typical site in this land use category.

In surface water, terbacil was detected at frequencies ranging from 1.40% of samples in undeveloped settings to 1.82% in mixed land use settings, 1.98% in urban settings, and 4.52% in agricultural settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.540 µg/L, was found in an agricultural setting (Martin et al., 2003).

Table 4-2 USGS National Synthesis Summary of NAWQA Monitoring of Terbacil in Ambient Ground Water, 1992-2001

Land Use Type	No. of Wells	Detection Frequency	50th Percentile (Median) Concentration	95th Percentile Concentration	Maximum Concentration
Agricultural	1,438	0.76%	<RL	<RL	0.495 µg/L
Mixed (Major Aquifer)	2,708	0.26%	<RL	<RL	0.891 µg/L
Undeveloped	67	0.0%	<RL	<RL	<RL
Urban	830	1.20%	<RL	<RL	0.093 µg/L

Source: Kolpin and Martin (2003)
RL = Reporting limit. Reporting limits for terbacil varied, but did not exceed 0.034 µg/L.
All terbacil concentrations are estimated concentrations.
The USGS Pesticide National Synthesis considered each well a distinct site in this analysis. Each well was represented by one sample: normally the first one taken, but possibly a later sample if the first sample was not analyzed for the full range of analytes.
Percentile Concentrations were drawn from the range of detects and non-detects. The method for calculating Percentile Concentrations varied depending on how much of the data was censored at particular levels by the laboratory.

In ground water, terbacil detection frequencies ranged from 0.0% in undeveloped settings to 0.26% in mixed land use (major aquifer) settings, 0.76% in agricultural settings, and 1.20% in urban land use settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.891 µg/L, was in a mixed land use (major aquifer) setting (Kolpin and Martin, 2003).

4.3 Drinking Water Occurrence

4.3.1 Data Sources, Data Quality, and Analytical Methods

In 1999, EPA developed the UCMR1 program in coordination with the CCL and the National Drinking Water Contaminant Occurrence Database (NCOD) to provide national occurrence information on unregulated contaminants. EPA designed the UCMR1 data collection with three parts (or tiers), primarily based on the availability of analytical methods. Terbacil belonged to the first tier, List 1.

List 1 Assessment Monitoring was performed for a specified number of chemical contaminants for which analytical methods have been developed. With the exception of transient non-community systems and systems that purchase 100% of their water, EPA required all large PWSs (systems serving more than 10,000 people), plus a statistically representative national sample of 800 small PWSs (systems serving 10,000 people or fewer) to conduct Assessment Monitoring. Approximately one-third of the participating small systems were scheduled to monitor for these contaminants during each calendar year from 2001 through 2003. Large systems could conduct one year of monitoring anytime during the 2001-2003 UCMR1 period. EPA specified a quarterly monitoring schedule for surface water systems and a twice-a-

year, six-month interval monitoring schedule for ground water systems. Although UCMR1 monitoring was conducted primarily between 2001 and 2003, some results were not collected and reported until as late as 2006.

The objective of the UCMR1 sampling approach for small systems was to collect contaminant occurrence data from a statistically selected, nationally representative sample of small systems. The small system sample was stratified and population-weighted, and included some other sampling adjustments such as allocating a selection of at least two systems from each State. With contaminant monitoring data from all large PWSs and a statistical, nationally representative sample of small PWSs, the UCMR1 List 1 Assessment Monitoring program provides a contaminant occurrence data set suitable for national drinking water estimates.

4.3.2 CCL Health Reference Level

To evaluate the systems and populations exposed to terbacil through PWSs, the monitoring data were analyzed against the Minimum Reporting Level (MRL) and a benchmark value for health that is termed the Health Reference Level (HRL). Two different approaches were used to derive the HRL, one for chemicals that cause cancer and exhibit a linear response to dose and the other applies to noncarcinogens and carcinogens evaluated using a non-linear approach.

The RfD for terbacil is 0.013 mg/kg/day based on a chronic toxicity feeding study with beagle dogs where critical effects included increased relative thyroid weights, increased liver weights, and elevated liver enzymes (Wazeter et al., 1967a). Additional detail concerning the RfD can be found in section 6.2. The Agency established the HRL for terbacil using the RfD and a 20 percent relative source contribution as follows:

HRL = [(0.013 mg/kg/day x 70 kg)/2 L/day] x 20% = 0.091 mg/L (or 90 µg/L using the round number)

4.3.3 Results

As a List 1 contaminant, terbacil was scheduled to be monitored by all large CWSs and NTNCWSs and a statistically representative sample of small CWSs and NTNCWSs. The data presented in this report reflect UCMR1 analytical samples submitted and quality-checked under the regulation as of March 2006. Terbacil data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,076 (99.2 percent) of the 3,100 large systems defined as eligible for the UCMR1 large system census. Terbacil data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥MRL, or ≥2 µg/L), exceedances of the health reference level (>HRL, or >90 µg/L), and exceedances of one-half the value of the HRL (>½HRL, or >45 µg/L).

Results of the analysis are presented in Tables 4-3 and 4-4. No detections of terbacil were found in any samples and, thus, there also were no exceedances of the HRL or one-half the HRL.

Table 4-3 Summary UCMR1 Occurrence Statistics for Terbacil in Small Systems (Based on Statistically Representative National Sample of Small Systems)

Frequency Factors	UCMR Data - Small Systems		National System & Population Numbers[1]
Total Number of Samples	3,251		--
Percent of Samples with Detections	0.00%		--
99th Percentile Concentration (all samples)	< MRL		--
Health Reference Level (HRL)	90 µg/L		--
Minimum Reporting Level (MRL)	2 µg/L		--
Maximum Concentration of Detections	< MRL		--
99th Percentile Concentration of Detections	< MRL		--
Median Concentration of Detections	< MRL		--
Total Number of PWSs	797		60,414
Number of GW PWSs	590		56,072
Number of SW PWSs	207		4,342
Total Population	2,760,570		45,414,590
Population of GW PWSs	1,939,815		36,224,336
Population of SW PWSs	820,755		9,190,254
Occurrence by System	**Number**	**Percentage**	**National Extrapolation[2]**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%	0
PWSs (GW & SW) > 1/2 HRL	0	0.00%	0
PWSs (GW & SW) > HRL	0	0.00%	0
Occurrence by Population Served			
Population Served by PWSs with Detections	0	0.00%	0
Population Served by PWSs > 1/2 HRL	0	0.00%	0
Population Served by PWSs > HRL	0	0.00%	0

1. Total PWS and population numbers are from EPA September 2004 Drinking Water Baseline Handbook, 4th edition.
2. National extrapolations are generated by multiplying the system/population percentages and the national Baseline Handbook system/population numbers.

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½HRL, or PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½HRL benchmark, or exceeding the HRL benchmark, respectively; Population Served by PWSs with detections, by PWSs >½HRL, or by PWSs >HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½HRL benchmark, or exceeding the HRL benchmark, respectively
Notes:
-Small systems are those that serve 10,000 persons or fewer.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.
-Due to differences between the ratio of GW and SW systems with monitoring results and the national ratio, extrapolated GW and SW figures might not add up to extrapolated totals.

Table 4-4 Summary UCMR1 Occurrence Statistics for Terbacil in Large Systems (Based on the Census of Large Systems)

Frequency Factors	UCMR Data - Large Systems	
Total Number of Samples	30,549	
Percent of Samples with Detections	0.00%	
99th Percentile Concentration (all samples)	< MRL	
Health Reference Level (HRL)	90 µg/L	
Minimum Reporting Level (MRL)	2 µg/L	
Maximum Concentration of Detections	< MRL	
99th Percentile Concentration of Detections	< MRL	
Median Concentration of Detections	< MRL	
Total Number of PWSs Number of GW PWSs Number of SW PWSs	3,076 1,380 1,696	
Total Population Population of GW PWSs Population of SW PWSs	223,491,907 53,405,539 170,086,368	
Occurrence by System	**Number**	**Percentage**
PWSs (GW & SW) with Detections (≥ MRL)	0	0.00%
PWSs (GW & SW) > 1/2 HRL	0	0.00%
PWSs (GW & SW) > HRL	0	0.00%
Occurrence by Population Served		
Population Served by PWSs with Detections	0	0.00%
Population Served by PWSs > 1/2 HRL	0	0.00%
Population Served by PWSs > HRL	0	0.00%

Abbreviations:
PWS = Public Water Systems; GW = Ground Water; SW = Surface Water; N/A = Not Applicable; Total Number of Samples = the total number of samples on record for the contaminant; 99th Percentile Concentration = the concentration in the 99th percentile sample (out of either all samples or just samples with detections); Median Concentration of Detections = the concentration in the median sample (out of samples with detections); Total Number of PWSs = the total number of PWSs for which sampling results are available; Total Population Served = the total population served by PWSs for which sampling results are available; PWSs with detections, PWSs > ½HRL, and PWSs > HRL = PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½HRL benchmark, or exceeding the HRL benchmark; Population Served by PWSs with detections, by PWSs >½HRL, and by PWSs >HRL = population served by PWSs with at least one sampling result greater than or equal to the MRL, exceeding the ½HRL benchmark, or exceeding the HRL benchmark.

Notes:
-Large systems are those that serve more than 10,000 persons.
-Only results at or above the MRL were reported as detections. Concentrations below the MRL are considered non-detects.

4.4 Summary

Under the NAWQA program, USGS monitored terbacil between 1992 and 2001 in representative watersheds and aquifers across the country. The 95th percentile concentrations in surface and ground water were less than the reporting limit in all land use settings. Terbacil was detected more frequently in ambient surface water than in ambient ground water in all land use settings (1.40% vs. 0% of samples from undeveloped areas; 1.82% vs. 0.26% of samples from mixed land use settings; 1.98% vs. 1.20% of urban samples; and 4.52% vs. 0.76% of agriculture samples).

For UCMR1, terbacil was scheduled to be monitored by all large CWSs and NTNCWSs and a statistically representative sample of small CWSs and NTNCWSs. The data that were available in March of 2006 were analyzed at the level of simple detections (at or above the minimum reporting level, ≥MRL, or 9 µg/L), exceedances of the health reference level (>HRL, or >90 µg/L), and exceedances of one-half the value of the HRL (>½HRL, or >45 µg/L). No detections of terbacil were found in any samples and, thus, there were also no exceedances of the HRL or one-half the HRL.

5.0 EXPOSURE FROM MEDIA OTHER THAN WATER

This section summarizes human population exposures to terbacil from food, air, and soil. The primary purpose is to estimate average daily intakes of terbacil by members of the general public. When exposure data on sub-populations were located, such as occupationally exposed persons, these data were summarized and included in this section.

5.1 Exposure from Food

Terbacil is used to control broad leaf non-essential plants in food and feed crop areas such as apples, mint, sugarcane, asparagus, berries, peach, alfalfa, and sainfoin. Terbacil is believed to be persistent in the environment; therefore, the general population may be exposed to terbacil through diet.

5.1.1 Concentration in Non-Fish Food Items

The U.S. EPA (1998a) evaluated the estimated environmental concentrations (EECs) of terbacil on avian or mammalian food items immediately following a direct single application at 1 lb a.i./A in nongranular form (e.g., liquid or dust). The table below presents the predicted maximum and mean residues on these products.

Table 5-1 Estimated Environmental Concentrations on Avian and Mammalian Food Items

Food Items	EEC (ppm) Predicted Maximum Residue*	EEC (ppm) Predicted Mean Residue*
Short grass	240	85
Tall grass	110	36
Broadleaf/forage plants, and small insects	135	45
Fruits, pods, seeds, and large insects	15	7

* U.S. EPA 1998a

In 1977, Cessna measured terbacil residue in highbush and lowbush blueberries that were treated with only terbacil as well as those that were treated with a combination of herbicides. The maximum residue measured was 2.0 ppb where the limit of detection was 1.0 ppb based on a 25 g sample and recoveries were in the order of 90%.

Cessna (1991) conducted a 2-year study on terbacil residues extracted from asparagus spears from established agricultural sites in British Columbia and Ontario following pre-emergence and early post-emergence applications at 0.6, 1.1, and 2.2 kg/ha. At one site, maximum residues in the pre-emergence samples were found to be 14±3 µg/kg for the 2.2 kg/ha application rate, whereas maximum residues in the early post-emergence samples for the 2.2 kg/ha application rate were 493±250 µg/kg at a second site. Recoveries of terbacil from fortified

asparagus tissue were 96±19%, 87.2±11.9% and 83.3±7.8% at 10, 50, and 100 µg/kg, respectively.

The New Jersey Department of Environmental Protection reported in 2004 that 0.010 µg/g of terbacil was detected in the squash grown in the State (NJFMEP, 2004).

Maier-Bode et al.(1970) reviewed the presence of terbacil residues, along with other herbicides in cultivated crops in Germany. They determined that when terbacil was used as indicated by the directions, the chemical did not leave any measurable residues at harvest time.

5.1.2 Concentrations in Fish and Shellfish

EPA's Registration Eligibility Decision (RED) for Terbacil (1998a) noted that terbacil bioaccumulated (<8 µg/g) in bluegill sunfish tissues under static conditions of 0.01 and 1.00 µg/mL, but declined below the detection limit (<0.01 µg/g) within 3 days of depuration. It was thus concluded that terbacil did not bioaccumulate in fish tissues.

5.1.3 Intake of Terbacil from Food

Data on dietary concentrations from food were not located in the available literature. Consequently, calculating the intake of terbacil from food is not possible. However, pesticide tolerance values have been established for terbacil in a variety of fruits, including berries, and asparagus (U.S. EPA, 2006). Accordingly, some exposure through ingestion of these foods is possible. Terbacil does not bioaccumulate in fish; therefore, it is anticipated that there would typically be no chronic exposure to terbacil via fish consumption.

5.2 Exposure from Air

Terbacil is used as an herbicide. Although terbacil does not readily volatilize due to its low vapor pressure and low Henry's Law constant, when in the atmosphere, terbacil may exist in the vapor and particulate phase (HSDB, 2004). Terbacil may enter the atmosphere as a result of being sprayed onto fields where crops are grown for weed control. Data on concentrations of terbacil in air were not located in the available literature; consequently, the intake of terbacil from air cannot be calculated.

5.3 Exposure from Soil

The half-life of terbacil in a variety of soils suggests that terbacil, its degradates, or both can persist in treated areas for many months after treatment (Chapter 3). However, results of terbacil monitoring in ambient soils were not identified in the published literature. Because terbacil use is not wide spread, exposure from soils in nontreated areas is unlikely.

5.4 Other Residential Exposures (not drinking water related)

No data were identified for residential exposures to terbacil.

5.5 Occupational Exposures

An occupational exposure assessment is required for an active ingredient if (1) certain toxicological criteria are triggered and (2) there is potential exposure to handlers during use or to persons entering treated sites after application is complete. The Toxicity Endpoint Selection Committee found that neither dermal nor inhalation toxicity criteria were triggered for terbacil (U.S. EPA, 1998a). Although terbacil is present in certain manufacturing settings, normal control measures usually limit the amount of worker exposure. Industrial employees, such as railroad workers, and agricultural workers are exposed to the wettable powders and aqueous emulsions (Clayton and Clayton, 1993-1994). In occupational settings where terbacil is produced or used, inhalation of dusts and sprays along with skin contact with dusts, emulsions, and sprays are the two main routes of exposure (Clayton and Clayton, 1993-1994). Data on concentrations of terbacil in the work environment were not located in the available literature.

5.6 Summary

There is currently no substantial data documenting the concentration and estimated intake values of terbacil from media other than water.

6.0 HAZARD AND DOSE-RESPONSE ASSESSMENT

6.1 Characterization of Hazard

6.1.1 Synthesis and Evaluation of Major Noncancer Effects

Information regarding the noncancerous and cancerous effects of terbacil were identified primarily from the EPA re-registration eligibility decision on terbacil (U.S. EPA, 1998a). A recent literature search did not result in the identification of any newly published material. Consequently, the studies discussed below are those reported in the re-registration document and are noted as secondary sources. There were no epidemiological, case, or other studies in humans identified. Experimental studies in animals and *in vitro* systems characterize the major effects that are attributable to terbacil exposure.

Terbacil is toxic to rodents and rabbits when exposed orally, dermal, and by inhalation at high concentrations. Table 6-1 summarizes the acute toxicity tests and results for terbacil. As depicted in the table, rats exhibited an oral LD_{50} of > 5000 mg/kg/day when exposed to terbacil 80% wettable powder (Haskell Laboratories, 1965a,b). Additionally, terbacil causes mild ocular irritation in rabbits; however, it does not cause dermal sensitization in guinea pigs.

Table 6-1 Acute Toxicity Data for Terbacil

Test [a]	%AI	Species	Result	Reference
Oral LD_{50}	80.0	Rats	> 5000 mg/kg/day	Haskell Laboratories, 1965a,b
Inhalation LC_{50}	97.8	Rats	> 4.4 mg/L	Burgess et al., 1982
Dermal LD_{50}	80.0	Rabbits	> 5000 mg/kg/day	Haskell Laboratories, 1965a,b
Eye Irritation	96.1	Rabbits	Mild conjunctival irritant up to 72 hours	Hood, 1966
Dermal Sensitization	96.1	Guinea Pigs	Not a dermal sensitizer	Henry, 1986

a Exposure scenarios for the acute oral toxicity studies were not provided in detail in U.S. EPA (1998a).

Only one oral subchronic study was identified, in which many of the details regarding the exposure scenario and study conduct are not available. According to the information provided, rats were exposed to terbacil (% a.i. not reported) for a 90-day feeding period. An NOAEL of 100 ppm (equivalent to 5 mg/kg/day) and LOAEL of 500 ppm (equivalent to 25 mg/kg/day) were established based on increased absolute and relative liver weights, vacuolization, and hypertrophy of hepatocytes (Haskell Laboratories, 1965c; Wazeter et al., 1964).

One subchronic dermal toxicity study was reported in the EPA re-registration eligibility decision on terbacil. In the study, terbacil (80% a.i.) was applied to prepared skin at 5000 mg/kg/day, 5 hours/day, 5 days/week, over 21 days to male and female rabbits (Hood, 1966). There was no systemic toxicity observed; mild scaling and staining were reported at the test sites.

Several chronic toxicity studies have been conducted. As in the oral subchronic study, two chronic studies also resulted in liver toxicity. Terbacil (80% a.i.) was administered via diet to beagle dogs (4/sex/group) for 2 years at concentrations of 50, 250, or 2500/10,000 ppm (equivalent to 1.25, 6.25, and 62.5/250 mg/kg/day, respectively). It was not reported (within the Reregistration Eligibility Decision for Terbacil; U.S. EPA, 1998a) when the increase in the maximum dose level occurred. An NOAEL of 50 ppm (equivalent to 1.25 mg/kg/day) and an LOAEL of 250 ppm (equivalent to 6.25 mg/kg/day) were established based on increased thyroid to body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels. Relative liver weights also were increased at 2500 and 10,000 ppm in dogs that were sacrificed at both 1 and 2 years (Wazeter et al., 1967a).

In another chronic toxicity study (Malek, 1993), terbacil (97.4% a.i.) was administered via diet to male and female Sprague-Dawley rats (Crl:CD BR) for two years. Administered concentrations were 0, 25, 1500, or 7500 ppm (approximately equivalent to 0, 0.9, 58, and 308 mg/kg/day for males, respectively; 0, 1.4, 83, and 484 mg/kg/day for females, respectively). According to the study design, an interim sacrifice (10 animals/sex/dose) occurred 12 months into the study. Excessive mortality was observed in the control and low-dose groups, and the study was terminated at 23 months. No treatment-related clinical signs of toxicity were reported.

Treatment-related effects of the study included significantly decreased body weight (7500-ppm males and females; 1500-ppm females) and body weight gain (7500-ppm males and females; 1500-ppm females), and increased serum cholesterol levels (significant at 7500 ppm for males and females; marginal increase at 1500 ppm for females). Hepatotoxic effects included significantly increased mean liver to body weight ratios (7500-ppm males and females; 1500-ppm females) and significantly increased mean liver weight (7500-ppm males). These increases were accompanied by an increase in centrilobular hepatocyte hypertrophy (7500-ppm males and females; 1500-ppm females), biliary hyperplasia (7500-ppm females), and eosinophilic folic of cellular alteration in the liver. Eosinophilic folic of cellular alteration in the liver was observed in treated males and females with a significant trend; however, this finding is considered of equivocal importance because it was not accompanied by hypertrophic or hyperplastic changes or hepatocellular tumors). There was no evidence of increased tumor incidence in the treated animals when compared to the controls. Although excess mortality was observed in the control and low-dose groups, a systemic NOAEL of 25 ppm (equivalent to 0.9 mg/kg/day for males and 1.4 mg/kg/day for females) and LOAEL of 1500 ppm (equivalent to 56 mg/kg/day for males and 83 mg/kg/day for females) based on the liver effects and decreased body weight gain in females was established (Malek, 1993).

The excessive mortality in the study conducted by Malek (1993), raises concerns to the overall quality and conduct of the study. However, both studies showed evidence of liver toxicity and set NOAEL and LOAEL values based on liver effects and increased liver weights. This evidence supports the theory that the target organ for terbacil is the liver.

Two developmental studies regarding terbacil were identified. In a study conducted by Haskell Laboratories (1980), terbacil (% a.i. not reported) was administered via diet to female rats at concentrations of 0, 250, 1250 or 5000 ppm (equivalent to 0, 12.5, 62.5, and 250

mg/kg/day, respectively) from gestation days (gd) 6 through 15. A developmental NOAEL of 250 ppm (12.5 mg/kg/day) and an LOAEL of 1250 ppm (62.5 mg/kg/day) were established, based on a significantly decreased number of live fetuses per litter apparently due to fetal loss occurring before or near the time of implantation. The maternal NOAEL was determined to be 250 ppm (12.5 mg/kg/day), and the LOAEL, based on decreased body weight, was determined to be 1250 ppm (62.5 mg/kg/day).

Terbacil (% a.i. not reported) also was administered via gavage to rabbits at concentrations of 0, 30, 200, or 600 mg/kg/day on gd 7 through 19. The maternal NOAEL was 200 mg/kg/day and the maternal LOAEL was 600 mg/kg/day, based on maternal deaths (5 died and 2 were sacrificed *in extremis*). The developmental NOAEL was 200 mg/kg/day and the LOAEL was 600 mg/kg/day, based on decreased live fetal weights (Solomon, 1984).

One reproductive study regarding terbacil was identified. Terbacil (% a.i. not reported) was administered via diet to male and female rats at concentrations of 0, 50, or 250 ppm (equivalent to 0, 2.5, and 12.5 mg/kg/day, respectively) over 3 generations. The first litter of each generation was discarded, while the second litter was bred to produce the next generation. A systemic NOAEL of $\leq$50 ppm (2.5 mg/kg/day) and an LOAEL of 250 ppm were established based on decreased body weight gains in 250 ppm male offspring. This effect was not considered to be a reproductive effect because the decreased weight gain appeared at late periods in the study and not in the early development of the offspring. No reproductive effects were observed and, therefore, the reproductive NOAEL was $\geq$250 ppm (12.5 mg/kg/day) (Wazeter, 1967b).

Lastly, technical terbacil (96.1% a.i.) was tested and found negative for clastogenicity in a chromosomal aberration study in rat bone marrow cells, at doses up to 500 mg/kg (Cortina, 1984). It also was negative in a CHO (HGPRT) (Chinese hamster ovary cell/hypoxanthine-guanine phosphoribosyl-transferase) gene mutation assay when tested up to cytotoxic levels, with and without S9 activation (cytotoxicity > 3.0 mM without activation; > 2.75 mM with activation) (Haskell Laboratories, 1984). Technical terbacil (% a.i. not reported) also was negative for unscheduled DNA synthesis when tested up to cytotoxic levels (5 mM) in the rat.

6.1.2 Synthesis and Evaluation of Carcinogenic Effects

Information regarding the noncancerous and cancerous effects of terbacil were identified primarily from the EPA re-registration eligibility decision on terbacil (U.S. EPA, 1998a). A recent literature search did not result in the identification of any newly published material. Consequently, the studies discussed below are those reported in the re-registration document and are noted as secondary sources. There were no epidemiological, case, or other studies in humans identified. Experimental studies in animals and *in vitro* systems characterize the major effects that are attributable to terbacil exposure

Two oral studies examined the carcinogenic effects of terbacil. Both studies conclude that oral administration of terbacil did not show evidence of increased tumor incidence in the treated animals when compared to the controls.

In the 2-year dietary study in rats by Malek (1993) (details provided above), there was no evidence of carcinogenicity reported. Goldenthal et al. (1981) administered terbacil (% a.i. not reported) via diet to male and female mice in a 2-year oncogenicity study at doses of 0, 50, 1250, or 5000/7500 ppm (equivalent to 7, 179, and 714/1071 mg/kg/day). The increase in the maximum dose level occurred after week 54. A systemic NOAEL of 50 ppm is based on the LOAEL of 1250 ppm, which resulted in mild hypertrophy of the centrilobular hepatocytes, and decreased pituitary weights in males. Pituitary weights also were decreased in high-dose females. Additionally, there was an increased incidence lung neoplasms (adenomas and adenocarcinomas) in all treated male mice. The increases were within the range of similar tumors observed in historical control mice, and therefore not considered to be treatment related. Administration of terbacil did not significantly increase the incidence of any proliferative hepatocellular carcinomas, single/multiple adenomas, foci of cellular alteration, or combined hepatocellular adenomas and carcinomas in either sex

6.1.3 Weight of Evidence Evaluation for Carcinogenicity

Terbacil is classified as *not likely to be carcinogenic to humans* (U.S. EPA, 2005a). This is because animal evidence failed to demonstrate a carcinogenic effect in at least two well-designed and well-conducted studies in two appropriate animal species (U.S. EPA, 2005a).

6.1.4 Potentially Sensitive Populations

There were no potentially sensitive populations identified. Data do not suggest increased pre- or post-natal sensitivity of children and infants to terbacil exposure because developmental NOAELs were the same as those for maternal toxicity. Additionally, the NOAEL for systemic (parental) toxicity was set at a lower concentration than the NOAEL for reproductive toxicity, indicating that the reproductive system is less sensitive to terbacil.

6.2 Reference Dose

The reference dose (RfD) is an estimate of the daily oral exposure to the human population that is likely to be without appreciable risk of deleterious effects over a lifetime. The RfD is derived from the NOAEL in the critical or most sensitive study, which is then divided by a variable uncertainty factor as follows:

$$\text{RfD} = 1.25 \text{ mg/kg/day}/100 = \sim 0.013 \text{ mg/kg/day}$$

where 1.25 is the NOAEL from the critical study (Wazeter et al., 1967a) and 100 is the uncertainty factor.

6.2.1 Choice of Principle Study and Critical Effect

The principal study for determining the RfD is a chronic toxicity study, in which beagle dogs (4/sex/group) were administered terbacil via diet for 2 years at concentrations of 50, 250, or 2500/10,000 ppm (equivalent to 1.25, 6.25, 62.5/250 mg/kg/day, respectively) (Wazeter et al., 1967a). The NOAEL was determined to be 1.25 mg/kg/day and the LOAEL was determined to be 6.25 mg/kg/day, based on increased thyroid to body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels.

6.2.2 Application of Uncertainty Factor(s) and Modifying Factor(s)

An uncertainty factor of 100 is used in calculating the RfD to ensure the protection of infants and children from exposure to terbacil. A factor of 10 is used to account for interspecies differences, while another factor of 10 is used to account for intraspecies differences.

6.3 Carcinogen Assessment

Terbacil is classified as not likely to be carcinogenic to humans (U.S. EPA, 1998a) according to the 1996 draft of the Agency's revised procedures for carcinogen risk assessment. This classification remains appropriate under the final 2005 guidelines. Accordingly, there is no need for a quantitative assessment of cancer risk.

6.4 Sensitive Population Considerations

The available literature does not suggest any increased pre- or post-natal sensitivity of children and infants to terbacil (see Section 6.1.5), nor any indication of gender sensitivity. Therefore, there are no special considerations needed for a sensitive population

6.5 Post Re-registration Health Effects Publications

There were no post re-registration health effects publications identified.

6.6 CCL Health Reference Level

The CCL health reference level is 0.091 mg/L. EPA derived the HRL using an RfD approach as follows: HRL = (RfD ×70 kg)/2 L/day × RSC, where:

RfD = An estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure (mg/kg/day) to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. It can be derived from an NOAEL, LOAEL, or BMD, with uncertainty factors generally applied to reflect limitations of the data used;

70 kg = The assumed body weight of an adult;

2 L = The assumed daily water consumption of an adult;

RSC = The relative source contribution, or the level of exposure believed to result from drinking water when compared to other sources (e.g., air), and is assumed to be 20% unless noted otherwise.

Therefore, the HRL = $\frac{0.013 \text{ mg/kg/day} \times 70\text{kg}}{2\text{L/day}} \times 0.20 = 0.091$ mg/L

A discussion of the HRL as a benchmark for evaluating occurrence using monitoring data from public water systems is found in Section 4.3.2.

7.0 REGULATORY DETERMINATION AND CHARACTERIZATION OF RISK FROM DRINKING WATER

7.1 Regulatory Determination for Chemicals on the CCL

The Safe Drinking Water Act (SDWA), as amended in 1996, required the Environmental Protection Agency (EPA) to establish a list of contaminants to aid the Agency in regulatory priority setting for the drinking water program. EPA published a draft of the first Contaminant Candidate List (CCL) on October 6, 1997 (62 Federal Register [FR] 52193, U.S. EPA, 1997). After review of and response to comments, the final CCL was published on March 2, 1998 (63 FR 10273, U.S. EPA, 1998d).

On July 18, 2003 EPA announced final Regulatory Determinations for one microbe and 8 chemicals (68 FR 42897, U.S. EPA, 2003a) after proposing those determinations on June 3, 2002 (67 FR 36222, U.S. EPA, 2002b). The remaining 40 chemicals and ten microbial agents from the first CCL became CCL 2 and were published in the Federal Register on April 2, 2004 (69 FR 17406, U.S. EPA 2004b) and finalized on February 24, 2005 (70 FR 9071, U.S. EPA, 2005b).

EPA proposed Regulatory Determinations for 11 chemicals from CCL2 on May 1, 2007 (72FR 24016) (U.S. EPA, 2007). Determinations for all 11 chemicals were negative based on a lack of national occurrence at levels of health concern. The Agency is given the freedom to determine that there is no need for a regulation if a chemical on the CCL fails to meet one of three criteria established by the SDWA and described in section 7.1.1. After review of public comments and submitted data, the negative determinations for the 11 contaminants have been retained. Each contaminant will be considered in the development of future CCLs if there are changes in health effects and/or occurrence.

7.1.1 Criteria for Regulatory Determination

These are the three criteria used to determine whether or not to regulate a chemical on the CCL:

- The contaminant may have an adverse effect on the health of persons.

- The contaminant is known to occur or there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern.

- In the sole judgment of the Administrator, regulation of such contaminant presents a meaningful opportunity for health risk reduction for persons served by public water systems.

The findings for all criteria are used in making a determination to regulate a contaminant. As required by the SDWA, a decision to regulate commits the EPA to publication of a Maximum Contaminant Level Goal (MCLG) and promulgation of a National Primary Drinking Water Regulation (NPDWR) for that contaminant. The Agency may determine that there is no need for a regulation when a contaminant fails to meet one of the criteria. A decision not to regulate is

considered a final Agency action and is subject to judicial review. The Agency can choose to publish a Health Advisory (a nonregulatory action) or other guidance for any contaminant on the CCL independent of the regulatory determination.

7.1.2 National Drinking Water Advisory Council Recommendations

In March 2000, the EPA convened a Working Group under the National Drinking Water Advisory Council (NDWAC) to help develop an approach for making regulatory determinations. The Working Group developed a protocol for analyzing and presenting the available scientific data and recommended methods to identify and document the rationale supporting a regulatory determination decision. The NDWAC Working Group report was presented to and accepted by the entire NDWAC in July 2000.

Because of the intrinsic difference between microbial and chemical contaminants, the Working Group developed separate but similar protocols for microorganisms and chemicals. The approach for chemicals was based on an assessment of the impact of acute, chronic, and lifetime exposures, as well as a risk assessment that includes evaluation of occurrence, fate, and dose-response. The NDWAC protocol for chemicals is a semi-quantitative tool for addressing each of the three CCL criteria. The NDWAC requested that the Agency use good judgment in balancing the many factors that need to be considered in making a regulatory determination.

The EPA modified the semi-quantitative NDWAC suggestions for evaluating chemicals against the regulatory determination criteria and applied them in decision-making. The quantitative and qualitative factors for terbacil that were considered for each of the three criteria are presented in the sections that follow.

7.2 Health Effects

The first criterion asks if the contaminant may have an adverse effect on the health of persons. Because all chemicals have adverse effects at some level of exposure, the challenge is to define the dose at which adverse health effects are likely to occur, and estimate a dose at which adverse health effects are either not likely to occur (threshold toxicant), or have a low probability for occurrence (non-threshold toxicant). The key elements that must be considered in evaluating the first criterion are the mode of action, the critical effect(s), the dose-response for critical effect(s), the reference dose (RfD) for threshold effects, and the slope factor for nonthreshold effects.

A full description of the health effects information and dose-response assessment associated with exposure to terbacil is presented in Chapter 6 of this document and summarized below in Sections 7.2.2 and 7.2.3.

7.2.1 Health Criterion Conclusion

There are no current studies that examine the human health effects due to terbacil exposure. According to dog and rat studies, the liver appears to be the target organ in oral subchronic and chronic studies. Observed hepatotoxic effects include increased liver weights

(absolute and/or relative) (Haskell Laboratories, 1965c; Malek, 1993; Wazeter et al., 1964; Wazeter et al., 1967a); increased incidence of vacuolization and hypertrophy of hepatocytes (Haskell Laboratories, 1965c; Wazeter et al., 1964); and increased incidence in centrilobular hepatocyte hypertrophy, biliary hyperplasia, and eosinophilic foci of cellular alteration in the liver (Malek, 1993). Additionally, terbacil is acutely toxic to rodents and rabbits when exposure occurs orally, dermally, and by inhalation at high concentrations; terbacil was negative in assays of mutagenicity.

The RfD, which was verified by EPA (1989b), is ~0.013 mg/kg/day. This value was calculated using an NOAEL of 1.25 mg/kg/day (Wazeter et al., 1967a) that was divided by a 100-fold uncertainty factor, which accounted for inter- and intraspecies differences. The critical effect associated with the RfD is increased thyroid to body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels.

Based on these considerations, the evaluation of the first criterion for terbacil is positive; terbacil may have an adverse effect on human health.

7.2.2 Hazard Characterization and Mode of Action Implications

Terbacil is acutely toxic to rodents and rabbits when exposure occurs orally, dermally, and by inhalation at high concentrations. Rats exhibited an oral LD_{50} of > 5000 mg/kg/day when exposed to 80% terbacil as a wettable powder (Haskell Laboratories, 1965a,b). Additionally, terbacil causes mild ocular irritation in rabbits; however, it does not cause dermal sensitization in guinea pigs.

The liver appears to be the target organ for terbacil, with oral subchronic and chronic studies in dogs and rats showing hepatotoxic effects. Observed hepatotoxic effects include increased liver weights (absolute and/or relative) (Haskell Laboratories, 1965c; Malek, 1993; Wazeter et al., 1964; Wazeter et al., 1967a); increased incidence of vacuolization and hypertrophy of hepatocytes (Haskell Laboratories, 1965c; Wazeter et al., 1964); and increased incidence in centrilobular hepatocyte hypertrophy, biliary hyperplasia, and eosinophilic foci of cellular alteration in the liver (Malek, 1993).

Additionally, developmental and reproductive effects have been observed in rats and rabbits. Developmental effects included significantly decreased number of live fetuses per litter apparently due to fetal loss occurring before or near the time of implantation in rats (Haskell Laboratories, 1980) and decreased live fetal weights in rabbits (Solomon, 1984). Additionally, decreased body weight gains in 250-ppm male offspring were observed when terbacil was administered orally over 3 generations at doses of 0, 50, or 250 ppm (equivalent to 0, 2.5, and 12.5 mg/kg/day, respectively). These results, however, do not suggest that offspring exhibited an increase in pre- or post-natal sensitivity to terbacil exposure because developmental NOAELs were the same as those for maternal toxicity. Additionally, the NOAEL for systemic (parental) toxicity was set at a lower concentration than the NOAEL for reproductive toxicity, indicating that the reproductive system is less sensitive to terbacil than other organ systems.

7.2.3 Dose-Response Characterization and Implications in Risk Assessment

The RfD, which was verified by EPA (1989b), is ~0.013 mg/kg/day. This value was calculated using an NOAEL of 1.25 mg/kg/day (Wazeter et al., 1967a) that was divided by a 100-fold uncertainty factor accounting for inter- and intraspecies differences. The critical effect associated with the RfD is increased thyroid to body weight ratios, slight increases in liver weights, and elevated alkaline phosphatase levels.

Terbacil is not genotoxic. Terbacil was tested and found negative in a chromosomal aberration study in rat bone marrow cells, found negative in a gene mutation assay (with and without S9 activation), and did not induce unscheduled DNA synthesis when tested up to cytotoxic levels in rats (Cortina, 1984; Haskell Laboratories,1984). Terbacil is classified as *not likely to be carcinogenic to humans* because animal evidence failed to demonstrate a carcinogenic effect in at least two well-designed and well-conducted studies in two appropriate animal species (U.S. EPA, 2005a).

The health reference level (HRL) for terbacil is established by using its RfD (0.013 mg/kg/day), and applying a lifetime Health Advisory methodology with a 20% relative source contribution. The HRL is calculated to be 0.091 mg/L or 90 µg/L when using the round number.

7.3 Occurrence in Public Water Systems

The second criterion asks if the contaminant is known to occur or if there is a substantial likelihood that the contaminant will occur in public water systems with a frequency and at levels of public health concern. In order to address this question the following information was considered:

- Monitoring data from public water systems
- Ambient water concentrations and releases to the environment
- Environmental fate

Data on the occurrence of terbacil in public drinking water systems were the most important determinants in evaluating the second criterion. EPA looked at the total number of systems that reported detections of terbacil, as well as those that reported concentrations of terbacil above an estimated drinking-water HRL. For noncarcinogens, the estimated HRL level was calculated from the RfD assuming that 20% of the total exposure would come from drinking. For carcinogens, the HRL was the 10^{-6} risk level (i.e, the probability of one excess tumor in a population of a million people). The HRLs are benchmark values that were used in evaluating the occurrence data while the risk assessments for the contaminants were being developed.

The available monitoring data, including indications of whether or not the contaminant is a national or a regional problem, are included in Chapter 4 of this document and summarized below. Additional information on production, use, and fate are found in Chapters 2 and 3.

7.3.1 Occurrence Criterion Conclusion

The available data for terbacil production, use, and environmental releases all show an increasing trend. However, monitoring data show no detections of terbacil in any of the large (i.e., serving more than 10,000 people) community water systems (CWSs), large non-transient non-community water systems (NTNCWSs), or the statistically representative national sample of 800 small (i.e., serving 10,000 people or fewer) CWSs and NTNCWSs.

The physicochemical properties of terbacil suggest that terbacil appears to be persistent (i.e., does not break down easily) in terrestrial areas and may reach groundwater sources due to its mobility in water. Because of these physicochemical properties, coupled with the increasing use of terbacil, there is some concern regarding terbacil exposure.

Based its physicochemical properties and increasing use, it is unclear whether terbacil will occur in public water systems at frequencies or concentration levels that are of public health concern. Thus, the evaluation for the second criterion is equivocal.

7.3.2 Monitoring Data

Under the National Water-Quality Assessment (NAWQA) program, U.S. Geological Survey (USGS) monitored terbacil between 1992 and 2001 in representative watersheds and aquifers across the country. Terbacil was not monitored in bed sediment or biota. Reporting limits varied but did not exceed 0.034 µg/L. In surface water, terbacil was detected at the following frequencies in samples: 1.40% in undeveloped land settings; 1.82% in mixed land-use settings; 1.98% in urban settings; and 4.52% in agricultural settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest maximum concentration, estimated at 0.540 µg/L, occurred in an agricultural land-use setting (Martin et al., 2003).

In ground water, terbacil detection frequencies were as follows: 0.0% in undeveloped settings; 0.26% in mixed land-use (major aquifer) settings; 0.76% in agricultural settings; and 1.20% in urban land-use settings. The 95th percentile concentrations were less than the reporting limit in all settings. The highest concentration, 0.891 µg/L, was in a mixed land-use (major aquifer) setting (Kolpin and Martin, 2003).

Additionally, the first Unregulated Contaminant Monitoring Regulation (UCMR1) collected information on the national occurrence of select emerging contaminants in drinking water. EPA designed the UCMR1 data collection with three parts (or tiers), primarily based on the availability of analytical methods. Terbacil belonged to the first tier, List 1. As a List 1 contaminant, EPA requires all large PWSs (systems serving more than 10,000 people), plus a statistically representative national sample of 800 small PWSs (systems serving 10,000 people or fewer) to conduct Assessment Monitoring, with the exception of transient non-community systems and systems that purchase 100% of their water.

Approximately one-third of the participating small systems were scheduled to monitor for these contaminants during each calendar year from 2001 through 2003. Large systems could

conduct one year of monitoring anytime during the 2001-2003 UCMR1 period. EPA specified a quarterly monitoring schedule for surface water systems and a twice-a-year, six-month interval monitoring schedule for ground water systems. Although UCMR1 monitoring was conducted primarily between 2001 and 2003, some results were not collected until as late as 2006.

The data presented in this report reflect UCMR1 analytical samples submitted and quality-checked under the regulation as of March 2006. Terbacil data were collected and submitted by 797 (99.6 percent) of the 800 small systems selected for the small system sample and 3,076 (99.2 percent) of the 3,100 large systems defined as eligible for the UCMR1 large system census. Terbacil data have been analyzed at the level of simple detections (at or above the minimum reporting level, ≥MRL, or ≥2 µg/L), exceedances of the health reference level (>HRL, or >90 µg/L), and exceedances of one-half the value of the HRL (>½HRL, or >45 µg/L). No detections of terbacil were found in any samples, and thus there were also no exceedances of the HRL or one-half the HRL.

7.3.3 Use and Fate Data

Terbacil is an herbicide used to selectively control many annual and some perennial weeds (Tomlin, 1997) in crops (e.g., apples, mint, sugarcane, asparagus, blackberries, boysenberries, dewberries, loganberries, raspberries, youngberries, strawberries, and peaches), forestry (e.g., cottonwood), and feed crops (e.g., alfalfa, sainfoin, and forage) (U.S. EPA, 1998a). The chemical in the form of 80% (20% a.i.) wettable powder (WP; EPA Reg. No. 352-317) is manufactured from an unregistered Technical Grade Active Ingredient (TGAI) (95% a.i) by E.I. du Pont de Nemours and Company, Inc. (U.S. EPA, 1998a).

Although there was an increase in the use of terbacil during the 1990s, recent monitoring data indicate that there were no detections of terbacil in any of the finished water samples, and thus no exceedances of the HRL or one-half the HRL.

Although terbacil has not been detected in finished water samples to date, fate data indicate that depending on the use of the herbicide and the amount of rain, the compound may infiltrate groundwater sources. This is due to terbacil's low sorption affinity to soil (K_{ad}=0.39 to 1.3 mL/g; K_{oc}=44 to 61 mL/g) and relatively high solubility in water (710 mg/L). Additionally, terbacil appears to be persistent (i.e., does not break down easily) in terrestrial environments. The low vapor pressure (4.8×10^{-7} mmHg at 29.5°C) and Henry's Law constants (1.9×10^{-9} atm m^3/mole), suggest that terbacil is not likely to volatilize into the air to a significant extent.

The BCF (the bioconcentration factor) of terbacil is estimated to be 16, which indicates that bioconcentration of terbacil in aquatic organisms is unlikely (HSDB, 2004). Its log K_{ow} (Hansch, 1995) and regression-derived equation (Meylan, 1999) also point to a low estimated bioconcentration of terbacil in aquatic organisms.

Because of its physicochemical properties and increasing use, there is some concern regarding terbacil exposure.

7.4 Risk Reduction

The third criterion asks if, in the sole judgment of the Administrator, regulation presents a meaningful opportunity for health risk reduction for persons served by public water systems. In evaluating this criterion, EPA looked at the total exposed population, as well as the population exposed to levels above the estimated HRL. Estimates of the populations exposed and the levels to which they are exposed were derived from the monitoring results. These estimates are included in Chapter 4 of this document and summarized in section 7.4.2 below.

In order to evaluate risk from exposure through drinking water, EPA considered the net environmental exposure in comparison to the exposure through drinking water. For example, if exposure to a contaminant occurs primarily through ambient air, regulation of emissions to air provides a more meaningful opportunity for EPA to reduce risk than does regulation of the contaminant in drinking water. In making the regulatory determination, the available information on exposure through drinking water (Chapter 4) and information on exposure through other media (Chapter 5) were used to estimate the fraction that drinking water contributes to the total exposure. The EPA findings are discussed in Section 7.4.3 below.

In making its regulatory determination, EPA also evaluated effects on potentially sensitive populations, including the fetus, infants and children. Sensitive population considerations are included in section 7.4.4.

7.4.1 Risk Criterion Conclusion

The presence of terbacil in water is rare. To date, there have been no detections of terbacil in any of the analyzed samples. Consequently, there also have been no exceedances of the HRL or one-half of the HRL. Thus, the evaluation of the third criterion is negative.

7.4.2 Exposed Population Estimates

Terbacil was scheduled to be monitored in all large (i.e., serving more than 10,000 people) community water systems (CWSs) and large non-transient non-community water systems (NTNCWSs), plus a statistically representative national sample of 800 small (i.e., serving 10,000 people or fewer) CWSs and NTNCWSs. As of March 2006, there have been no detections of terbacil in any of the samples. Therefore, it appears that the general population is not exposed to terbacil through water consumption or use.

7.4.3 Relative Source Contribution

Relative source contribution analysis compares the magnitude of exposure expected via drinking water to the magnitude of exposure from intake of terbacil in other media, such as food, air, and soil. In situations where terbacil occurs in drinking water, the water is likely to be the major source of exposure. There are no national data for the intake of terbacil in foods, air, or soil. Recent residue measurements on foods indicate that 0.010 µg/g of terbacil was detected in the squash grown in New Jersey (NJFMEP, 2004). Additionally, Cessna (1991) conducted a two year study on terbacil residues extracted from asparagus spears from established agricultural

sites in British Columbia and Ontario following pre-emergence and early post-emergence applications at 0.6, 1.1, and 2.2 kg/ha (hectare). At one site, maximum residues in the pre-emergence samples were found to be 14±3 µg/kg for the 2.2 kg/ha application rate, whereas maximum residues in the early post-emergence samples for the 2.2 kg/ha application rate were 493±250 µg/kg at a second site. Recoveries of terbacil from fortified asparagus tissue were 96±19%, 87.2±11.9% and 83.3±7.8% at 10, 50, and 100 µg/kg, respectively. Concentrations of terbacil in air and soil have not been reported. However, these exposure routes should be considered when analyzing the relative source contribution. This is because terbacil may exist in the vapor and particulate phase (HSDB, 2004) as a result of being sprayed onto fields where crops are grown for weed control. Additionally, terbacil is believed to be persistent (i.e., does not break down easily) and mobile in soil depending on the application rate, soil type, and rainfall. Due to the lack of national data for the intake of terbacil in foods, air, or soil, an RSC value other than the default value of 20% is not needed.

7.4.4 Sensitive Populations

There were no potentially sensitive populations identified. Data do not suggest increased pre- or post-natal sensitivity of children and infants to terbacil exposure because developmental NOAELs were the same as those for maternal toxicity. Additionally, the NOAEL for systemic (parental) toxicity was set at a lower concentration than the NOAEL for reproductive toxicity, indicating that the reproductive system is less sensitive to terbacil than are other systems.

7.5 Regulatory Determination Decision

As stated in Section 7.1.1, a positive finding for all three criteria is required in order to make a determination to regulate a contaminant. In the case of terbacil, the criterion on health effects is positive and the criterion on occurrence is equivocal. Terbacil may have an adverse effect on human health. Although monitoring is not yet complete, data available as recently as March 2006 have been analyzed and indicate that there were no detections of terbacil in any of the water samples analyzed. As a result, no exceedances of the HRL or one-half the HRL were reported. However, because of its physicochemical properties and increasing use, there is some concern with terbacil exposure. Nevertheless, because a positive finding was not met for all three criteria, a determination to regulate terbacil is not appropriate.

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APPENDIX A: Abbreviations and Acronyms

A	acre
a.i.	active ingredient
atm	atmosphere
BCF	bioaccumulation factor
BMD	benchmark dose
CAS	Chemical Abstracts Registry
CCL	Contaminant Candidate List
CHO (HGPRT)	Chinese hamster ovary cell/hypoxanthine-guanine phosphoribosyl-transferase
cm	centimeter
CWS	community water system
EEC	estimated environmental concentration
EPA	Environmental Protection Agency
FR	Federal Register
GENEEC	Generic Expected Environmental Concentration
GUS	Groundwater Ubiquity Score
ha	hectare
Hg	mercury
hr	hour
HRL	health reference level
HSDB	Hazardous Substances Database
K_{ads}	adsorption coefficient
kg	kilogram
K_{oc}	organic carbon partitioning coefficient
K_{ow}	octanol-water partition coefficient
L	liter
lb	pound
LOAEL	lowest-observed-adverse-effect-level
m	meter
MCLG	Maximum Contaminant Level Goal
mg	milligram
mL	milliliter
mm	millimeter
mM	millimolar
MRL	minimum reporting level
NAWQA	National Water Quality Assessment
NCOD	National Drinking Water Contaminant Occurrence Database
NDWAC	National Drinking Water Advisory Council
NJFMEP	New Jersey Department of Environmental Protection, Food Monitoring & Evaluation Program
NOAEL	no-observed-adverse-effect-level
NPDWR	National Primary Drinking Water Regulation
NTNCWS	non-transient non-community water system
OPP	Office of Pesticides Programs

PATRIOT	Pesticide Assessment Tool for Rating Investigations of Transport
PBPK	physiologically-based pharmacokinetic
ppm	parts per million
PWS	Public Water Systems
QAPP	Quality Assurance Project Plan
RED	Re-registration Eligibility Document
RfD	reference dose
RL	reporting level
RSC	relative source contribution
SCI-GRO	Screening Concentrations In Ground Water
SDTF	Spray Drift Task Force
SDWA	Safe Drinking Water Act
SVOCs	select semivolatile organic compounds
$t_{1/2}$	half-life
UCMR1	Unregulated Contaminant Monitoring Regulation 1
µg	microgram
U.S. EPA	United States Environmental Protection Agency
USGS	United States Geological Service
UV	ultraviolet
TRI	Toxic Release Inventory
VOC	volatile organic compound
WP	wettable powder