requirements of section 3 of Executive Order 12291.

Unfunded Mandates Reform Act: Title II of the Unfunded Mandates Reform Act of 1995 (UMRA), Pub. L. 104–4, establishes requirements for Federal agencies to assess the effects of their regulatory actions on State, local and tribal governments and the private sector. Under section 202 of the UMRA, the EPA must prepare a written statement, including a cost benefit analysis, for proposed and final rules with ''federal mandates'' that may result in expenditures to State, local, and tribal governments, in the aggregate, or to the private sector of $100 million or more in any one year.

Today's document contains no Federal mandates (under the regulatory provisions of Title of the UMRA) for State, local, or tribal governments or the private sector. Today's document would merely acknowledge the adequacy of a portion of an existing State program. The EPA has determined that this document would not contain any Federal mandate that may result in expenditures of $100 million or more for state, local, and tribal governments, in the aggregate or the private sector in any one year. Therefore, today's document is not subject to the requirements of section 202 of the UMRA.

Certification Under the Regulatory Flexibility Act: Pursuant to the provisions of 5 U.S.C. 605(b), I hereby certify that this approval will not have a significant economic impact on a substantial number of small entities. It does not impose any new burdens on small entities. This rule, therefore, does not require a regulatory flexibility analysis.

Authority: This notice is issued under the authority of section 4005 of the Solid Waste Disposal Act as amended; 42 U.S.C. 6946.

Dated: August 26, 1998.

Jerry Clifford,

Deputy Regional Administrator, Region 6.

[FR Doc. 98–24738 Filed 9–15–98; 8:45 am]

BILLING CODE 6560–50–P

ENVIRONMENTAL PROTECTION AGENCY

[OPP–30443A; FRL–6029–2]

LidoChem Inc.; Approval of a Pesticide Product Registration

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces Agency approval an application to register the pesticide product eKsPunge, containing an active ingredient not included in any previously registered product pursuant to the provisions of section 3(c)(5) of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), as amended.

FOR FURTHER INFORMATION CONTACT: Rita Kumar, Regulatory Action Leader, Biopesticides and Pollution Prevention Division (7511C), Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460, Office location/telephone number and e-mail address: Rm. 902W5, CM #2, 1921 Jefferson Davis Hwy, Arlington, VA, 703–308–8291; e-mail: kumar.rita@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

Electronic Availability: Electronic copies of this document and the Fact Sheet are available from the EPA home page at the **Federal Register**-Environmental Documents entry for this document under ''Laws and Regulations'' (http://www.epa.gov/fedrgstr/).

EPA issued a notice, published in the **Federal Register** of December 9, 1997 (62 FR 64831) (FRL–5756–3), which announced that LidoChem Inc., 20 Village Court, Hazlet, NJ 07730, had submitted an application to register the pesticide product eKsPunge (EPA File Symbol 70644–R), containing the new active ingredient monopotassium phosphate (KH_2PO_4) at 100%, an active ingredient not included in any previously registered product.

The active ingredient for the registered product was amended to read ''Potassium Dihydrogen Phosphate'' commonly known as monopotassium phosphate.

The application was approved on August 12, 1998, as eKsPunge for the control of powdery mildew on apples, cherries, cucumbers, grapes, mangoes, melons, nectarines, peaches, peppers, plums, summer/winter squash, tomatoes, watermelons, and roses (EPA Registration Number 70644–1).

The Agency has considered all required data on risks associated with the proposed use of potassium dihydrogen phosphate, and information on social, economic, and environmental benefits to be derived from use. Specifically, the Agency has considered the nature of the pesticide and its pattern of use, application methods and rates, and level and extent of potential exposure. Based on these reviews, the Agency was able to make basic health safety determinations which show that use of potassium dihydrogen phosphate when used in accordance with widespread and commonly recognized practice, will not generally cause unreasonable adverse effects to the environment.

More detailed information on this registration is contained in an EPA Pesticide Fact Sheet on potassium dihydrogen phosphate.

A copy of the fact sheet, which provides a summary description of the pesticides, use patterns and formulations, science findings, and the Agency's regulatory position and rationale, may be obtained from the National Technical Information Service (NTIS), 5285 Port Royal Road, Springfield, VA 22161.

In accordance with section 3(c)(2) of FIFRA, a copy of the approved label, the list of data references, the data and other scientific information used to support registration, except for material specifically protected by section 10 of FIFRA, are available for public inspection in the Public Information and Records Integrity Branch, Information Resources and Services Division (7502C), Office of Pesticide Programs, Environmental Protection Agency, Rm. 119, CM #2, Arlington, VA 22202 (703–305–5805). Requests for data must be made in accordance with the provisions of the Freedom of Information Act and must be addressed to the Freedom of Information Office (A-101), 401 M St., SW., Washington, D.C. 20460. Such requests should: (1) Identify the product name and registration number and (2) specify the data or information desired.

Authority: 7 U.S.C. 136.

List of Subjects

Environmental protection, Pesticides and pests, Product registration.

Dated: September 4, 1998.

Kathleen D. Knox,

Acting Director, Biopesticides and Pollution Prevention Division, Office of Pesticide Programs.

[FR Doc. 98–24842 Filed 9–15–98; 8:45 am]

BILLING CODE 6560–50–F

ENVIRONMENTAL PROTECTION AGENCY

[PF–830; FRL 6025–8]

Notice of Filing of Pesticide Petitions

AGENCY: Environmental Protection Agency (EPA).

ACTION: Notice.

SUMMARY: This notice announces the initial filing of pesticidepetitions proposing the establishment of regulations for residues of certain

pesticide chemicals in or on various food commodities.

DATES: Comments, identified by the docket control number PF–830, must be received on or before October 16, 1998.

ADDRESSES: By mail submit written comments to: Public Information and Records Integrity Branch (7502C), Information Resources and Services Division, Office of Pesticide Programs, Environmental Protection Agency, 401 M St., SW., Washington, DC 20460. In person bring comments to: Rm. 119, CM #2, 1921 Jefferson Davis Highway, Arlington, VA.

Comments and data may also be submitted electronically to: opp-docket@epamail.epa.gov. Follow the instructions under ''SUPPLEMENTARY INFORMATION.'' No confidential business information should be submitted through e-mail.

Information submitted as a comment concerning this document may be claimed confidential by marking any part or all of that information as Confidential Business Information (CBI). CBI should not be submitted through e-mail. Information marked as CBI will not be disclosed except in accordance with procedures set forth in 40 CFR part 2. A copy of the comment that does not contain CBI must be submitted for inclusion in the public record. Information not marked confidential may be disclosed publicly by EPA without prior notice. All written comments will be available for public inspection in Rm. 119 at the address given above, from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays.

FOR FURTHER INFORMATION CONTACT: The product manager listed in the table below:

Product Manager	Office location/telephone number	Address
Beth Edwards, ...	Rm. 216, CM #2, 703–305–5400; e-mail: edwards.beth@epamail.epa.gov.	1921 Jefferson Davis Hwy., Arlington, VA
Treva Alston, ...	Rm. 707B, CM #2, 703–308–8373; e-mail: alston.treva@epamail.epa.gov.	Do.

SUPPLEMENTARY INFORMATION: EPA has received pesticide petitions as follows proposing the establishment and/or amendment of regulations for residues of certain pesticide chemicals in or on various food commodities under section 408 of the Federal Food, Drug, and Comestic Act (FFDCA), 21 U.S.C. 346a. EPA has determined that these petitions contain data or information regarding the elements set forth in section 408(d)(2); however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

The official record for this notice of filing, as well as the public version, has been established for this notice of filing under docket control number PF–830 (including comments and data submitted electronically as described below). A public version of this record, including printed, paper versions of electronic comments, which does not include any information claimed as CBI, is available for inspection from 8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays. The official record is located at the address in ''ADDRESSES'' at the beginning of this document.

Electronic comments can be sent directly to EPA at:

opp-docket@epamail.epa.gov

Electronic comments must be submitted as an ASCII file avoiding the use of special characters and any form of encryption. Comments and data will also be accepted on disks in Wordperfect 5.1/6.1 or ASCII file format. All comments and data in electronic form must be identified by the docket control number PF–830 and appropriate petition number. Electronic comments on this notice may be filed online at many Federal Depository Libraries.

List of Subjects

Environmental protection, Agricultural commodities, Feed additives, Food additives, Pesticides and pests, Reporting and recordkeeping requirements.

Dated: September 2, 1998.

James Jones,

Director, Registration Division, Office of Pesticide Programs.

Summaries of Petitions

Petitioner summaries of the pesticide petitions are printed below as required by section 408(d)(3) of the FFDCA. The summaries of the petitions were prepared by the petitioners and represent the views of the petitioners. EPA is publishing the petition summaries verbatim with minor, non-substantive editorial changes. The petition summary announces the availability of a description of the analytical methods available to EPA for the detection and measurement of the pesticide chemical residues or an explanation of why no such method is needed.

1. Dow AgroSciences

PP 8F5002

EPA has received a pesticide petition (PP 8F5002) from Dow AgroSciences, 9330 Zionsville Road, Indianapolis, IN 46254 proposing pursuant to section 408(d) of the (FF DCA), 21 U.S.C. 346a(d), to amend 40 CFR part 180 by establishing a tolerance for residues of the insecticide spinosad in or on the raw agricultural commodities corn grain including field, sweet (K+CWHR), and pop at 0.02 part per million (ppm); forage, fodder, straw, and hay of cereal grains at 1.0 ppm; legume vegetables (succulent including soybeans) at 0.3 ppm; cucurbits at 0.3 ppm; sorghum grain at 1.0 ppm; sorghum aspirated grain fractions at 3.0 ppm; stone fruit at 0.2 ppm; and wheat grain at 0.02 ppm. Because of the amount of spinosad residue found in corn, sorghum, and wheat products used in animal feeds as well as those commodities with existing residue tolerances that are potentially used in animal rations, the following increases in livestock residue tolerances are being proposed: livestock, meat residue tolerance of 0.1 ppm; livestock, meat byproduct residue tolerance of 0.4 ppm; livestock, fat residue tolerance of 1.5 ppm; a milk residue tolerance of 0.1 ppm; a milk fat residue tolerance of 1.5 ppm. In addition, the following poultry residue tolerances are being proposed: poultry, fat at 0.2 ppm; poultry, meat and meat byproducts at 0.02 ppm; and eggs at 0.02 ppm. An adequate analytical method is available for enforcement purposes. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA; however, EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of spinosad in plants (apples, cabbage, cotton, tomato, and turnip) and animals

(goats and poultry) is adequately understood for the purposes of these tolerances. A rotational crop study showed no carryover of measurable spinosad related residues in representative test crops.

2. *Analytical method*. There is a practical method (immunoassay) for detecting 0.005 ppm and measuring 0.01 ppm levels of spinosad in or on food with a limit of detection that allows monitoring of food with residues at or above the levels set for these tolerances. The method has had a successful method tryout in the EPA's laboratories.

3. *Magnitude of residues*. Magnitude of residue studies were conducted for stone fruit (7 sites for cherries, 6 sites for peaches, 4 sites for plums, and 2 sites for prunes); cucurbits (6 sites for cucumbers, 6 sites for muskmelons, and 3 sites for summer squash); sweet corn (12 sites); field corn (5 sites at 5 x label rate); legume vegetables (11 sites for snap beans, 7 sites for snow peas, and 7 sites at 5 x label rate for soybeans); sorghum (9 sites); and wheat (6 sites at 5 x label rate). Residues found in these studies ranged from ND to 0.14 ppm on stone fruit; ND to 0.19 ppm in cucurbits; ND for field corn grain and sweet corn (K=CWHR); 0.09 to 0.57 ppm for corn forage; 0.03 to 0.82 ppm for corn fodder; ND to 0.23 ppm for legume vegetables; 0.03 to 0.68 ppm for sorghum grain; 0.06 to 0.18 ppm for sorghum forage; 0.06 to 0.29 ppm for sorghum fodder; 2.02 ppm for sorghum aspirated grain fractions; ND to 0.09 ppm for wheat grain; ND to 0.07 ppm for wheat forage; 0.01 to 0.20 ppm for wheat hay; and 0.01 to 0.73 ppm for wheat straw.

B. Toxicological Profile

1. *Acute toxicity*. Spinosad has low-acute toxicity. The rat oral LD_{50} is 3,738 milligram/kilogram (mg/kg) for males and > 5,000 mg/kg for females, whereas the mouse oral LD_{50} is > 5,000 mg/kg. The rabbit dermal LD_{50} is > 5,000 mg/kg and the rat inhalation LC_{50} is > 5.18 mg/1 air. In addition, spinosad is not a skin sensitizer in guinea pigs and does not produce significant dermal or ocular irritation in rabbits. End use formulations of spinosad that are water based suspension concentrates have similar low acute toxicity profiles.

2. *Genotoxicty*. Short-term assays for genotoxicity consisting of a bacterial reverse mutation assay (Ames test), an *in vitro* assay for cytogenetic damage using the Chinese hamster ovary cells, an *in vitro* mammalian gene mutation assay using mouse lymphoma cells, an *in vitro* assay for DNA damage and repair in rat hepatocytes, and an *in vivo* cytogenetic assay in the mouse bone marrow (micronucleus test) have been conducted with spinosad. These studies show a lack of genotoxicity.

3. *Reproductive and developmental toxicity*. Spinosad caused decreased body weights in maternal rats given 200 mg/kg/day by gavage (highest dose tested). This was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The no-observed-effect levels (NOELs) for maternal and fetal toxicity in rats were 50 and 200 mg/kg/day, respectively. A teratology study in rabbits showed that spinosad caused decreased body weight gain and a few abortions in maternal rabbits given 50 mg/kg/day (highest dose tested). Maternal toxicity was not accompanied by either embryo toxicity, fetal toxicity, or teratogenicity. The NOELs for maternal and fetal toxicity in rabbits were 10 and 50 mg/kg/day, respectively. In a two-generation reproduction study in rats, parental toxicity was observed in both males and females given 100 mg/kg/day (highest dose tested). Perinatal effects (decreased litter size and pup weight) at 100 mg/kg/day were attributed to maternal toxicity. The NOEL for maternal and pup effects was 10 mg/kg/day.

4. *Subchronic toxicity*. Spinosad was evaluated in 13-week dietary studies and showed NOELs/no-observed-adverse-effect levels (NOAELs) of 4.89 and 5.38 mg/kg/day, respectively in male and female dogs; 6 and 8 mg/kg/day, respectively in male and female mice; and 33.9 and 38.8 mg/kg/day, respectively in male and female rats. No dermal irritation or systemic toxicity occurred in a 21–day repeated dose dermal toxicity study in rabbits given 1,000 mg/kg/day.

5. *Chronic toxicity*. Based on chronic testing with spinosad in the dog and the rat, the EPA has set a reference dose (RfD) of 0.027 mg/kg/day for spinosad. The RfD has incorporated a 100-fold safety factor to the NOELs found in the chronic dog study to account for inter- and intra-species variation. The NOELs shown in the dog chronic study were 2.68 and 2.72 mg/kg/day, respectively for male and female dogs. The NOELs (systemic) shown in the rat chronic/carcinogenicity/neurotoxicity study were 9.5 and 12.0 mg/kg/day, respectively for male and female rats. Using the Guidelines for Carcinogen Risk Assessment published September 24, 1986 (51 FR 33992), it is proposed that spinosad be classified as Group E for carcinogenicity (no evidence of carcinogenicity) based on the results of carcinogenicity studies in two species. There was no evidence of carcinogenicity in an 18-month mouse feeding study and a 24-month rat feeding study at all dosages tested. The NOELs shown in the mouse oncogenicity study were 11.4 and 13.8 mg/kg/day, respectively for male and female mice. A maximum tolerated dose was achieved at the top dosage level tested in both of these studies based on excessive mortality. Thus, the doses tested are adequate for identifying a cancer risk. Accordingly, a cancer risk assessment is not needed.

6. *Animal metabolism*. There were no major differences in the bioavailability, routes or rates of excretion, or metabolism of spinosyn A and spinosyn D following oral administration in rats. Urine and fecal excretions were almost completed in 48-hours post-dosing. In addition, the routes and rates of excretion were not affected by repeated administration.

7. *Metabolite toxicology*. The residue of concern for tolerance setting purposes is the parent material (spinosyn A and spinosyn D). Thus, there is no need to address metabolite toxicity.

8. *Neurotoxicity*. Spinosad did not cause neurotoxicity in rats in acute, subchronic, or chronic toxicity studies.

9. *Endocrine effects*. There is no evidence to suggest that spinosad has an effect on any endocrine system.

C. Aggregate Exposure

1. *Dietary exposure*. For purposes of assessing the potential dietary exposure from use of spinosad on stone fruit, cucurbits, corn (field, sweet, and pop), legume vegetables (succulent including soybeans), sorghum, and wheat as well as from other existing spinosad crop uses, a conservative estimate of aggregate exposure is determined by basing the theoretical maximum residue concentrations (TMRC) on the proposed tolerance levels for spinosad and assuming that 100% of these proposed new crops and other existing (registered for use) crops grown in the United States were treated with spinosad. The TMRC is obtained by multiplying the tolerance residue levels by the consumption data which estimates the amount of crops and related foodstuffs consumed by various population subgroups. The use of a tolerance level and 100% of crop treated clearly results in an overestimate of human exposure and a safety determination for the use of spinosad on crops cited in this summary that is based on a conservative exposure assessment.

2. *Drinking water*. Another potential source of dietary exposure are residues in drinking water. Based on the available environmental studies conducted with spinosad wherein it's properties show little or no mobility in soil, there is no anticipated exposure to residues of spinosad in drinking water.

In addition, there is no established maximum concentration level (MCL) for residues of spinosad in drinking water.

3. *Non-dietary exposure*. Spinosad is currently registered for use on a number of crops including cotton, fruits, and vegetables in the agriculture environment. Spinosad is also currently registered for outdoor use on turf and ornamentals at low rates of application (0.04 to 0.54 lb active ingredient (a.i.) per acre) and indoor use for drywood termite control (extremely low application rates used with no occupant exposure expected). Thus, the potential for non-dietary exposure to the general population is considered negligible.

D. Cumulative Effects

The potential for cumulative effects of spinosad and other substances that have a common mechanism of toxicity is also considered. In terms of insect control, spinosad causes excitation of the insect nervous system, leading to involuntary muscle contractions, prostration with tremors, and finally paralysis. These effects are consistent with the activation of nicotinic acetylcholine receptors by a mechanism that is clearly novel and unique among known insecticidal compounds. Spinosad also has effects on the Gamma aminobatopic acid (GABA) receptor function that may contribute further to its insecticidal activity. Based on results found in tests with various mammalian species, spinosad appears to have a mechanism of toxicity like that of many amphiphilic cationic compounds. There is no reliable information to indicate that toxic effects produced by spinosad would be cumulative with those of any other pesticide chemical. Thus it is appropriate to consider only the potential risks of spinosad in an aggregate exposure assessment.

E. Safety Determination

1. *U.S. population*. Using the conservative exposure assumptions and the proposed RfD described in Unit 1.B.5 of this document, the aggregate exposure to spinosad use on stone fruit, cucurbits, corn (field, sweet, and pop), legume vegetables (succulent including soybeans), sorghum, and wheat and other existing crop uses will utilize 25.4% of the RfD for the U.S. population. A more realistic estimate of dietary exposure and risk relative to a chronic toxicity endpoint is obtained if average (anticipated) residue values from field trials are used. Inserting the average residue values in place of tolerance residue levels produces a more realistic, but still conservative risk assessment. Based on average or anticipated residues in a dietary risk analysis, the use of spinosad on the list in this unit of pending crop uses and other existing crop uses will utilize 4.0% of the RfD for the U.S. population. EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health. Thus, it is clear that there is reasonable certainty that no harm will result from aggregate exposure to spinosad residues on existing and pending crop uses.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of spinosad, data from developmental toxicity studies in rats and rabbits and a 2-generation reproduction study in the rat are considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from pesticide exposure during prenatal development. Reproduction studies provide information relating to effects from exposure to the pesticide on the reproductive capability and potential systemic toxicity of mating animals and on various parameters associated with the well-being of pups.

FFDCA section 408 provides that EPA may apply an additional safety factor for infants and children in the case of threshold effects to account for pre- and post-natal toxicity and the completeness of the database. Based on the current toxicological data requirements, the database for spinosad relative to pre- and post-natal effects for children is complete. Further, for spinosad, the NOELs in the dog chronic feeding study which was used to calculate the RfD (0.027 mg/kg/day) are already lower than the NOELs from the developmental studies in rats and rabbits by a factor of more than 10-fold.

Concerning the reproduction study in rats, the pup effects shown at the highest dose tested were attributed to maternal toxicity. Therefore, it is concluded that an additional uncertainty factor is not needed and that the RfD at 0.027 mg/kg/day is appropriate for assessing risk to infants and children.

In addition, the EPA has determined that the 10 x factor to account for enhanced sensitivity of infants and children is not needed because:

i. The data provided no indication of increased susceptibility of rats or rabbits to *in utero* and/or post-natal exposure to spinosad. In the prenatal developmental toxicity studies in rats and rabbits and 2-generation reproduction in rats, effects in the offspring were observed only at or below treatment levels which resulted in evidence of parental toxicity.

ii. No neurotoxic signs have been observed in any of the standard required studies conducted.

iii. The toxicology data base is complete and there are no data gaps.

Using the conservative exposure assumptions previously described (tolerance level residues), the percent RfD utilized by the aggregate exposure to residues of spinosad on stone fruits, cucurbits, corn (field, sweet, and pop), legume vegetables (succulent including soybeans), sorghum and wheat and existing crop uses is 51.0% for children 1 to 6 years old, the most sensitive population subgroup. If average or anticipated residues are used in the dietary risk analysis, the use of spinosad on these crops will utilize 9.2% of the RfD for children 1 to 6 years old. Thus, based on the completeness and reliability of the toxicity data and the conservative exposure assessment, it is concluded that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to spinosad residues on the above proposed including existing crop uses.

F. International Tolerances

There are no Codex Maximum Residue Levels established for residues of spinosad on stone fruit, cucurbits, corn (field, sweet, and pop), legume vegetables (succulent including soybeans), sorghum, and wheat or any other food or feed crop. (Beth Edwards)

2. Zeneca Ag Products

PP 6F3344

EPA has previously received a pesticide petition (PP 6F3344) from Zeneca Ag Products, 1800 Concord Pike, Wilmington, DE proposing pursuant to section 408(d) of the (FFDCA), 21 U.S.C 346a(d) to amend 40 CFR part 180 by establishing tolerances for the inert ingredient safener *N*,*N*-diallyl dichloroacetamide (dichlormid) of 0.05 ppm when applied to the raw agricultural commodities field corn grain, field corn fodder and field corn forage. Based on that petition EPA established time-limited tolerances on March 18, 1994, contingent upon submission of data from two chronic feeding/oncogenicity studies. The registrant provided those data on March 27, 1998, and is herein proposing that EPA extend that petition and remove the time-limitations previously imposed. EPA has determined that the petition contains data or information regarding the elements set forth in section 408(d)(2) of the FFDCA;

however EPA has not fully evaluated the sufficiency of the submitted data at this time or whether the data supports granting of the petition. Additional data may be needed before EPA rules on the petition.

A. Residue Chemistry

1. *Plant metabolism*. The metabolism of dichlormid in corn plants is understood for the purposes of the proposed tolerances. The metabolism of dichlormid in corn is extensive and rapid. The principle route involves the displacement of the chlorine atoms, probably through glutathione mediated reductive dechlorination, followed by oxidation to *N,N*-diallyl glycolamide. The glycolamide is subsequently further oxidized to form the oxamic acid or conjugated with natural sugars. The presence of $^{14}CO_2$ evolved from the plants following treatment of the soil demonstrates the catabolism of the ^{14}C atom and its probable inclusion in natural biosynthetic pathways. EPA has previously determined that dichlormid is the residue of concern for tolerance setting purposes.

2. *Analytical methods*. An enforcement method is available and involves extraction, filtration, and concentration, followed by analysis by Gas Liquid Chromatography (GLC) with a selective thermionic detector. The method has been validated by the EPA at the Beltsville laboratory and included in the Pesticide Analytical Manual, Vol. II (PAM II). The validated limit of quantitation of the method allows monitoring of field corn and processed fractions at the proposed tolerances for dichloromid of 0.05 ppm.

3. *Magnitude of the residues*. Many crop residue field trials have been conducted on field corn covering the major growing areas in the United States with dichlormid applied pre emergence at application rates up to 1.0 lb a.i. per acre. In all trials dichloromid residues in grain and processed fractions were all < 0.05 ppm. In a separate trial corn was treated with two applications of dichlormid (one pre emergence and one post emergence) at a rate of 0.83 lb a.i. per acre (to simulate an exaggerated rate of 1.66 lb a.i. per acre). Samples of grain from this trial were processed under conditions which simulated commercial practice. Dichlormid residues in grain and processed fractions were all < 0.05 ppm. Dichlormid has been shown to be stable in field corn crop fractions for a minimum of 3 years when stored at -18 °C. No transfer of residues to animals through the diet is expected.

B. Toxicological Profile

1. *Acute toxicity*. Dichlormid has low acute toxicity, available data include: two rat acute oral studies with LD_{50}'s of 2,080 mg/kg for males/2,030 for females and 2,816 mg/kg for males and 2,146 mg/kg for females, respectively; a rat acute dermal study with an LD_{50} of > 2,040 mg/kg and a rabbit acute dermal study with an LD_{50} of > 5,000 mg/kg; two rat inhalation studies with LC_{50}'s of > 5.5 mg/l and > 5.6 mg/l, respectively; two primary eye irritation studies in the rabbit showing no irritation and slight irritation, respectively; two primary dermal irritation studies in the rabbit showing mild to moderate skin irritation, and a skin sensitization study which showed that dichlormid was a mild skin sensitizer in the guinea pig.

2. *Genotoxicity*. Dichlormid was not mutagenic in a range of *in vitro* assays including the Salmonella/microsome (Ames) assay, the human lymphocyte cytogenetic assay (both assays with and without metabolic activation) and an unscheduled DNA synthesis (DNA repair) assay in hepatocytes. In the L5178Y mouse lymphoma assay small increases in mutant frequency were observed only at cytotoxic concentrations and were not considered to be significant. *In vivo*, dichlormid was negative in the mouse micronucleus test and in the rat unscheduled DNA synthesis assay, when tested at the maximum tolerated dose.

3. *Developmental toxicity*. i. In an initial rat developmental effects study, previously submitted and accepted by EPA, female albino rats were dosed at 0, 10, and 40 mg/kg dichlormid in the diet from days 6 through 15 of gestation and a NOEL of 40 mg/kg/day for both maternal toxicity and developmental toxicity was determined.

ii. In a second study, rats were dosed orally by gavage with 0, 10, 40, or 160 mg/kg/day. The NOEL for maternal toxicity was 10 mg/kg/day based on a reduction in bodyweight gain and food consumption at 40 and 160 mg/kg/day. The developmental NOEL was determined to be 40 mg/kg/day based on marginal foetotoxic effects, including extra 14th ribs probably due to maternal stress, slight sternebra misalignment and some centra unossification, at 160 mg/kg/day.

iii. In an additional developmental effects study, rabbits were dosed orally by gavage with 0, 5, 30, or 180 mg/kg/day. The lowest-observed-effect level (LOEL) for both maternal and foetotoxicity was 180 mg/kg/day, characterized by reduced body weight gain and food consumption and a small increase in post-implantation loss, partial ossification and misshapen/fused sternebrae. The NOEL for both maternal and developmental toxicity was 30 mg/kg/day.

4. *Subchronic toxicity*. i. In an initial 90 day subchronic oral feeding study in the rat, previously submitted and accepted by EPA, animals were dosed at 0, 10, 40, and 160 mg/kg/day in the diet and a NOEL of 10 mg/kg/day was established.

ii. In a second study, groups of 12 male and 12 female Wistar-derived alpk: APfSD rats were fed diets containing 0, 20, 200, or 2,000 ppm dichlormid for 90 days. Significant reductions in body/weight gain and food consumption were seen in male and female rats receiving 2,000 ppm dichlormid and to a lesser degree in females at 200 ppm. The liver was identified as the principal target organ (enlargement, increased (APDM) activity in females, centrilobular hypertrophy, increased bile duct pigmentation) in the 2,000 ppm group. The NOEL was 20 ppm (equivalent to approximately 1 mg/kg/day (see discussion under Chronic toxicity in Unit 2.B.5. of this document) and the LOEL was 200 ppm, based on reduced body/weight gain and food consumption and a marginal increase in APDM activity in females and liver enlargment in males.

iii. In 90–day dog feeding study, previously submitted and accepted by EPA, animals were dosed (4 dogs/sex/dose) at 0, 1, 5, 25, and 50 mg/kg/day. The NOEL was 5 mg/kg/day and the LOEL 25 mg/kg/day based on reduced bodyweight gain, degenerative changes in voluntary muscle and increased liver weight with an associated increase in plasma alkaline phosphatase activity.

iv. In a 14-week rat inhalation study, groups of 18 Sprague-Dawley CD rats were subjected to a whole body exposure of 0, 2.0, 19.9, or 192.5 mg/m^3 for 6 hours per day, 5 days per week. The NOEL was 2.0 mg/m^3 based on histopathologic tissue alterations to the nasal olfactory epithelium at 19.9 and 192.5 mg/m^3, suggesting that dichlormid was a mild irritant to the nasal cavity. An increase in relative liver, kidney, and lung weights, that was not supported by gross or histopathological observations, was considered due to a combination of stress and inappetance at 19.9 and 192.5 mg/m^3.

5. *Chronic toxicity*. Rats (64/sex/group) were fed diets containing 0, 20, 100, or 500 ppm dichlormid (0, 1.3, 6.5, 32.5 mg/kg/day for males and 0, 1.5, 7.5, 37.5 mg/kg/day for females) for up to 2 years. At 500 ppm in both males and females there were treatment-related effects on growth and food consumption, minor reductions in

plasma triglycerides and in males, increased liver weights, accompanied by hepatocyte vaculolation and pigmentation effects. In females there was a slight overall increase in malignant tumors, primarily uterine adenocarcinomas, at 500 ppm but this specific increase was within the spontaneous incidence observed within historical control values. It was concluded that there was no evidence of oncogenicity associated with dichlormid treatment. The NOEL for chronic toxicity was 100 ppm (6.5 and 7.5 mg/kg/day for males and females respectively). In an 18-month oncogenicity study, mice (55/sex/group) were fed dichlormid at doses of 0, 10, 50, or 500 ppm (0, 1.4, 7.0, 70 mg/kg for males and 0, 1.84, 9.2, 92 mg/kg for females). At 500 ppm there was a slight increase in mortality for females from week 64 onwards and bodyweights and food utilization were reduced in males, and to a lesser extent in females. Also mice fed 500 ppm dichlormid showed non-neoplastic changes which were minor and consisted of changes in severity or incidence of common spontaneous findings. Based on these effects, the chronic NOEL was 50 ppm (7.0 and 9.2 mg/kg/day for males and females respectively). There was a marginal increase in Harderian gland adenomas in males at 500 ppm but this was considered to reflect the variable spontaneous tumor rate seen in this strain and sex of mouse. It was concluded there was no evidence of oncogenicity associated with dichormid treatment.

Based on available chronic toxicity data, Zeneca believes the RfD for dichlormid is 0.07 mg/kg/day. This RfD is based on the 2-year feeding study in rats with an NOEL of 7 mg/kg/day. An uncertainty factor of 100 was used to account for inter-species extrapolation and intra-species variability. The 2 year rat study is consistent with, but supersedes, the 90 day rat study. The 2 year rat NOEL of 7 mg/kg/day lies between 1.7 and 17 mg/kg/day derived from the NOEL and LOEL figures of 20 and 200 ppm respectively for the most recent 90 day rat study. Thus the overall NOEL in the rat for both chronic and subchronic exposure should be regarded as 7 mg/kg/day. Based on the proposed Guidelines for Carcinogenic Risk Assessment (April 23, 1996) Zeneca believes that dichlormid is not likely to be a human carcinogen, and a margin of exposure (MOE) approach should be used for human risk assessment.

6. *Animal metabolism*. In the rat dichlormid is readily absorbed and fairly rapidly excreted with extensive metabolism; the major route results in the formation of *N,N*-diallylglycolamide and its glucuronide conjugate. The glycolamide is subsequently oxidized to the *N,N*-diallyloxamic acid. An alternative pathway involves cleavage of dichlormid to form dichloroacetic acid, which was also a significant urinary metabolite. The further biotransformation of this metabolite and of *N,N*-diallyloxamic acid would lead to the observed evolution of carbon dioxide.

7. *Metabolite toxicity*. No unique plant or soil metabolites have been identified that warrant a separate toxicological assessment.

8. *Endocrine disruption*. No specific tests have been conducted with dichlormid to determine whether the chemical may have an effect in humans that is similar to an effect produced by a naturally occurring estrogen or other endocrine effects. However, there is no overall trend in the toxicology database that indicates that dichlormid would have endocrine disrupting activity.

C. Aggregate Exposure

1. *Food*. To assess the potential dietary exposure using the proposed tolerances of 0.05 ppm, Zeneca has estimated the aggregate exposure based on the theoretical maximum residue contribution (TMRC). This is a highly conservative over-estimation of human exposure, based on tolerance level residues (0.05 ppm) and 100% crop treated. The analysis was determined using the DEEM software and the USDA CSF II 94–95 data.

2. *Drinking water*. Dichlormid is very rapidly degraded in soil (laboratory measured aerobic half life of 8 days), and applied at a maximum rate of 1.0 lb/acre, so despite only exhibiting moderate adsorption to soil, (Koc 36–49), the leaching potential for dichlormid to reach ground water is expected to be low. The impact of the interactive processes of adsorption and degradation on leaching have been assessed using EPA mathematical models of pesticide movement in soil.

Drinking water estimate concentrations (DWEC) were calculated using (SCI-GROW) and (GENEEC). These predict a ground water concentration of 0.02 ppb, and surface water concentrations of 49.71 ppb for an instaneous peak and 49.27 for a 56 day average. Drinking water levels of concern (DWLOC) were calculated for both chronic and acute exposure according to the EPA (SOP). All the values are less than the DWEC. As EPA believes there is negligible risk at values less than 100% of the DWEC, Zeneca does not expect exposure to dichlormid residues in drinking water to be a concern.

3. *Non-dietary exposures*. As dichlormid is used only on agricultural crops and is not used in or around the home, exposure to the general population is unlikely.

D. Cumulative Effects

Zeneca has considered the potential for cumulative effects of dichlormid and other substances that have a common mechanism of toxicity. Zeneca does not have any reliable information to suggest that dichlormid has any toxic effects that arise from toxic mechanisms, that are common to other substances. Therefore, a consideration of common mechanism and cumulative effects with other substances is not appropriate for dichlormid and Zeneca is considering only the potential risks of dichlormid in this exposure assessment.

E. Safety Determination

1. *U.S. population*—i. *Chronic risk*. Using the conservative exposure assumptions described above and based on the completeness and reliability of the toxicity data base for dichlormid, Zeneca has calculated the aggregate exposure will be 0.1% (0.00006 mg/kg/day) of the RfD (0.07 mg/kg/day) for the U.S population. The most highly exposed subgroup is non-nursing infants a TMRC of 0.000149 mg/kg/day or 0.27% of the RfD. As EPA generally has no concern for exposures below 100% of the RfD because the RfD represents the level at or below which daily aggregate dietary exposure over a lifetime will not pose appreciable risks to human health, Zeneca concludes that there is a reasonable certainty that no harm will result from aggregate exposure to dichlormid residues.

ii. *Acute risk*. The acute toxicity of dichlormid is low, and there are no concerns for acute-dietary, occupational or non-occupational exposures to dichlormid.

2. *Infants and children*. In assessing the potential for additional sensitivity of infants and children to residues of dichlormid, data from developmental toxicity studies in the rat and rabbit have been considered. The developmental toxicity studies are designed to evaluate adverse effects on the developing organism resulting from maternal pesticide exposure during gestation. There was no evidence to suggest that dichlormid was a developmental toxicant in either the rat or rabbit. It was also observed that there was no risk below maternally toxic doses as the NOEL for developmental effects in the rat was 40 mg/kg/day as opposed to the maternal NOEL of 10

mg/kg/day and, in the rabbit study, the NOEL for both maternal and developmental effects was 30 mg/kg/day. For both these reasons, and the fact that the RfD is based on the chronic rat study which has a NOEL considerably lower than the developmental NOELs, Zeneca believes that an additional uncertainty factor is not warranted for the safety of infants and children. Reliable data supports the use of a 100-fold uncertainty factor (MOE) to account for inter-species extrapolation and intra-species variability which will be appropriate to protect infants and children. Using the same conservative exposure assumptions used for the determination in the general population, Zeneca has concluded that the percentage of RfD that will be utilized by aggregate exposure to dichlormid is 0.2% for non-nursing infants (the group at highest risk). Therefore, based on the completeness and reliability of the toxicity data base and the conservative exposure assessment, Zeneca concludes that there is a reasonable certainty that no harm will result to infants and children from aggregate exposure to dichlormid residues.

F. International Tolerances

A Maximum Residue Level has not been established for dichlormid by the Codex Alimentarius Commission. (Treva Alston)

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ENVIRONMENTAL PROTECTION AGENCY

[PB–402404–OK; FRL–6027–3]

Lead-Based Paint Activities in Target Housing and Child-Occupied Facilities; State of Oklahoma's Authorization Application

AGENCY: Environmental Protection Agency (EPA).
ACTION: Notice; request for comments and opportunity for a public hearing.

SUMMARY: On August 10, 1998, the State of Oklahoma submitted an application for EPA approval to administer and enforce training and certification requirements, training program accreditation requirements, and work practice standards for lead-based paint activities in target housing and child-occupied facilities under section 402 of the Toxic Substances Control Act (TSCA). This notice announces the receipt of Oklahoma's application, and provides a 45–day public comment period and an opportunity to request a public hearing on the application. Oklahoma has provided a certification that this program meets the requirements for approval of a State program under section 404 of TSCA. Therefore, pursuant to section 404, the program is deemed authorized as of the date of submission. If EPA finds that the program does not meet the requirements for approval of a State program, EPA will disapprove the program, at which time a notice will be issued in the **Federal Register** and the Federal program will be established.

DATES: The State program became effective August 10, 1998. Submit comments on the authorization application on or before November 2, 1998. Public hearing requests must be submitted on or before September 30, 1998.

If a public hearing is requested and granted, the hearing will be held on October 7, 1998, at 1 p.m., at the Oklahoma Department of Environmental Quality, 707 North Robinson, Multi-Purpose Room, 1st Floor, Oklahoma City, Oklahoma. If a public hearing is not requested, this meeting time and place will be canceled. Therefore, individuals are advised to verify the status of the public hearing by contacting the Regional Lead Coordinator at the telephone number or address provided in the ''FOR FURTHER INFORMATION CONTACT'' unit of this notice after September 30, 1998 and before the October 7, 1998, scheduled public hearing date.

ADDRESSES: Submit all written comments and/or requests for a public hearing identified by docket control number ''PB–402404–OK'' (in duplicate) to: Environmental Protection Agency, Region 6, 6PD-T, 1445 Ross Avenue., Suite 1200, Dallas, TX 75202-2733.

Comments, data, and requests for public hearing may also be submitted electronically to robinson.jeffrey@epamail.epa.gov. Follow the instructions under Unit IV. of this document. No Confidential Business Information (CBI) should be submitted through e-mail.

FOR FURTHER INFORMATION CONTACT: Jeffrey Robinson, Regional Lead Coordinator, 1445 Ross Avenue, Suite 1200, 6PD-T, Dallas, TX 75202-2733. telephone: 214–665–7577; e-mail address: robinson.jeffrey@epamail.epa.gov.

SUPPLEMENTARY INFORMATION:

I. Background

On October 28, 1992, the Housing and Community Development Act of 1992, Pub. L. 102-550, became law. Title X of that statute was the Residential Lead-Based Paint Hazard Reduction Act of 1992. That Act amended TSCA (15 U.S.C. 2601 *et seq.*) by adding Title IV (15 U.S.C. 2681-92), entitled Lead Exposure Reduction.

Section 402 of TSCA (15 U.S.C. 2682) authorizes EPA to promulgate final regulations governing lead-based paint activities. Lead-based paint activities is defined in Section 402(b) of TSCA and authorizes EPA to regulate lead-based paint activities in target housing, public buildings built prior to 1978, commercial buildings, bridges and other structures or superstructures. Those regulations are to ensure that individuals engaged in such activities are properly trained, that training programs are accredited, and that individuals engaged in these activities are certified and follow documented work practice standards. Under section 404 of TSCA, a State may seek authorization from EPA to administer and enforce its own lead-based paint activities program.

On August 29, 1996 (61 FR 45777) (FRL–5389–9), EPA promulgated final TSCA section 402/404 regulations governing lead-based paint activities in target housing and child-occupied facilities (a subset of public buildings). Those regulations are codified at 40 CFR part 745, and allow both States and Indian Tribes to apply for program authorization. On August 31, 1998, EPA will institute the Federal program in States or Indian Country without an authorized program, as provided by section 404(h) of TSCA.

States and Indian Tribes that choose to apply for program authorization must submit a complete application to the appropriate Regional EPA office for review. Those applications will be reviewed by EPA within 180 days of receipt of the complete application. To receive EPA approval, a State or Indian Tribe must demonstrate that its program is at least as protective of human health and the environment as the Federal program, and provides adequate enforcement (section 404(b) of TSCA, 15 U.S.C. 2684(b)). EPA's regulations (40 CFR part 745, subpart Q) provide the detailed requirements a State or Tribal program must meet in order to obtain EPA approval.

A State may choose to certify that its lead-based paint activities program meets the requirements for EPA approval by submitting a letter signed by the Governor or Attorney General stating that the program meets the requirements of section 404(b) of TSCA. Upon submission of such certification letter, the program is deemed authorized until such time as EPA disapproves the program application or withdraws the authorization.