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UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

010091

MAR 1 8 1993

OFFICE OF
PREVENTION, PESTICIDES
AND TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: Reregistration Eligibility Document on OBPA (Oxybisphenoxarsine)

FROM: Flora Chow, Acting Head F. Chow 8 Mar 93
Reregistration Section
Chemical Coordination Branch
Health Effects Division (H7509C)

THROUGH: Esther Saito, Chief E. Saito 3-8-93
Chemical Coordination Branch
Health Effects Division (H7509C)

Penelope Penner-Crisp, Ph.D., Director
Health Effects Division (H7509C) 3/12/93

TO: Lois Rossi, Chief
Reregistration Branch
Special Review and Reregistration Branch (H7508W)

The Health Effects Division has completed the Human Health Assessment for the OBPA Reregistration Eligibility Document. OBPA is an antimicrobial pesticide used in plastics and caulking. There are no tolerances for residues of OBPA in or on food/feed or on processed commodities.

The toxicology and human exposure database on OBPA is adequate and will support reregistration eligibility. Human health risks from exposure to OBPA are expected to be minimal because of the low potential for exposure to workers, consumers or residential occupants. Protective clothing and eyewear are recommended when mixing/loading and handling OBPA during the manufacture of plastics or technical and formulated products containing OBPA.

Attachments

cc: L. Kutney
P. McLaughlin
J. Evans

I. PRODUCT CHEMISTRY

IDENTIFICATION OF THE ACTIVE INGREDIENT

OBPA (10,10'-Oxybisphenoxarsine) is an antimicrobial and antifungal pesticide used in plastics and caulking.

Case Number: 0044
Chemical Number: 012601

PRODUCT CHEMISTRY

The Product Chemistry portion of this RED will be provided by the Registration Division. There are no registered uses for OBPA in or on food or feed.

II. HUMAN HEALTH ASSESSMENT

TOXICOLOGY ASSESSMENT

The toxicological database on OBPA is adequate and will support reregistration.

Acute Toxicity

Several acute toxicity studies of each type have been done with OBPA, some of which do not provide complete information. The more completely reported studies are summarized here. In general, the acute oral toxicity studies found LD_{50}'s from about 15 to 40 mg/kg. A more complete report of a test in rats with technical OBPA had an LD_{50} of 36.9 mg/kg in males and of 31.6 in females (Anspach, 1977, GS044002). The toxic effects included congested adrenals, kidneys, and lungs, irritated digestive systems, and depleted fat stores. These results fall into toxicity category I.

The results found in acute dermal toxicity studies varied widely. A study with rabbits using technical OBPA found an LD_{50} of 414 mg/kg (Anspach, 1977, GS044002). One that used technical OBPA on rats reported an LD_{50} of 121 mg/kg (Litton Bionetics, 1978, GS044039). The Agency places OBPA in Toxicity category I for dermal toxicity.

An acute inhalation toxicity study with guinea pigs estimated the LC_{50} at 1279 mg/L, results which place this compound in toxicity category IV for inhalation (Ballantyne, 1978, 05015857).

Two primary eye irritation studies in rabbits with technical OBPA found corneal opacity, conjunctivitis, and iritis (Anspach, 1977, 00013643; GS044002). In one study the effects lasted through 72 hours. The Agency considers this material to be in toxicity category I for eye irritation.

Results in different primary skin irritation studies were quite varied. One study with technical OBPA applied to intact and abraded skin of rabbits found slight to severe edema and peripheral or spotted erythema, results which fall in toxicity category II (Anspach, 1977, 0013643). Another study applied technical material to intact and abraded rabbit skin for 24 hours, to find the results were in toxicity category III (Anspach, 1977, GS044002). The Agency has considered that OBPA is in category II for dermal irritation.

TABLE OF ACUTE TOXICITY DATA

TEST	RESULT (mg/kg)	TOXICITY CATEGORY
Acute Oral LD_{50} (rat)	15-40	I
Acute Dermal LD_{50} (rat, rabbit)	121-414	I
Acute Inhalation LD_{50} (guinea pig)	1279	IV
Eye Irritation (rabbit)	Corneal Opacity	I
Skin Irritation (rabbit)	Varies (edema, erythema)	II

Subchronic Toxicity

In a 35-day feeding study, 10 rats/sex/dose were given 0, 1, 10, 30, 100, or 300 ppm of OBPA in the diet (the percent active ingredient was not stated; Franz and Shrader 1959, 00024936; Oxen 1959, 00024940). Generally, animals at the two higher doses had retarded growth, higher liver weights, increased testes weights, proliferation of the portal bile duct, and accumulation of arsenic in liver and kidneys. The NOEL was 10 ppm (approximately 0.5 mg/kg/day).

A rat feeding study lasting 92 days gave doses of 0, 0.03, 0.1, 0.3, 1.0 or 3.0 mg/kg/day to 10 rats/sex/dose (unspecified percent active ingredient; McCollister et al. 1969, GS044042).

The high dose group had retarded growth as well as lack of fat in the mesentery; there were microscopic liver lesions and inflammatory cellular infiltrates in the periportal area with bile duct hyperplasia. Arsenic accumulated in the liver and kidney at all doses and in the fat and hair in some of the higher doses. The NOEL was 1.0 mg/kg/day.

In an inhalation study, both rats and guinea pigs were exposed to 1-2 mg/M^3 of technical OBPA (percent not stated) during five days per week for 30 days (IPC 1957, 00013591; Ballantyne 1978, 05015857). Half of the animals were sacrificed 48 hours after the last exposure and the others were kept without further exposure for four months. The rats and guinea pigs killed at 48 hours had mild to moderate pulmonary congestion and hemorrhaging; only the rats had hepatic involvement. The animals killed four months later showed no effects of OBPA.

Metabolism

Metabolism studies in which OBPA was applied to the skin generally found arsenic accumulation in the liver and kidney with removal from the body through both feces and urine (Olson et al. 1959, 00026092; Dow 1964, 00024935; Ballantyne 1978, 05015857). There are indications that the arsenic is cleared from the body after two weeks.

Reproduction and Developmental Toxicity

An investigation of the effect of OBPA on fetal development and toxicity used dermal applications at 0.3, 3.0 or 30.0 mg/kg on pregnant rats (Beliles and Makris 1978, GS0440007). However, the animals also ingested the material put on their skin and the actual doses are uncertain. All high-dose rats and one mid-dose rat died during the test. There was no evidence of compound-related teratogenicity or inhibition of fetal development from the surviving animals. There was some evidence of fetal toxicity in the mid-dose group and the NOEL was estimated at 0.3 mg/kg.

Mutagenicity

A mouse lymphoma test was considered negative for mutagenicity with and without activation (Brusick and Weir 1976, 00013644). A mutagenicity study in S. cerevisiae and S. typhimurium with and without activation did not show mutagenicity (Brusick and Weir 1976, 00013647). An investigation of the metabolites of OBPA used the urine of rats treated with OBPA in the method of Durston and Ames. There was no indication of mutagenic activity with urine from treated or untreated rats (Beliles and Makris 1978, GS044007).

EXPOSURE ASSESSMENT

Dietary Exposure

OBPA is not used on food, feed or processed commodities; dietary exposure to OBPA is not expected.

Occupational and Residential Exposure

OBPA is an antimicrobial and antifungal pesticide used to treat polyvinylchloride (PVC), polyurethane, and ethylene-vinylacetate copolymer plastics. These plastics are fabricated into shower curtains, floor coverings, wall coverings, ditch liners, coated fabrics (including mattresses), vinyl molding, marine upholstery, automotive vinyl trim, tarpaulins, awnings, gaskets (automotive, refrigerator, dishwasher), weather stripping, caulking, and swimming pool liners. Plastic products intended for indoor use contain up to 0.06% OBPA and plastic products intended for outdoor use contain up to 0.05% OBPA.

OBPA formulations range in concentrations from 1 to 5%. The OBPA formulations are either liquids or non-dusting powder forms utilizing a resin carrier.

Occupational and residential exposure data were not required in the 1981 Registration Standard for OBPA based on the following reasons:

1) direct occupational exposure to OBPA is mitigated by the use of closed systems, when producing OBPA technical and OBPA formulations, or when producing plastic products containing OBPA;

2) indirect human exposure to OBPA in treated plastics is low due to the low percent of OBPA added to plastic coupled with the slow release of small amounts of OBPA from the plastic matrix; and

3) that no additional uses are proposed that may result in significant increases in human exposure.

(Inhalation exposure from OBPA-treated products is negligible since it has a low vapor pressure.)

Based on the low potential for exposure to OBPA, exposure data are not required to support the reregistration of the current uses of OBPA, provided that (1) manufacturing is under closed systems, and (2) appropriate Personal Protective Equipment (PPE) is used.

RISK CHARACTERIZATION

Dietary

There are no registered food or feed uses of OBPA, and therefore, dietary exposure or risk to the OBPA is not expected.

Occupational and Residential

The potential for occupational exposure to OBPA is minimal provided that OBPA is used in a closed system and that appropriate protective eyewear and gloves are used. Therefore, the potential risk for acute or chronic toxicity from exposure to OBPA is also likely to be minimal.

In 1991, the Agency received a research proposal concerning the potential linkage between OBPA-treated mattresses and Sudden Infant Death Syndrome (SIDS). The proposal was submitted by Dr. Julius Goldberg of the Loyola University Medical Center in Chicago, Illinois. The hypothesis was that toxic gases may be produced by microfungi which attack PVC plastic materials. Some of these fungi can break down the arsenates, in the PVC, and produce trimethyl arsine (Allsop and Hawksworth, 1992). However, recent tests appear to show no correlation between SIDS and the use of plastics treated with OBPA.

BIBLIOGRAPHY

TOXICOLOGICAL BIBLIOGRAPHY

GS044002 Anspach, P.S. (1977) Acute Toxicity and Irritation Studies of OBPA (95.6%). (Unpublished study prepared by Hill Top Testing Services, Inc.)

00013643 Anspach, P.S. (1977) Acute Toxicity and Irritation Studies of 99.9% 10,10'-Oxybisphenoxarsine: 76-990-21. (Unpublished study received Feb. 16, 1977 under 2829-115; prepared by International Bio-Research, Inc., submitted by Ventron Corp., Beverly, Mass.; CDL:228087-B)

05015857 Ballantyne, B. (1978) The comparative short-term mammalian toxicology of phenarsazine oxide and phenoxarsine oxide. Toxicology 10(4):341-461.

GS0440007 Beliles, R.P.; Makris, S.L. (1978) Teratology Study in Rats. 10,10'-Oxybisphenoxarsine: LBI Project No. 20816. (Unpublished study prepared by Litton Bionetics, Inc.)

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00026092 Olson, K.J.; Nunemaker, R.B.; Shrader, S.A.; et al. (1959) Skin Contact Absorption (Unpublished study received Nov. 2, 1964; prepared by Biochemical Research Laboratory with Dow Chemical Co., CDL:121914-F)

00024940 Oxen, F. (1959) Summary of Pathology on Male & Female Rats Fed 10,10'-Oxybisphenoxarsine in the Diet for 30 Days. (Unpublished study received Sep.2, 1965; CDL:121914-G)

OCCUPATIONAL AND RESIDENTIAL EXPOSURE BIBLIOGRAPHY

Kelley,J., Allsopp, D., and Hawksworth, D.L. 1992. Sudden Infant Death Syndrome (SIDS) and the Toxic Gas Hypothesis: Microbiological Studies of Cot Mattresses. Human and Experimental Toxicology 11, 347-355.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
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OFFICE OF
PESTICIDES AND TOXIC
SUBSTANCES

MEMORANDUM

Subject: Toxicology Review for the Reregistration Eligibility Document on OBPA or Oxybisphenoxarsine

To: Esther Saito, Acting Chief
Chemical Coordination Branch
Health Effects Division (H7509C)

From: Patricia McLaughlin, Ph.D. P M^cLaughlin 1/28/93
Toxicology Branch II, HED (H7509C)

Thru: Elizabeth Doyle, Ph.D., Head E.A. Doyle 1/28/93
Section IV, Toxicology Branch II, HED (H7509C)

Marcia van Gemert, Ph.D., Chief M. van Gemert 2/2/93
Toxicology Branch II, HED (H7509C)

Chemical: 10,10'-Oxybisphenoxarsine
Case/chemical number: 0044/12601

S429653/D185099

Products: antimicrobial in plastics and caulking

Considerations: The standard on OBPA, published in October 1981, utilized an assemblage of studies and information, not all of which would stand up well to detailed scrutiny today. However, considering the uses and situation involved, the general aspects of safety can be viewed, from the standpoint of the total assemblage, to be sufficient to move forward with regulatory processes.

1. Toxicology Data Base

The toxicological data base on OBPA is adequate and will support reregistration eligibility.

a. Acute Toxicity

Several acute toxicity studies of each type have been done with OBPA, some of which do not provide complete information. The more completely reported studies are summarized here. In general, the acute oral toxicity studies found LD_{50}'s from about 15 to 40 mg/kg. A more complete report of a test in rats with technical OBPA had an LD_{50} of 36.9 mg/kg in males and of 31.6 in females (Anspach, 1977, GS044002). The toxic effects included congested adrenals, kidneys, and lungs, irritated digestive systems, and depleted fat stores. These results fall into toxicity category I.

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erythema, which falls in toxicity category II (Anspach, 1977, 0013643). Another study applied technical material to intact and abraded rabbit skin for 24 hours, to find the results were in toxicity category III (Anspach, 1977, GS044002). The agency has considered that OBPA is in category II for dermal irritation.

b. Subchronic Toxicity

In a 35-day feeding study, 10 rats/sex/dose were given 0, 1, 10, 30, 100, or 300 ppm of OBPA in the diet (the percent active ingredient was not stated; Franz and Shrader 1959, 00024936; Oxen 1959, 00024940). Generally, animals at the two higher doses had retarded growth, higher liver weights, increased testes weights, proliferation of the portal bile duct, and accumulation of arsenic in liver and kidneys. The NOEL was 10 ppm.

A rat feeding study lasting 92 days gave doses of 0, 0.03, 0.1, 0.3, 1.0 or 3.0 mg/kg/day to 10 rats/sex/dose (unspecified percent active ingredient; McCollister et al. 1969, GS044042). The high dose group had retarded growth as well as lack of fat in the mesentary; there were microscopic liver lesions and inflammatory cellular infiltrates in the periportal area with bile duct hyperplasia. Arsenic accumulated in the liver and kidney at all doses and in the fat and hair in some of the higher doses. The NOEL was 1.0 mg/kg/day.

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c. Metabolism

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Metabolism studies in which OBPA was applied to the skin generally found arsenic accumulation in the liver and kidney with removal from the body through both feces and urine (Olson et al. 1959, 00026092; Dow 1964, 00024935; Ballantyne 1978, 05015857). There are indications that the arsenic is cleared from the body after two weeks.

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e. Mutagenicity

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FEB 8 1993

OFFICE OF
PREVENTION, PESTICIDES AND
TOXIC SUBSTANCES

MEMORANDUM

SUBJECT: OCCUPATIONAL AND RESIDENTIAL EXPOSURE ASSESSMENT AND RECOMMENDATIONS FOR THE REREGISTRATION ELIGIBILITY DOCUMENT FOR OXYBISPHENOXARSINE (OBPA)

FROM: Jeff Evans, Biologist
Reregistration Section
Occupational and Residential Exposure Branch
Health Effects Division (H7509C)

TO: Linda Kutney, Chemical Coordinator
Chemical Coordination Branch
Health Effects Division (H7509C)

THRU: Alan P. Nielsen, Section Head
Reregistration Section

Larry C Dorsey, Chief
Occupational and Residential Exposure Branch
Health Effects Division (H7509C)

Please find the OREB review of ....

DP Barcode: D185098

Pesticide Chemical Codes: 012601

EPA Reg. No.: N/A

EPA MRID No.: N/A

Review Time: 2 days

PHED: No

This memorandum presents the OREB science chapter review for the oxybisphenoxarsine (OBPA) Reregistration Eligibility Document (RED). Occupational and residential exposure data requirements to support the reregistration of OBPA are discussed in this chapter. Precautionary label language recommendations regarding personal protective equipment are also addressed.

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Occupational and Residential Exposure

OBPA is an antimicrobial pesticide used to control bacteria and fungus that may attack polyvinylchloride (PVC), polyurethane, and ethylene-vinylacetate copolymer, plastics. These plastics are fabricated into shower curtains, floor coverings, wall coverings, ditch liners, coated fabrics (including mattresses), vinyl molding, marine upholstery, automotive vinyl trim, tarpaulins, awnings, gaskets (automotive, refrigerator, dishwasher), weather stripping, and swimming pool liners.

OBPA formulations range in concentrations of 1 to 5% with the remaining percentages consisting of various plasticizer components such as epoxidized soybean oil, diisodecyl phthalate, and epoxy plasticizer. These formulations are incorporated into plastic compounds by substituting the OBPA formulation with an equal amount of plasticizer normally used to make the manufactured plastic product. Plastic products intended for indoor use contain up to 0.06% OBPA and plastic products intended for outdoor use contain up to 0.05% OBPA. The OBPA formulations are either liquids or non-dusting powder forms utilizing a resin carrier. They are applied using "closed systems" since OBPA is an eye and skin irritant.

The majority of toxicity data used to prepare the OBPA Registration Standard came from studies available in the open literature. In that standard, it was reported that the toxicity of formulations containing OBPA varies according to the components within a given formulation and that technical OBPA possesses a high order of acute and oral toxicity. Available toxicity information in HED's Toxicity Oneliners indicate formulations containing 5% OBPA are in Toxicity category III for acute oral and dermal toxicity and in toxicity category I for primary eye and dermal irritation. In the standard, it was also determined that formulated products were in Toxicity Category IV for acute inhalation and that inhalation exposure as a result of exposure to OBPA treated products would be negligible since it has a low vapor pressure (less than 1×10^{-6} mm Hg). However, since OBPA is an arsenical compound, arsenic (a known carcinogen) is the toxic constituent of concern. Formulations containing 5% OBPA are reported as containing 0.6 to 1.5% elemental arsenic with water soluble arsenic (as elemental) less than 0.01%.

In the standard, it was determined that there are two routes of exposure to OBPA, direct and indirect. Direct exposure is the potential human exposure that may occur during the production of OBPA or when adding OBPA during the plastic manufacturing process. Indirect exposure is the potential human exposure to plastic products containing OBPA. Indirect exposure routes for the purposes of occupational and/or residential exposure include dermal exposure and inhalation exposure.

Occupational and residential exposure data were not required in the 1981 Registration Standard for OBPA based on the following reasons:

1) direct occupational exposure to OBPA is mitigated by the use of "closed systems", when producing OBPA technical and OBPA formulations, or when producing plastic products containing OBPA ;

2) indirect human exposure to OBPA in treated plastics is low due to the low percent of OBPA added to plastic coupled with the slow release of small amounts of OBPA from the plastic matrix;

3) that no additional uses are proposed that may result in significant increases in human exposure.

OREB was notified by SRRD via the June 5, 1991 Data Call-In package that EPA received a research proposal concerning the potential linkage between OBPA treated mattresses and Sudden Infant Death Syndrome (SIDS). The proposal was submitted by Dr. Julius Goldberg of the Loyola University Medical Center in Chicago, Illinois. There is a hypothesis that toxic gases may be produced by microfungi which attack PVC plastic materials. Some of these fungi can break down the arsenates, in the PVC, and produce trimethyl arsine[1]. According to Jerome Blondell/OREB, recent tests show no correlation between SIDS and the use of OBPA or any other fabric preservative.

OREB recommends that based on the low potential for either direct or indirect exposure to OBPA, occupational and residential exposure data are not required to support the reregistration of the current uses of OBPA providing that:

the use of "closed systems" during the manufacturing of OBPA and when using OBPA to produce plastic products be continued;

the use of appropriate Personal Protective Equipment (PPE) including protective eyewear and gloves as specified in 40 CFR §156.10 when mixing/loading and handling OBPA during the manufacture of plastics or technical and formulated products containing OBPA.

1/ Kelley,J., Allsopp, D., and Hawksworth, D.L. 1992. Sudden Infant Death Syndrome (SIDS) and the Toxic Gas Hypothesis: Microbiological Studies of Cot Mattresses. Human and Experimental Toxicology 11, 347-355.

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cc: J. Evans, OREB
J. Blondell, OREB
C. Childress, SRRD
Correspondence File
Chemical File (012601)

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Chemical:	**10,10'-Oxybisphenoxarsine**
PC Code:	**012601**
HED File Code	**14000 Risk Reviews**
Memo Date:	**03/18/93**
File ID:	**TX010091**
Accession Number:	**412-02-0011**

HED Records Reference Center
02/21/2002