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Terbuthylazine (TBA) in Drinking-water

Background document for development of
WHO *Guidelines for Drinking-water Quality*

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Preface

One of the primary goals of WHO and its member states is that "all people, whatever their stage of development and their social and economic conditions, have the right to have access to an adequate supply of safe drinking water." A major WHO function to achieve such goals is the responsibility "to propose regulations, and to make recommendations with respect to international health matters ...."

The first WHO document dealing specifically with public drinking-water quality was published in 1958 as International Standards for Drinking-Water. It was subsequently revised in 1963 and in 1971 under the same title. In 1984–1985, the first edition of the WHO Guidelines for drinking-water quality (GDWQ) was published in three volumes: Volume 1, Recommendations; Volume 2, Health criteria and other supporting information; and Volume 3, Surveillance and control of community supplies. Second editions of these volumes were published in 1993, 1996 and 1997, respectively. Addenda to Volumes 1 and 2 of the second edition were published in 1998, addressing selected chemicals. An addendum on microbiological aspects reviewing selected microorganisms was published in 2002.

The GDWQ are subject to a rolling revision process. Through this process, microbial, chemical and radiological aspects of drinking-water are subject to periodic review, and documentation related to aspects of protection and control of public drinking-water quality is accordingly prepared/updated.

Since the first edition of the GDWQ, WHO has published information on health criteria and other supporting information to the GDWQ, describing the approaches used in deriving guideline values and presenting critical reviews and evaluations of the effects on human health of the substances or contaminants examined in drinking-water.

For each chemical contaminant or substance considered, a lead institution prepared a health criteria document evaluating the risks for human health from exposure to the particular chemical in drinking-water. Institutions from Canada, Denmark, Finland, France, Germany, Italy, Japan, Netherlands, Norway, Poland, Sweden, United Kingdom and United States of America prepared the requested health criteria documents.

Under the responsibility of the coordinators for a group of chemicals considered in the guidelines, the draft health criteria documents were submitted to a number of scientific institutions and selected experts for peer review. Comments were taken into consideration by the coordinators and authors before the documents were submitted for final evaluation by the experts meetings. A "final task force" meeting reviewed the health risk assessments and public and peer review comments and, where appropriate, decided upon guideline values. During preparation of the third edition of the GDWQ, it was decided to include a public review via the world wide web in the process of development of the health criteria documents.

During the preparation of health criteria documents and at experts meetings, careful consideration was given to information available in previous risk assessments carried out by the International Programme on Chemical Safety, in its Environmental Health

Criteria monographs and Concise International Chemical Assessment Documents, the International Agency for Research on Cancer, the joint FAO/WHO Meetings on Pesticide Residues, and the joint FAO/WHO Expert Committee on Food Additives (which evaluates contaminants such as lead, cadmium, nitrate and nitrite in addition to food additives).

Further up-to-date information on the GDWQ and the process of their development is available on the WHO internet site and in the current edition of the GDWQ.

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The work of the following coordinators was crucial in the development of this document and others in the Addendum:

P. Chambon, Health Environment Hygiene Laboratory of Lyon, Lyon, France (inorganic constituents)
U. Lund, Water Quality Institute, Horsholm, Denmark (organic constituents)
H. Galal-Gorchev, Urban Environmental Health, World Health Organization, Geneva, Switzerland (pesticides)
E. Ohanian, Environmental Protection Agency, Washington, DC, USA (disinfectants and disinfection by-products)

The coordinators for the overall administrative and technical aspects of this document were, respectively, J. Kenny and H. Galal-Gorchev, Urban Environmental Health, WHO, Geneva, Switzerland.

Ms Marla Sheffer of Ottawa, Canada, was responsible for the scientific editing of the document.

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GENERAL DESCRIPTION

Identity *(Tomlin, 1994)*
CAS no: 5915-41-3

Molecular formula: $C_9H_{16}ClN_5$

The IUPAC name for terbuthylazine (TBA) is 6-chloro-*N*-(1,1-dimethylethyl)-*N*'-ethyl-1,3,5-triazine-2,4-diamine.

Physicochemical properties *(Green, 1991)*

Property	*Value*
Melting point	177–179°C
Vapour pressure	0.15 mPa at 25°C
Volatility	0.014 mg/m^3 at 20°C
Density	1.188 at 20°C
Octanol–water partition coefficient	1096
Water solubility	8.5 mg/litre at 20°C
Hydrolytic stability (half-life in water at 20°C)	at pH 1: 8 days at pH 5: 86 days at pH 7: >200 days at pH 9: >200 days at pH 13: 12 days

Major uses *(Anonymous, 1989, 1995; Green, 1991; Schneider, 1995; Werner, 1996)*

TBA is a herbicide that belongs to the chloro-triazine family. In plants, it acts as a powerful inhibitor of photosynthesis. The substance is taken up through both roots and leaves and is distributed throughout the plant after being taken up through the roots. This enables it to be used in both pre- and post-emergence treatment. TBA is a selective herbicide for maize, sorghum, potatoes, peas, sugar cane, vines, fruit trees, citrus, coffee, oil palm, cocoa, olives, rubber, and forestry in tree nurseries and new planting. It is particularly effective against annual dicotyledons.

Environmental fate

Metabolism of TBA in maize was investigated using ^{14}C-labelled TBA. When TBA was applied to the leaves, most of the radioactivity remained *in situ*; when applied to the roots, it quickly spread throughout the entire plant. TBA degrades rapidly through the following process: hydrolysis at the chlorine substituent (to form the hydroxy-derivative) or *N*-dealkylation of a side chain, preferably the ethyl chain; *N*-dealkylation of the second side chain with formation of 2-chloro-4,6-diamino-*s*-triazine (or hydroxy-diamino-*s*-triazine = ammeline); and hydroxylation of the ammeline to 2-amino-4,6-dihydroxy-*s*-triazine (ammelide) (Green, 1991).

In soil, metabolism studies using ^{14}C-labelled TBA show cleavage of the side chains and hydrolysis of the chlorine substituent, followed by mineralization by cleavage of the heterocycle with the formation of nitrogen-containing derivatives and carbon dioxide. Bacteria and fungi are capable of breaking down *s*-triazines. Decomposition by means of photolysis was also observed on the surface of the soil. Degradation of TBA occurs under a variety of environmental conditions. The speed of decomposition is strongly influenced by temperature, moisture levels, microbial activity, pH, and aeration. Soil mobility studies showed an adsorption of TBA onto soil particles within 2 hours; adsorption increased with

humus content of the soil. The mobility of TBA is lower than that of atrazine but highly dependent upon soil type (Green, 1991; Anonymous, 1995; Schneider, 1995; Werner, 1996).

Degradation of TBA in natural water depends on the presence of sediments and biological activity. Although studies with Rhine River and pond water gave half-lives of >1 year, newer findings indicate a half-life of approximately 50 days. In studies investigating the photolysis of TBA in aqueous solution, a half-life of >1 month was estimated using a sun-like light source, in agreement with a reported half-life of about 3 months in natural sunlight. However, for very high sunlight intensities (full midday sunlight), the half-life was 39 hours (Green, 1991; Anonymous, 1995; Schneider, 1995; Palm & Zetzsch, 1996; Werner, 1996).

TBA has a low vapour pressure and can therefore be expected to have little volatility. Traces of TBA in air are diluted by diffusion and degraded by reactions with hydroxyl radicals. Under these conditions, the half-life of TBA in air is approximately 1.5 days (Zetzsch, 1993).

ANALYTICAL METHODS

TBA is extracted from water by solid-liquid extraction on RP-C18 material (RP = reversed phase), eluted with a solvent and then separated, identified and quantified by high-performance liquid chromatography using ultraviolet detection at 220 nm. The limit of detection is about 0.1 µg/litre (ISO, 1997).

ENVIRONMENTAL LEVELS AND HUMAN EXPOSURE

Air

No data are available, but results of rain analysis (Palm, 1995) indicate that TBA concentrations in air would be very low.

Water

TBA levels were monitored in the River Po (Italy) over a period of 3 years (1988–1991); concentrations between 0.0 and 0.3 µg/litre were observed (Brambilla, 1993). In the Friuli-Venezia area in Italy, well-water samples from seven wells were analysed up to 29 times between October 1991 and April 1992. TBA was detected in 7 of a total of 200 samples: 3 samples contained TBA concentrations between 0.1 and 0.2 µg/litre, and 4 contained TBA levels above 0.2 µg/litre (Barbina, 1993).

In Germany, a large survey of TBA in water was carried out over a 3-year period (1990–1992). Out of 9565 samples analysed, 94.2% were negative, 5.3% were positive (below 0.1 µg/litre), and 0.4% contained TBA at concentrations above 0.1 µg/litre. Samples were taken from drinking-water, groundwater, and surface water (Wolter, 1993). Among the drinking-water samples, 0.7% were positive. The detection limit was not given but was probably around 0.01 µg/litre.

In the Rhine River in 1994, the mean TBA concentration found was 0.013 µg/litre at Kembs and Laufenburg (Güggi, 1995). In 1995, mean TBA concentrations had reached 0.020 µg/litre at Kembs and 0.022 µg/litre at Laufenburg (Güggi, 1996).

In 1993, a survey was conducted at one of the main tributaries of the Nidda River (one of the tributaries of the Main River in Germany). Out of 105 samples analysed, 5 contained TBA concentrations above 0.1 µg/litre, with a maximum concentration of 0.28 µg/litre (Seel, 1994).

In Switzerland between March 1988 and March 1989, TBA was found in Lake Zurich at a mean concentration of 0.013 µg/litre. During the same period, rainwater was collected from roofs and analysed. Concentrations of TBA were between 0.01 and 0.198 µg/litre. The presence of TBA could have resulted from direct volatilization during herbicide application or wind erosion of soil particles from treated areas (Buser, 1990).

The groundwater situation in different countries was surveyed by the French Ministry of Agriculture and Fisheries. In Germany and Sweden, 22 out of 3204 samples and 6 out of 230 samples were positive for TBA (above 0.1 µg/litre), respectively (Dabène, 1993).

Food

In the USA, a large survey of pesticide residues on food products was carried out from 1989 to 1991: 6970 samples (80% domestic and 20% foreign) were taken from 81 varieties of vegetables and fruit. No TBA residues were found at a detection limit of 0.5 mg/kg (Schattenberg, 1992).

TBA application rates used on most annual crops are in the range of 0.4–2.0 kg/ha; at this concentration, no measurable residues of TBA (0.01–0.1 mg/kg) were observed among harvest products. Residues were found only in young maize grown for silage (maximum 0.1 mg/kg); this maize was given experimentally to cattle and poultry, and no measurable residues were detected in meat, eggs, or milk (Green, 1991; Anonymous, 1995).

Estimated total exposure and relative contribution of drinking-water

At a concentration of 0.2 µg/litre in drinking-water, the daily intake of TBA would be 0.4 µg. Data are insufficient to allow the relative contribution of drinking-water to total intake to be determined.

KINETICS AND METABOLISM IN LABORATORY ANIMALS AND HUMANS

^{14}C-labelled TBA administered orally to rats is quickly absorbed, completely metabolized, and excreted via the urine and faeces with a half-life of 16–17 hours (Green, 1991). After a single oral dose, at least 60% was absorbed from the gastrointestinal tract and 75% was excreted within the first 24 hours. After 168 hours, 99% of TBA was excreted. About 46%, 14%, and 2% of the administered dose was eliminated in bile, urine, and faeces, respectively (Werner, 1996). Seven days after a single oral administration of TBA at 0.5 mg/kg of body weight, tissue residues were below 0.007 mg of TBA equivalents per kg, except in kidneys (0.02–0.05 mg/kg), liver (0.01 mg/kg), and blood (0.01 mg/kg) (Green, 1991).

Following dermal application to rats, 30% of the applied dose was found in urine and faeces (Werner, 1996).

A metabolic study in the cow using ^{14}C-labelled TBA at a dose of 1 mg/kg of body weight per day for 10 days showed that almost 100% of the radioactivity was excreted in urine and faeces. About 0.1% was detected in milk (Werner, 1996).

The main metabolic degradation of TBA occurs through oxidative de-ethylation, oxidation of one methyl group of the *tert*-butyl group, and subsequent conjugation of the alcohol with glucuronic acid. The alcohol may be further oxidized into carboxylic acid. Minor pathways have been described with the formation of sulfate esters of the alcohol derivative, dechlorination via glutathione with subsequent formation of mercapturic acid derivatives, and the formation of 2-hydroxytriazine metabolites (Werner, 1996).

EFFECTS ON EXPERIMENTAL ANIMALS AND *IN VITRO* TEST SYSTEMS

Acute exposure
Acute exposure (Werner, 1996)

The oral LD_{50} for TBA in mice was 7700 mg/kg of body weight.
Five male and five female Tif/RAI f rats received TBA at 2000 mg/kg of body weight. One female died on day 2 after administration. All surviving animals recovered within 6–7 days. In a second rat study, TBA was administered to 20 male and 20 female OFA.SD rats at dose levels of 1000, 1590, or 2510 mg/kg of body weight. Fifteen out of 40 animals (3, 6, and 6 at the low, middle, and high doses, respectively) died within 3 days. The LD_{50} was found to be 1590 mg/kg of body weight. Clinical observation revealed piloerection, prostration, and diarrhoea. The main necropsy findings were marked congestion of the lung and autolysis of the alimentary canal.

In rats, the dermal LD_{50} was above 2000 mg/kg of body weight, and the acute inhalation LC_{50} was above 5300 mg/m^3.

Short-term exposure *(Werner, 1996)*

TBA was fed to groups of 10 male and 10 female rats in the diet at dose levels of 0, 6, 30, 100, or 300 mg/kg of feed for 90 days. A treatment-related decrease in body-weight gain was observed in both sexes at dose levels of 100 and 300 mg/kg of feed. Changes in haematology and clinical chemistry parameters were observed without macroscopic or histopathological modifications; most were reversible during a 4-week recovery period given to the 300 mg/kg of feed group. The NOAEL was 30 mg/kg of feed, equal to 2.1 mg/kg of body weight per day.

In a 1984 oral toxicity study, groups of five male and five female New Zealand white rabbits were administered TBA at dose levels of 0, 5, 50, or 500 mg/kg of body weight per day for 28 days on a 5 days/week basis. Because of high mortality, dose levels were reduced to 0, 5, 20, and 100 mg/kg of body weight per day 3 days after the start of treatment. Marked toxicity was still observed, with high mortality and body-weight loss at 500/100 mg/kg of body weight per day. Three males and two females at the top dose level were kept for a 14-day recovery period. Signs of systemic toxicity were noted at all dose levels, including sedation, dyspnoea, diarrhoea, and tremors. A decrease in food consumption was observed at all dose levels. Haematology revealed a decrease in red blood cell parameters at 500/100 mg/kg of body weight per day. A number of organ weights decreased, including liver (at all dose levels) and heart, thymus, and testes at 20 and 500/100 mg/kg of body weight per day. Organ-weight changes were mostly reversible during the 2-week recovery period. Congestion and haemorrhage of the respiratory and digestive tracts, thymus, bone marrow, spleen, mediastinum, and lymph nodes were observed in rabbits found dead. Additionally, atrophy of thymus, lymph nodes, and spleen as well as immature testes were observed in rabbits at the highest dose. It was not possible to determine the NOAEL in this study.

In a 1987 oral toxicity study, groups of five male and five female New Zealand white rabbits were fed TBA at dose levels of 0, 0.05, 0.5, or 5 mg/kg of body weight per day for 28 days. No deaths occurred at the highest dose level, and no significant indications of any target organ toxicity were observed. The NOAEL in this study was 5 mg/kg of body weight per day, the highest dose tested. The difference in results between the 1984 and 1987 studies may be due to the different origins of the rabbits used.

Administration of TBA to dogs in the diet for 1 year at dose levels of 0, 10, 50, 250, or 500 mg/kg of feed resulted in decreased body-weight gain and food consumption at dose levels =50 mg/kg of feed and slight reduction in some red blood cell parameters in females at dose

levels =250 mg/kg of feed, but no specific signs of toxicity were noted. The NOAEL in this study was 10 mg/kg of feed, equal to 0.4 mg/kg of body weight per day.

Long-term exposure and carcinogenicity *(Werner, 1996)*

Administration of TBA to mice (50 per sex per dose) in the diet at dose levels of 0, 30, 150, or 750 mg/kg of feed for 2 years resulted in an increased survival of treated males, which was statistically significant only at 30 and 750 mg/kg of feed. A moderate decrease in body-weight gain and food consumption was observed in both sexes at 750 mg/kg of feed. No treatment-related neoplastic findings were observed. The NOAEL in this study was 150 mg/kg of feed, equal to 15 mg/kg of body weight per day.

In two lifetime studies, Tif/RAI f rats (1120 rats in total; number per sex per dose not specified) were fed TBA at dietary levels of 0, 30, 150, or 750 mg/kg of feed and 0, 6, or 30 mg/kg of feed. The second study was started 6 months after the beginning of the first one, after it became clear that, because of the effects on body weight, 30 mg/kg of feed was not the NOAEL.

In the first study, an increased survival of high-dose males (60% survival compared with 22% in the control group) and a dose-dependent decrease in body-weight gain in both sexes were noted. Haematology revealed effects on red blood cell parameters in mid- and high-dose females. Pathology revealed an increased incidence of non-neoplastic lesions in the liver, lung, thyroid, and testis. An increased incidence of mammary gland carcinomas was observed in high-dose females, but the number of fibroadenomas was simultaneously decreased at this dose. The incidence of mammary gland tumours was within the historical control range, and the number of females bearing mammary gland tumours was identical in control and high-dose females. The increased incidence of Leydig cell tumours in high-dose males seems to be related to the increased "time of risk," as most of the tumours were observed in old rats after 2 years or at terminal sacrifice. The NOAEL was not determined in this study.

In the second study, the only effect noted was a slight decrease in body-weight gain at 30 mg/kg of feed (equal to 1 mg/kg of body weight per day) in both sexes. The NOAEL in this study was 6 mg/kg of feed, equal to 0.22 mg/kg of body weight per day.

Reproductive and developmental toxicity *(Werner, 1996)*

Groups of 24 pregnant Tif/RAI f SPF rats were administered TBA by gastric intubation at dose levels of 0, 1, 5, or 30 mg/kg of body weight per day from days 6 to 15 of gestation. Decreased body-weight gain and food consumption were noted at the high dose level. No effects on reproductive parameters were noted. As indicated by delayed or absent ossification of phalanges, fetal development was slightly delayed at 30 mg/kg of body weight per day. No treatment-related external, visceral, or skeletal malformations or abnormalities were recorded. The maternal and fetal NOAELs were both determined to be 5 mg/kg of body weight per day.

Groups of 16–22 New Zealand white rabbits were treated with TBA at dose levels of 0, 0.5, 1.5, or 4.5 mg/kg of body weight per day by oral intubation during days 7–19 of gestation. No significant effects on reproductive parameters were observed.

Groups of 21 pregnant Russian rabbits (Chbb:HM) were administered TBA at dose levels of 0, 0.5, 1.5, or 5 mg/kg of body weight per day by oral intubation during days 7–19 of gestation. Increased body-weight loss was observed at the highest dose during the treatment period and was later compensated by a body-weight gain during the post-treatment period. A dose-related decrease in food consumption was observed in the mid- and high-dose groups during the treatment period. No effects on reproductive or fetal parameters were observed. Based on the effect on food consumption at 1.5 mg/kg of body weight per day, the maternal

NOAEL was 0.5 mg/kg of body weight per day. In the absence of any treatment-related fetal findings, the fetal NOAEL was 5 mg/kg of body weight per day, the highest dose tested.

In a two-generation study, the effects on the reproductive performance of Sprague-Dawley rats were assessed by feeding them TBA in the diet at dose levels of 0, 6, 60, or 300 mg/kg of feed. The F_0 generation consisted of groups of 32 male and 32 female rats. In the F_1 generation, groups of 28 male and 28 female rats were used. Animals of the F_0 generation were approximately 6 weeks old at the beginning of treatment; treatment was maintained for 70 days prior to mating (age: 16 weeks). On day 21 post-partum, 28 male and 28 female pups (F_1) were retained. F_0 males and females as well as surplus F_1 pups were sacrificed. The selected F_1 animals were maintained on their respective diets and mated at the age of 16 weeks. All F_{2a} pups were sacrificed at day 21 post-partum. Apparently non-pregnant F_1 females were mated for the second time with males that had been successful at the first mating. The F_{2b} pups as well as the F_1 males and females were sacrificed on day 4 post-partum. At the high dose level, decreased body-weight gain, decreased food and water consumption, slightly higher number of infertile pairings, slightly higher pup mortality, and retarded pup growth were observed. At the mid-dose level, parental (decreased body-weight gain and food consumption) but no reproductive effects were observed. The NOAEL for parental effects was 6 mg/kg of feed, equal to 0.4 mg/kg of body weight per day; the NOAEL for reproductive effects was 60 mg/kg of feed, equal to 21.3 mg/kg of body weight per day.

Mutagenicity *(Werner, 1996)*

No increased incidence of back mutations was observed in Salmonella typhimurium strains TA98, TA100, TA1535, or TA1537 with or without metabolic activation at TBA concentrations up to 1 mg per plate. In a second study with the same strains plus Escherichia coli WP2 uvrA at TBA concentrations up to 5 mg per plate, no mutagenic activity could be detected with or without metabolic activation.

With mouse lymphoma cells at TBA concentrations up to 1 mg/ml, no increase in mutant frequency was observed (with or without metabolic activation). In a test with Chinese hamster V79 cells, no induction of gene mutations resulted with TBA concentrations up to 380 mg/ml, with or without metabolic activation. In another test with Chinese hamster cells, no increased incidence of specific aberrations was observed at TBA concentrations up to 380 mg/ml, with or without metabolic activation.

With human lymphocytes, no increased number of specific chromosomal aberrations was observed up to a TBA concentration of 500 mg/ml in experiments with or without metabolic activation. With human fibroblasts or rat hepatocytes, no induction of unscheduled DNA synthesis was observed at concentrations of TBA up to 125 and 1000 mg/ml, respectively.

In in vivo tests, doses of TBA up to 3000 mg/kg of body weight were administered to Chinese hamsters twice within 24 hours. No increased incidence of chromosomal aberrations was observed. A micronucleus test in mice with dose levels up to 5000 mg/kg of body weight gave the same results.

EFFECTS ON HUMANS

There are no data available on the effects of TBA on humans.

GUIDELINE VALUE

There is no evidence that TBA is carcinogenic or mutagenic. A TDI approach was therefore used in the derivation of a guideline value for TBA in drinking-water.

A NOAEL of 0.22 mg/kg of body weight per day was identified in a 2-year toxicity and carcinogenicity study in rats for decreased body-weight gain at the next higher dose of 1 mg/kg of body weight per day. Using an uncertainty factor of 100 (for inter- and intraspecies variation), the TDI is 2.2 µg/kg of body weight.

Assuming a 60-kg person consumes 2 litres of drinking-water per day and allocating 10% of the TDI to drinking-water, a guideline value of 7 µg/litre (rounded figure) can be calculated for TBA in drinking-water.

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