



Draft Assessment Report (DAR)

- public version -

**Initial risk assessment provided by the rapporteur Member State
Germany for the existing active substance**

CALCIUM PHOSPHIDE

**of the third stage (part B) of the review programme
referred to in Article 8(2) of Council Directive 91/414/EEC**

Volume 3, Annex B, part 5, B.9

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Annex B

Calcium phosphide

B-9: Ecotoxicology

WARNING: This document forms part of an EC evaluation data package and should not be read in isolation. Registration must not be granted on the basis of this document.

B.9 Ecotoxicology

The following data are relevant concerning the conditions of use of the product POLYTANOL with 18 % calcium phosphide active substance. POLYTANOL is a technical product and no formulation.

Relevant chemical and physical properties

Plant protection product:	POLYTANOL
Active substance:	Calcium phosphide
Concentration of active substance:	18 w/w %
Type of formulation:	Granule (no formulation)
Function:	Rodenticide

Conditions of use of polytanol

Field of use:	Against damage by common voles (<i>Arvicola terrestris</i>) and moles (<i>Talpa europaea</i>) in grassland, agriculture, horticulture, orchards, domestic and amateur gardening.
Application rate:	Min: 8 g (21 pieces granule) pro run/tunnel; max: 10 g pro run/tunnel. Depending on the context of infestation, this is considered to correspond to a maximum field application rate of 8 - 10 kg/ha.
Water volume:	Not applicable, since the product is a ready-to-use solid grain bait not intended for use with water.
Number of treatments:	If required, at any time of year.
Method of application:	Covered application, with the aid of an applicator or drop tube or drop gun.

B.9.1 Effects on birds (Annex IIA 8.1; Annex IIIA 10.1)

B.9.1.1 Acute oral toxicity (Annex IIA 8.1; Annex IIA 10.1)

Reference number:	IIA 8.1.1
Author:	WHO; AVS 2005-25
Title:	Phosphine and selected metal phosphide.
Date:	1988
DocID:	Environmental Health Criteria 73 - IPCS International Programme on Chemical Safety, Geneva, Switzerland, pp. 56-59, 1988
Guidelines:	Not stated
GLP:	No
Validity:	Validation not possible, Literature summary

Material and methods

Zinc phosphide, phosphine. Dietary experiments and inhalation studies.

Findings:

Summary of studies on the lethality of zinc phosphide and phosphine on birds.

Ikeda (1971; see IIA 8.1.3, AVS 2002-21) studied the lethality of zinc phosphide for the quail and reported an oral LD₅₀ of 35 mg/kg body weight; there was a reduction in egg laying at 3.5 mg/kg.

Shivanandappa (1979, AVS2007-65) found that the acute oral LD₅₀ and LD₉₀ of zinc phosphide in poultry were 25 and 31 mg/kg body weight, respectively. Treatment of chickens with encapsulated doses of 14, 21, 31.5, or 47.2 mg/kg body weight, daily for 4 weeks, resulted in deaths at all doses. Mortality was 12 % at the lowest dose and 100 % at the highest dose, where death occurred within 6 – 18 h of administration of the first dose.

Hill et al. (1975; see IIA 8.1.2, AVS 96-00079) studied the effects of zinc phosphide administered in the diet for 5 days to mallard ducks. The dosing period was followed by 3 days of untreated feed. The zinc phosphide concentration of 1285 mg/kg diet was calculated to produce 50 % mortality in this short-term test.

Baxland and Gordon (1945) administered oral doses of zinc phosphide ranging from 15 to 400 mg/kg body weight to single domestic hens of 2 species. All the birds receiving 20 mg/kg or less survived.

Though the acute oral LD₅₀ for most avian species is generally in the range of 20 – 100 mg zinc phosphide/kg body weight, it has been reported that chickens fed 12 – 16 mg/kg body weight displayed toxic symptoms, including reduced red-cell counts, reduced haemoglobin concentration, and leukocytosis, 1 – 1.5 h after dosing (Kozhemyakin et al., 1971).

Klimmer (1969; for reference see IIA, 8.1.1, AVS2005-24) exposed 3 turkeys to phosphine at a concentration of 211 mg/m³ in an acute inhalation study. The turkeys exhibited apathy, restlessness, dyspnoea, and tonic-clonic convulsions, and died after 68, 74, and 80 min, respectively. When examined, organs were congested with oxygenated blood. Hens exhibited tonic-clonic convulsions and died after an average of 59 min (range, 50 – 64 min). Their organs were also congested with oxygenated blood.

Conclusion:

No studies were performed with calcium phosphide. According to the state of knowledge the toxicity of calcium phosphide is solely caused by release of phosphine (PH₃). Therefore, toxicities of phosphine and metal phosphides will be considered together and effects of calcium phosphide can be extrapolated from studies with other metal phosphides and phosphine. The data presented above are taken from the literature. They allow conclusions on the effects of the compounds. However, the studies do not fully comply with current standards for the testing of plant protection products and should be used as additional information for the risk assessment. The presented data on acute effects on birds is considered sufficient, and no further studies are required, since exposure of birds to calcium phosphide containing granules is not expected. Additionally, reasons of animal welfare are considered. The LD₅₀ values of 25 and 35 mg zinc phosphide/kg body weight are equivalent to 6.6 and

9.2 mg PH₃/kg body weight or extrapolated to Ca₃P₂ 17.7 and 24.7 mg/kg/body weight, respectively.

**An English translation for the following above cited study:
Kozhemyakin et al. (1971) is missing.**

B.9.1.2 Dietary toxicity (Annex IIA 8.1.2; Annex IIIA 10.1.1)

Reference number: IIA 8.1.2
Author: Anonymous, AVS 2006-179
Title: A Zinc phosphide dietary LC₅₀ study against bobwhite quail and mallard ducks.
Date: 1978. Submitted July 15, 1978 under 6704-78 to U.S. EPA by U.S. Dept. of Interior, Fish and Wildlife Service, Washington, D.C. CDL: 233244-A, U.S.
DocID: Report no.: EPA RED (1998): MRID 00006025
Guidelines: Acc. to Heath and Stickel (1965) with modifications
GLP: No
Validity: Acceptable

Materials and methods

Test item: Zinc phosphide technical; batch: not stated; purity: 94 %;
Test procedure: short term dietary toxicity of zinc phosphide;
Test animals: (i) bobwhite quail (*Colinus virginianus*), 10-14 days old at test begin (supplier of eggs : Oak Ridge Game Farm, Gravette, Arkansas, USA; rearing at Denver Wildlife Research Center, Colorado, USA), (ii) mallard ducks (*Anas platyrhynchos*), 10 days old at test begin (supplier: Whistling Wings Game Farm, Hanover, Illinois, USA);
Housing: each treatment group in one pen within the brooder, temperature: 34 °C, humidity and light: not controlled;
Body weight: not stated;
Number of animals: (i) 13 quails per group, (ii) 10 mallards per group;
Dose levels: (dilution factor of 2.25) (i) and (ii) 0.0, 20.0, 45.0, 101.2, 227.8, 512.6, 1153.3, and 2594.9 ppm as, additionally for (ii): 6487.2 and 16218.0 ppm as, control: basal diet with corn oil only;
Administration: dietary with vehicle,
Vehicle: basal diet with corn oil as adhesive,
Post-treatment: untreated diet;
Observations and examinations: mortality (daily), feed consumption (daily; treatment and post-treatment separately);
Test duration: 5 days exposure with subsequent 3 days of observation; statistics: LC₅₀ and 95 percent confidence limits, slope and standard deviation computed by log probit analysis (Daum and Killcreas, 1966, revised 1976, AVS2007-64).

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Findings:

Mortality: In both species, death occurred on day 1 through 6. The mortality rates are summarised in the following tables:

Table B.9.1-1: Mortality in bobwhite quail

Dietary concentration (ppm)	No. of dead animals at								Total mortality (no. of dead/ no. of animals)
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	
0.0	0	0	0	0	0	0	0	0	0/13
20.0	0	0	0	0	0	0	0	0	0/12 ¹
45.0	0	0	0	0	0	0	0	0	0/13
101.0	0	0	0	0	0	0	0	0	0/13
227.8	0	1	2	0	0	0	0	0	3/13
512.6	0	0	0	1	0	0	0	0	1/13
1153.3	1	0	0	2	2	2	0	0	7/13
2549.9	3	5	3	2	0	0	0	0	13/13

¹ One bird escaped prior to first treatment

Table B.9.1-2: Mortality in mallard ducks

Dietary concentration (ppm)	No. of dead animals at								Total mortality (no. of dead/ no. of animals)
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	
0.0	0	0	0	0	0	0	0	0	0/10
20.0	0	0	0	0	0	0	0	0	0/10
45.0	0	0	0	0	0	0	0	0	0/10
101.0	0	0	0	0	0	0	0	0	0/10
227.8	0	0	0	0	0	0	0	0	0/10
512.6	0	0	0	0	0	0	0	0	0/10
1153.3	0	0	0	0	0	1	0	0	1/10
2549.9	0	1	0	1	0	2	0	0	4/10
6487.2	0	5	4	0	0	0	0	0	9/10
16218.0	7	1	0	1	1	0	0	0	10/10

Signs of toxicity

The few birds observed while succumbing to zinc phosphide poisoning became lethargic and assumed arresting position. However, most birds died over night, and were not observed during dying.

Food consumption

Food consumption was markedly reduced in both bird species at and above 1153.3 ppm. At these concentration levels (1153 - 2594 ppm in quails and 1153 – 16218.0 ppm in mallards) consumption per bird is reduced but to a uniform level in the high dose level groups. According to the EPA evaluation this could be due to an emetic effect of zinc phosphide. In the post-treatment period noticeable differences in feed consumption between control and treatment levels were not observed in bobwhite quail. In mallards, a reduction effect of zinc phosphide on the feed consumption seems to be present in the post-treatment period at and above 1153.3 ppm. Nevertheless evaluation of feed consumption data is difficult due to

mortality in treatment groups. A summary of food consumption is given in Table B.9.1-3 and Table B.9.1-4.

Table B.9.1-3: Food consumption in bobwhite quail

Dietary concentration (ppm)	Treatment (5 days)			Post-treatment (3 days)		
	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)
0.0	327.5	65	5.04	223.5	39	5.73
20.0	311.5	60	5.19	202.0	36	5.61
45.0	316.5	65	4.87	232.5	39	5.96
101.0	348.0	65	5.35	224.0	39	5.74
227.8	305.5	58	5.27	205.0	30	6.83
512.6	320.5	64	5.01	242.5	36	6.74
1153.3	145.0	59	2.46	115.0	20	5.75
2549.9	~ 40 ²	30	~ 1.33 ²	-	-	-

¹ (Number of animals) (days of treatment or post-treatment, respectively)

² value could not be exactly calculated from the reference data due to insufficient copy quality

Table B.9.1-4: Food consumption in mallard ducks

Dietary concentration (ppm)	Treatment (5 days)			Post-treatment (3 days)		
	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)
0.0	3221	50	64.4	3000	30	100
20.0	3245	50	64.9	2944	30	98
45.0	3219	50	64.4	2989	30	100
101.0	3234	50	64.7	2998	30	100
227.8	3213	50	64.3	2996	30	100
512.6	3028	50	60.6	3000	30	100
1153.3	2018	50	40.4	2497	29	86
2549.9	746	45	16.6	1719	20	86
6487.2	297	27	11.0	292	3	97
16218.0	128	18	7.1	0	0	-

¹ (Number of animals) (days of treatment or post-treatment, respectively)

Conclusion:

Based on probit analysis, the median lethal dietary toxicity concentration (LC₅₀) for

- Bobwhite quails (*Colinus virginianus*) was calculated as 849 ppm zinc phosphide (confidence limit: 586 -1292 ppm), equivalent to an LC₅₀ of 224 mg PH₃/kg diet.

- Mallard ducks (*Anas platyrhynchos*) was calculated as 2885 ppm zinc phosphide (confidence limit: 1970–4329 ppm), equivalent to an LC₅₀ of 762 mg PH₃/kg diet.

Zinc phosphide was classified by the U.S. EPA as moderately toxic to quail and slightly toxic to mallards. Due to an emetic effect of zinc phosphide (according to EPA evaluation), food consumption was markedly reduced in both bird species at higher dose levels.

Reference number: IIA 8.1.2
Author: Matschke, G.H.; AVS 2006-180
Title: A Zinc phosphide dietary LC₅₀ study against bobwhite quail.
Date: 1978. Submitted March 15, 1978 under 6704-78 to U.S. EPA by U.S. Dept. of Interior Fish and Wildlife Service, Washington, D.C. CDL: 233244-A U.S.
DocID: Report no.: EPA RED 0: MRID 00006031 (1978)
Guidelines: Acc. to Heath and Stickel (1965) with modifications
GLP: No
Validity: Acceptable

Materials and methods

Test item: Zinc phosphide technical;
Batch: not stated; purity: 94 %;
Test procedure: 5-day dietary toxicity test;
Test animals: bobwhite quail (*Colinus virginianus*), 10-14 days old at test begin (supplier for eggs: Oak Ridge Game Farm, Gravette, Arkansas, USA; rearing at Denver Wildlife Research Center, Colorado; USA),
Number of animals: 15 quails per group;
Housing: each treatment group in one pen within the brooder, temperature: 34 °C, 16-8 hr light-dark cycle, humidity: not controlled;
Body weight: not available;
Dose levels: (constant interval of 154 ppm): 0.00, 381.0, 535.0, 689.0, 843.0, 997.0 and 1151.0 ppm (based on as), Control: basal diet with corn oil only;
Administration: dietary with vehicle:
Vehicle: basal bird diet with corn oil as adhesive,
Post-treatment: untreated diet;
Observations and examinations: mortality (daily), feed consumption (daily; treatment and post-treatment separately);
Test duration: 5 days exposure with subsequent 3 days of observation;
Statistics: LC₅₀ and 95 percent confidence limits, slope and standard error computed by log probit analysis (Daum, 1970, revised 1976, AVS2007-64).

Findings:

Table B.9.1-5: Mortality - dietary toxicity of zinc phosphide to bobwhite quail

Dietary concentration (ppm)	No. of dead animals at								Total mortality (no. of dead/ no. of animals)
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	
0.0	0	0	0	0	0	0	0	0	0/15
381.0	0	0	3	0	3	0	0	0	6/15
535.0	0	1	1	2	4	0	0	0	8/15
689.0	0	1	2	3	3	2	0	0	11/15
843.0	6	2	2	2	2	0	0	0	14/15
997.0	8	3	2	2	0	0	0	0	15/15
1151.0	6	5	1	2	1	0	0	0	15/15

Death began on day one of treatment and continued through days 6. No animals died in the control, and mortality in the two highest dose levels was 100 %.

Food consumption

Table B.9.1-6: Food consumption in bobwhite quails

Dietary concentration (ppm)	Treatment (5 days)			Post-treatment (3 days)		
	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)	Total amount consumed (g)	No. of animal units ¹	Average food consumption per bird (g)
0.0	298.8 ²	60 ³	5.0	335.2	45	7.4
381.0	239.1 ²	54 ³	4.4	208.6	27	7.7
535.0	228.4	68	3.4	111.2	21	5.3
689.0	173.8	65	2.7	79.3	14	5.7
843.0	107.7	39	2.8	24.8	3	8.3
997.0	78.7	28	2.8	-	-	-
1151.0	58.6	32	1.8	-	-	-

¹ (Number of animals) * (days of treatment or post-treatment, respectively)

² Feed consumption for day 3 was omitted because of spillage from one of three feed dishes

³ Animal units for day 3 were omitted because of spillage from one of three feed dishes

During the treatment, feed consumption decreased with increasing zinc phosphide concentration in the diet. The highest concentration level reached the least consumption per bird. In the post-treatment period noticeable differences in feed consumption between control and treatment levels were not observed. Statistical evaluation was not conducted as the birds were fed in groups and values are missing caused by treatment (death of birds).

Conclusion:

The median lethal dietary toxicity concentration (LC₅₀) for zinc phosphide in bobwhite quails (*Colinus virginianus*) calculated by log probit analysis was 468.5 ppm (confidence limit: 355.6-545.8 ppm) with a slope of 5.25 ppm and a standard error of 1.25 ppm. The LC₅₀ is equivalent to 173 mg PH₃/kg diet. Zinc phosphide was classified by the U.S. EPA as highly toxic to bobwhite quails. During the treatment period feed consumption decreased with increasing zinc phosphide concentration in the diet.

Reference number: IIA-8.1.2

Author: Hill, E.F. et al.; AVS 96-00079

Title: Lethal dietary toxicities of environmental pollutants to birds.

Date: 1975

DocID: Special Scientific Report Wildlife No. 191, 1-61, 1975

Guidelines: Not stated

GLP: None (Test was conducted at a time when GLP was not mandatory.)

Validity: Validation not possible, supplemental information

Materials and methods

Test item: Zinc phosphide (technical); batch: not stated; purity: not stated;

Test procedure: 5-day subacute dietary toxicity test;

Test animals: from wild stock of mallard duck (*Anas platyrhynchos*); age: 10 days;

Body weight: not stated;

Dosages: 6 concentrations in the diet (not specified) in a geometrical series;

Diet preparation:	dilution of the test compound in corn oil added to game-bird starter mesh at a ratio of 2:98 (w/w);
No. of animals:	10 birds per dose level;
Controls:	one negative (diet with vehicle) and one positive (Dieldrin);
Examinations:	mortality, clinical signs and food consumption;
Study period:	5 treatment days followed by 3 days without treatment.

The report is a compilation of the results of nearly 10 years of testing the subacute toxicities of pesticides and industrial chemicals to young bobwhites (*Colinus virginianus*), Japanese quail (*Coturnix japonica*), ring necked pheasants (*Phasianus colchicus*) and mallards (*Anas platyrhynchos*).

Findings:

A summary of the results as presented in the original publication is given in the following table.

Table B.9.1-7: Dietary toxicity of zinc phosphide on mallards

Age of mallards ^a	No. of conc. ^b	No. of birds/conc.	LC ₅₀ (ppm) ^c	95 % C.L.	Slope ^d	S.D.	RTD ^e	95 % C.L.
10 days	6	10	1285	1026-1620	3.980	0.944	10.1	7.2-14.6

^aAge of birds at start of test

^bNumber of dietary concentrations used for Probit analysis

^cLC₅₀: ppm compound (based on active substance) in ad libitum diet calculated to produce 50 % mortality

^dSlope: probit on log concentration

^eRelative toxicity of diel drin (RTD) read as : “Dieldrin is x times as toxic as the given compound tested.”

The median lethal dietary concentration (LC₅₀) was determined by Probit analysis to 1285 ppm for mallard ducks in a 5-day dietary sub-acute toxicity test. The authors classified zinc phosphide as slightly toxic (class IV, 1001-5000 ppm) on a scale ranging from I (highly toxic < 41 ppm) to V (practically non-toxic > 5000 ppm).

The dietary LC₅₀ is equivalent to 475 mg PH₃/kg diet.

Conclusion / Comment

The data could not be validated since the report is a compilation of results and no raw data were presented. The results can therefore only be used as supplemental information.

Reference number:	IIA 8.1.2
Author:	Hill, E.F., Camardese, M.B.; AVS 96-00078
Title:	Lethal Dietary Toxicities of Environmental Contaminants and Pesticides to Coturnix.
Date:	1986
DocID:	Fish and Wildlife Technical Report 2. Wash., D.C., U.S. Dep. of Interior Fish and Wildlife Service, pp. 1-20 and 138-142, 1986
Guidelines:	Similar to OECD 205
GLP:	No. (Test was conducted at a time when GLP was not mandatory.)
Validity:	Validation not possible, Literature summary

Materials and methods

Test item:	Zinc phosphide (technical); batch: not stated; purity: 94 %.
Test procedure:	Dietary toxicity of environmental contaminants and pesticides to Japanese quail about 5 days;
Test animals:	Japanese quail (<i>Coturnix japonica</i>); source: in-house breeding; age: 14 days;
Body weight:	not stated;
No. of animals:	15 birds per dose level;
Housing and feeding:	in pens, each group separately, food and water ad libitum;
Test conditions:	20 – 25 °C;
Dose levels:	6 concentrations (not specified) in the range 600 – 2100 ppm in the diet; food consumption: daily.
Controls:	one negative (diet with vehicle) and two positive (dicotophos and dieldrin);
Diet preparation:	dilution of the test compound in corn oil added to game-bird starter, mesh at a ratio of 2:98 (w/w);
Administration:	dietary with basal diet and adhesive corn oil.
Test duration:	5 treatment days followed by 3 days without treatment;
Observations:	clinical symptoms: daily; mortality: daily; body weight: not stated.
Statistics:	Probit analysis for determination of LC ₅₀ values.

Findings:

LC₅₀: 960 ppm / 95 % CI: 824 – 1119 ppm / slope: 6.51 / SE:1.15

Table B.9.1-8: Response chronology for japanese quail

Dietary concentration	Response chronology (day of occurrence)				
	Onset of signs	First death	Last death	Remission of signs	Total mortality
600 ppm	2	3	3	4	1/15
1634 ppm	1	1	4	5	14/15

Table B.9.1-9: Food consumption and total mortality

Dietary concentration	Food consumption (grams per bird-day)					Total mortality
	Day 1	Day 2	Day 3	Day 4	Day 5	
Control (n=3)	11.0	11.5	12.8	11.2	11.8	0/45
600 ppm	9.3	8.8	12.8	11.4	9.1	
Deaths	0	0	1	0	0	1/15
990 ppm	7.1	5.5	8.4	8.1	11.0	
Deaths	1	2	2	3	0	8/15

Conclusion:

The median lethal dietary concentration of zinc phosphide (LC₅₀) was determined by Probit analysis to 956 ppm for Japanese quail in a 5-day dietary sub-acute toxicity test.

The dietary LC₅₀ is equivalent to 354 mg PH₃/kg diet.

B.9.1.3 Subchronic toxicity and reproduction (Annex IIA 8.1.3; Annex IIIA 10.1)

Reference number: IIA 8.1.3
Author: Ikeda, S.; AVS 2002-21
Title: The toxic effect of Zinc phosphide on penned quail, *Coturnix coturnix japonica*.
Date: 1971
DocID: Bull. Gov. For. Exp. Sta. 238, 1971, 141-148
Guidelines: Not applicable
GLP: No
Validity: Validation not possible, Literature summary

Materials and methods

Test item: Zinc phosphide; batch: not stated; purity: not stated;
Test procedure: determination of the fertilisation rate (fertilised eggs per total number of laid eggs) and egg-laying rate (ratio of actual laid eggs to the total number of days);
Test animals: Japanese quail (*Coturnix coturnix japonica*);
Body weight: not stated;
Administration: orally by capsules containing zinc phosphide over a period of 10 days followed by 25 days under normal rearing and further 10 days of treatment;
Test groups: **A)** (1/10 of the LD₅₀ = 3.5 mg/kg) 16 males and 32 females (36-weeks old) divided in four subgroups of 2 controls (normal rearing and empty capsule) and two test groups,
B) (1/50 of the LD₅₀ = 0.7 mg/kg) 18 males and 36 females (18-weeks old) divided in two controls (normal rearing and empty capsule) and 16 test groups;
Housing of animals: one male with two females.

Findings:

Group (A): The fertilisation rate was markedly lower in the test groups than in the control groups given empty capsules or reared under normal conditions. In the two test groups, mortality became apparent after 40 days. The egg-laying rate was reduced in treatment groups compared to controls. However, the egg-laying rate was also reduced by 15 % in quails receiving placebo capsules compared to the normally reared control. This effect was interpreted by the authors as a result of the capsule treatment procedure.

Group (B): The fertilisation rate in the test groups compared to controls showed a drastic decline from day 30 onwards. The egg-laying rate in the test groups and controls was reduced in parallel from day 30 onwards, and rose again to the end of the test period, but the difference between both groups, although lower in the treatment groups, remained within 15 %.

Conclusion:

Oral administration of zinc phosphide at dose levels of 1/10 and 1/50 of the LD₅₀ (3.5 and 0.7 mg/kg) to male and female Japanese quails affected the fertilisation rate and egg-laying rate.

B.9.1.4 Toxicity of the formulation (Annex IIIA 10.1)

Justification for non-submission

No data were specifically generated for the product POLYTANOL. However, according to the risk assessment performed for birds, exposure of birds is very unlikely and restricted to rare individual cases, due to the special conditions of use. Additionally, the results from testing in mammals do not provide any evidence of significantly greater toxicity of the preparation compared to the active substance. For these reasons, the performance of an acute oral toxicity test is not warranted.

B.9.1.5 Other studies (Annex IIIA 10.1.2; 10.1.3; 10.1.4)

B.9.1.5.1 Supervised cage or field trials

Justification for non-submission

According to the risk assessment performed for birds, the GAP use of POLYTANOL is of low risk to birds, due to placing underground into target animal runs and tunnels. Furthermore, POLYTANOL is a very hard granule with a particle size of 2-12 mm, any pick up by birds is not to be expected and it is applied on sites to which birds have no or little access. Investigations on the attractiveness of POLYTANOL for birds are not considered to be sufficient for the prediction of the feeding behaviour of birds on POLYTANOL. Thus, supervised cage or field trials are not considered to be required.

B.9.1.5.2 Acceptance of bait, granules, or treated seeds by birds

Please refer to point B.9.1.5.1.

B.9.1.6 Summary of effects on birds

No studies on birds were performed with calcium phosphide. According to the state of knowledge the toxicity of calcium phosphide is solely caused by release of phosphine (PH_3). Therefore, toxic effects of phosphine and metal phosphides can be considered together and effects of calcium phosphide can be extrapolated from studies with other metal phosphides or phosphine. The presented data are partly taken from literature and these studies do not fully comply with current standards for the testing of plant protection products. Due to missing raw data a validation of the studies is not possible. The studies taken from literature can be used as additional information for the risk assessment. However, the presented data on acute, short-term and long-term effects on birds is considered sufficient for the current question and no further studies are required, since exposure of birds to calcium phosphide containing granules is not expected during the recommended use of POLYTANOL. Additionally, reasons of animal welfare are considered. In the following table, the results of the presented studies are summarised and the effect concentrations are extrapolated to the respective phosphine and calcium phosphide concentrations.

Table B.9.1-10: Toxicity of zinc phosphide in birds ¹⁾

Species	Test-substance	Endpoint	Endpoint ¹⁾ extrapolated to Phosphine	Endpoint ¹⁾ extrapolated to Calcium phosphide	Reference
Acute oral toxicity (mg/kg bw/d)					
<i>Colinus virginianus</i>	Zinc phosphide	LD ₅₀ 25-35	LD ₅₀ 6.6-9.2	LD ₅₀ 17.7-24.7	IIA 8.1.1, (WHO, AVS2005-25))
Short-term, dietary toxicity (mg/kg feed)					
<i>Colinus virginianus</i>	Zinc phosphide	LC ₅₀ 468.5	LC ₅₀ 124	LC ₅₀ 330	IIA 8.1.2 Matschke 1978 (AVS2006-180)
<i>Colinus virginianus</i>	Zinc phosphide	LC ₅₀ 849	LC ₅₀ 224	LC ₅₀ 599	IIA 8.1.2/01 Anonymous 1978 (AVS2006-179)
<i>Anas platyrhynchos</i>	Zinc phosphide	LC ₅₀ 2885	LC ₅₀ 762	LC ₅₀ 2037	IIA 8.1.2/03 Hill et al. 1975 (AVS96-00079)
<i>Anas platyrhynchos</i>	Zinc phosphide	LD ₅₀ 1285	LC ₅₀ 339	LC ₅₀ 907	IIA 8.1.2/04 Hill, Camardese 1986 (AVS96-00078)
<i>Coturnix japonica</i>	Zinc phosphide	LC ₅₀ 960	LC ₅₀ 253	LC ₅₀ 678	IIA 8.1.3, Ikeda 1971 (AVS2002-21)
Long-term, oral (mg/kg body weight)					
<i>Coturnix japonica</i>	Zinc phosphide 1/10 and 1/50 of LD ₅₀ of 35 mg/kg bw	Effects on fertilisation rate and egg- laying rate: 3.5 and 0.7 mg/kg bw	Effects on fertilisation rate and egg-laying rate: 0.92 and 0.18 mg/kg bw	Effects on fertilisation rate and egg-laying rate: 2.47 and 0.49 mg/kg bw	IIA 8.1.3, Ikeda 1971 (AVS2002-21)

¹⁾ calculation of PH₃ (33.98 g/mol) from Zn₃P₂ (258.11 g/mol) using factor 0.264, calculation of Ca₃P₂ (182.15 g/mol) from Zn₃P₂ with factor 0.706

B.9.1.7 Risk assessment

Reference number: IIIA 10.1

Author: Köhler, U.; AVS 2005-29

Title: Risk assessment for birds.

Date: February 8, 2002

DocID: Report no.: AP-ZAA-050927-00

Guidelines: Not applicable

GLP: Not applicable

Validity: Validation not possible, Expert statement

Findings

POLYTANOL is a dry granular solid which is used only underground (15 - 20 cm) with minimal possibilities for access by birds. Contact with mucous membranes and stomach acids will result in rapid evolution of phosphine. By extrapolation from studies with the analogue zinc phosphide it is considered that the acute oral toxicity of calcium phosphide would be in the region of 20 mg Ca₃P₂/kg (Table B.9.1-10). The dietary toxicity of zinc phosphide to turkeys is 150 ppm and to hens 158 ppm (please refer IIA, 8.1.1/02, B.9.2: WHO (1988) Environmental Health Criteria 73 - Phosphine and selected metal phosphide, Genf, 56-59, data not presented in the summary report IIA 8.1.1/02).

Conclusion of the author

POLYTANOL is a dry granular solid which is used only underground (15 – 25 cm) with minimal possibilities for access by birds. The risk of secondary poisoning for birds of prey is negligible. Poisoned voles contain only minimal quantities of phosphine. After inhalation of phosphine, the rapid process of conversion into non-hazardous phosphite and phosphate by oxidising in the voles begins.

B.9.1.7.1 Risk assessment of the active substance

Because of the subterranean application of POLYTANOL, an exposure of birds is unlikely. Gassing operations are normally conducted in areas where burrows can be sufficiently sealed to contain the phosphine. The calcium phosphide containing pellets are applied directly into the burrow systems, after application the hole is closed with a plug (e.g. stone or grass). It is therefore excluded that a bird would swallow the pellets. The evolved phosphine gas is heavier than air and will mainly remain and spread in the burrows. In the unlikely case that gas is escaping the burrows via uncovered holes phosphine will remain close to the ground and the strong smell of garlic, ammonia and carbide will drive off any bird coming near.

Conclusion:

During the recommended use of POLYTANOL, any relevant exposure of birds is not expected. It is concluded that there is no unacceptable risk from the proposed use of POLYTANOL to birds.

B.9.1.7.2 Risk assessment for metabolites

Contact with water will result in evolution of phosphine. According to the state of knowledge the toxicity of calcium phosphide is solely caused by release of phosphine (PH₃).

However, due to the special conditions of use, an exposure of birds is not expected (see B.9.1.7.1).

Conclusion

During the recommended use of POLYTANOL any relevant exposure of birds is not expected. It is concluded that there is no unacceptable risk from the proposed use of POLYTANOL to birds.

B.9.1.7.3 Refined risk assessment

Not required.

B.9.1.7.4 Bioaccumulation and food chain behaviour

The partition coefficient usually used for a first estimation of the bioaccumulation potential of an active substance cannot be determined. The compound decomposes rapidly in n-octanol/water into calcium hydroxide and phosphine. The only degradation product that may be relevant for bioaccumulation is gaseous phosphine. Phosphine residues are expected to be rapidly degraded in tissue. Thus, a potential for bioaccumulation is not expected. Furthermore, from the toxicological studies with mammals there is no indication for bioaccumulation of

phosphine in organisms. Compared to oral or inhalative uptake dermal absorption can be neglected.

Conclusion:

The risk of secondary poisoning for birds of prey is negligible during the recommended use of POLYTONAL. Poisoned voles contain only minimal quantities of phosphine. After inhalation of phosphine the rapid process of conversion in non-hazardous phosphite and phosphate by oxidising in the voles begins.

B.9.2 Effects on aquatic organisms (Annex IIA 8.2; Annex IIIA 10.2)

Calcium phosphide is a dry granular solid which decomposes very rapidly in contact with water to produce calcium hydroxide and phosphine gas. Phosphine is of very low water solubility (0.322 g/L). The use of calcium phosphide containing plant protection products involves laying out of such products in burrow systems. Any exposition of aquatic organisms by contamination of surface waters due to events related in general to pesticide application, such as over-spray, drift, run-off, atmospheric deposition, etc. is not expected. In contrast, a chronic contamination of surface waters is excluded by the specific conditions of use, which prevent a contact of the active substance with water. Furthermore, the insolubility of calcium phosphide in water and the rapid degradation in soil minimises the risk for aquatic organisms. It is therefore concluded that the potential for water contamination by calcium phosphide and phosphine is very small.

B.9.2.1 Toxicity data (Annex IIA 8.2; Annex IIIA 10.2.1)

B.9.2.1.1 Fish - acute toxicity

Reference number: IIA 8.2.1
Author: WHO; WAT 2005-45
Title: Environmental Health Criteria 73 - Phosphine and selected metal phosphide.
Date: 1988
DocID: IPCS International Programme on Chemical Safety, Geneva, Switzerland, p. 53, 1988
Guidelines: Not stated
GLP: No
Validity: Validation not possible, literature summary

Material and methods

Aluminium phosphide, determination of 96 h LC₅₀ value.

Findings

Despite its low solubility, phosphine in solution is acutely toxic. Aluminium phosphide is reported to be highly toxic for the Bluegill sunfish (*Lepomis macrochirus*): 96 h LC₅₀ = 0.178 mg/m³.

B.9.2.1.2 Fish - prolonged toxicity

Justification for non-submission

The submission of data or the performance of a test on chronic toxicity to juvenile fish of the active substance calcium phosphide is not considered to be required, since the use of calcium phosphide containing plant protection products involves laying out of such products in tunnels. Any exposure of fish by contamination of surface waters due to events related in general to pesticides, such as over-spray, drift, run-off, atmospheric deposition etc. is not to be expected. In contrast, a contamination of surface waters is excluded by the specific conditions of use which prevent a contact of the active substance with water.

B.9.2.1.3 Fish life cycle test

Justification for non-submission

The submission of data or the performance of a test on fish life cycle test of the active substance calcium phosphide is not considered to be required, for the reasons explained at the beginning of chapter B.9.2.

B.9.2.1.4 Bioconcentration in fish

Justification for non-submission

The partition coefficient usually used for a first estimation of the bioconcentration potential of an active substance cannot be determined. The compound decomposes rapidly in n-octanol/water into calcium hydroxide and phosphine. The only degradation product that may be relevant for bioconcentration is gaseous phosphine which escapes from water and is decomposed rapidly to ubiquitous phosphate. In addition, phosphine residues, if any, are expected to be rapidly degraded in tissue. Thus, a potential for bioaccumulation of the active substance is not expected, and the performance of a test for bioconcentration in fish is not warranted. The partition coefficient of the metabolite phosphine is estimated to be 0.9 using Zn_3P_2 indicating no potential for bioaccumulation.

B.9.2.1.5 Invertebrates - acute toxicity

Justification for non-submission

The submission of data or the performance of a test on acute toxicity to aquatic invertebrates of the active substance calcium phosphide is not considered to be required, for the reasons explained at the beginning of chapter B.9.2.

B.9.2.1.6 Invertebrates - long-term toxicity

Justification for non-submission

The submission of data or the performance of a test on chronic toxicity to aquatic invertebrates of the active substance calcium phosphide is not considered to be required, for the reasons explained at the beginning of chapter B.9.2.

B.9.2.1.7 Algae

Justification for non-submission

The submission of data or the performance of a test on effects on algal growth and growth rate for the rodenticidal active substance calcium phosphide is not considered to be required, for the reasons explained at the beginning of chapter B.9.2.

B.9.2.1.8 Effects on sediment-dwelling organisms

Justification for non-submission

The submission of data or the performance of a test on sediment dwelling organisms for calcium phosphide is not considered to be required, for the reasons explained at the beginning of chapter B.9.2.

B.9.2.1.9 Aquatic plants

Justification for non-submission

The submission of data or the performance of a test on effects on aquatic plants of calcium phosphide is not considered to be required, since calcium phosphide is not a herbicide, but a rodenticide, and for the reasons given at the beginning of chapter B.9.2.

B.9.2.1.10 Microcosm or mesocosm studies

Justification for non-submission

Not required due to lack of exposure (see beginning of chapter B.9.2).

B.9.2.2 Summary of aquatic toxicity data

A contamination of surface waters is excluded by the specific conditions of use of POLYTANOL which prevent a contact of the active substance with water. Calcium phosphide can not come in contact with water and aquatic organisms due to the special conditions of use which involves laying out of such products in tunnels. Any exposure of aquatic organisms by contamination of surface waters due to events related in general to pesticides, such as over-spray, drift, run-off, atmospheric deposition etc. is not to be expected. The notifier presented information on acute toxicity of aluminium phosphide or phosphine retrieved from literature confirming the high acute toxicity of phosphine which cause the toxicity of calcium phosphide (Table B. 9.2-1).

Table B. 9.2-1: Information on aquatic toxicity of phosphine

Organism type	Species	Test substance	Study type	Endpoint	Toxicity value (µg/L)	Reference
Active substance: aluminium phosphide						
Fish	Bluegill sunfish <i>Lepomis macrochirus</i>	96 h, static	Aluminium phosphide	EC ₅₀	0.178 µg/L	WHO, Environmental Health Criteria 73, WAT2005-45
Fish	Rainbow trout	96 h, static	Aluminium phosphide	EC ₅₀	9.7 x 10 ⁻³ ppm	Anonymous WAT2007-183
Invertebrates	<i>Daphnia magna</i>	24 h	Aluminium phosphide	EC ₅₀	0.200	Anonymous WAT2007-183
Metabolite: phosphine (biologically active)						
Fish	Bluegill sunfish <i>Lepomis macrochirus</i>	96 h, static	phosphine	EC ₅₀	0.105 µg/L ¹⁾	WHO, Environmental Health Criteria 73, WAT2005-45
Fish	Rainbow trout	96 h, static	phosphine	EC ₅₀	0.0056 µg/L ¹⁾	Anonymous WAT2007-183
Vertebrates	Frog	0,5 h	phosphine	EC ₅₀	0.56	WHO, Environmental Health Criteria 73, WAT2005-45
Invertebrates	<i>Daphnia magna</i>	24 h	phosphine	EC ₅₀	0.117 ¹⁾	Anonymous WAT2007-183

¹⁾calculated from values given for AIP

Conclusion:

Since a relevant exposure of surface waters is not expected during the recommended use of POLYTANOL, the existing data base on the toxicity of the active substance to aquatic organisms is considered sufficient, and further testing of the effects on either other aquatic organisms or aquatic plants is not considered to be required predominantly due to a lack of exposure.

B.9.2.3 Risk assessment

Reference number: IIIA 10.2
Author: Köhler, U.; WAT 2005-60
Title: Effects on aquatic organisms.
Date: February 8, 2002
DocID: Report no.: AP-ZAA-050927-00
Guidelines: Not applicable
GLP: Not applicable

Statement

In consideration of the intended GAP use of POLYTANOL as a rodenticide, any relevant exposure of surface water by Polytanol is not expected. POLYTANOL (calcium phosphide) is a dry granular solid which decomposes very rapidly in contact with water to produce calcium hydroxide and phosphine gas. Phosphine is of very low water solubility (0.322 g/L). But it must be pointed out that it is not allowed to use Polytanol to lakes, ponds or watercourses because of the spontaneously reaction of calcium phosphide in water. It is therefore concluded that the potential for water contamination by calcium phosphide and phosphine is very small. Thus, any contamination of surface waters by events related in general to pesticides, such as over-spray, drift, run-off, atmospheric deposition etc. is not to be expected. In contrast, a contamination of surface waters is excluded by the specific conditions of use. Therefore, experimental investigations of the toxicity of this product in aquatic organisms are not considered to be required.

Conclusion:

Since any relevant exposure of surface waters is not expected during the recommended use of POLYTANOL (outdoor use only) it is concluded that there is no unacceptable risk to aquatic organisms from the proposed use of POLYTANOL. Therefore, an estimation of predicted environmental concentrations in surface waters (PEC_{sw}) and consequently in sediments is not considered to be required (see B.8.6.1) and calculations of TER values are unnecessary.

Classification and labelling

Calcium phosphid, active substance:

N, R50: Dangerous to the environment. Very toxic to aquatic organisms

Phosphine, metabolite:

N, R50: Dangerous to the environment. Very toxic to aquatic organisms

Product POLYTANOL (18 % active substance)

N, R50: Dangerous to the environment. Very toxic to aquatic organisms

B.9.3 Effects on other terrestrial vertebrates (Annex IIIA 10.3)**PHOSPHINE**

Reference number: IIA 8.1.1

Author: Klimmer, O. R.; AVS 2005-24, BIO2005-106

Title: Contribution to the Study of the Action of Phosphine (PH₃) - The Question of the So-Called Chronic Phosphine Poisoning.

Date: 1969

DocID: Archiv für Toxikologie (Archives of Toxicology). 24, pp.164-187

Guidelines: Not stated

GLP: No

Validity: Validation not possible, literature summary

Material and methods

Phosphine, source: Phosphonium iodide (manufacturer: Fa. Schuchardt, Munich)

Test animals: Cats, Wistar rats, guinea pigs

In the experiments a gas-tight inhalation chamber having a capacity of 400 L ("Würzburger Modell") was used. The chamber could be aerated. Phosphine/air mixture flowing continuously through the inhalation chamber for 7 hours a day. The phosphine content was analysed before the start of the test and every hour during the inhalation tests ("flow test"). Phosphine was generated in an Erlenmeyer flask by dripping a 10 % KOH solution on pure phosphonium iodide. Phosphine dosage: 1 ppm, 2.5 ppm and 5 ppm respectively.

In the first series of subacute inhalation tests (1 ppm) 4 female cats weighing 2.1 to 2.95 kg and 10 male white Wistar rats having a mean initial weight of 110 g were used.

In the second series (2.5 ppm) 4 female cats (2.2, 2.5, 2.6 and 3.1 kg), 4 female guinea pigs (280 - 360 g) and 10 male wistar rats (weights as in the first series) were used.

In the third series (5 ppm) in 2 subseries a total of 6 white cats (2.6, 2.9, 3.1, 3.3, 2.4, 2.2 kg), 6 female guinea pigs (300 to 360 g) and 20 male Wistar rats (weights as in the first series) were used.

Test duration: Series I (1 ppm): 5 weekdays of 6 hours each, Saturdays 4 hours, total 816 and 650 hours respectively = 24 weeks. Series II (2.5 ppm): same, total 820 hours = 24 weeks. Series III (5 ppm): 8 days per 6 hours each (subseries IIIa) or 8 days per 6 hours each plus 4 days per 8 hours each (subseries IIIb); 48 and 80 hours respectively. Before the start of the tests, at half-time and after the end of the experiments all the experimental animals were subjected to urine analysis (protein, urobilinogen, sugar, sediment) and blood analysis (contents of oxyhaemoglobin and methaemoglobin determined with Beckman differential photometer, osmotic resistance, blood status). The organs fixed in formalin were sectioned by the histopathologist.

Findings:

Series I (1 ppm)

Once having become accustomed to the odor in the test chamber, all the experimental animals behaved completely calmly and, with the exception of two cats having fallen sick intercurrently, exhibited no striking symptoms. The animals were mostly asleep, but reacted at once to touch or acoustic impulses. After the end of the test term they ate with great appetite. The weights of the rats increased from an average of 110 g at the beginning to an average of 318 g at the end of the tests. In the urine of the 4 cats which was obtained by catheterising, no pathological material was found in addition to bacteria and crystals prior to the start of tests and half way between beginning and the end of the tests, while additional traces of proteins were found at the end of the tests only in the pregnant cat. In the urine of the rats all three analyses revealed in addition to the customary bacteria and crystals only individual and transient traces of proteins, without any other pathological components. The blood tests made on the cats at the half-time of the investigation (4 animals) and upon the termination of the test (2 animals) showed normal blood color, and the spectrophotometric investigation revealed the typing oxyhaemoglobin band, without any indication of methaemoglobin. The leucocyte count of the cats was found in all three investigations in the usual variation range of 8,000 to 14,500 which is known from many chronic tests.

The blood test on rats (tail vein) revealed a normal osmotic impedance and impedance range (0.452 to 0.48 and 0.28 to 0.35 % NaCl, respectively). The autopsy of the two cats and ten rats at the end of the tests showed a normal macroscopic aspect and the histological investigation of the most important organs revealed only a slight diffuse globular fatty infiltration in the liver parenchyma of the cats and a slight and isolated fatty infiltration in the adrenocortical system of the rats, a phenomenon encountered with well-fed experimental animals. Besides that, three rats exhibited a slight cloudy swelling of the tubular epithelia. The investigation of the brain section of all animals did not reveal any pathological findings.

Series II (2.5 ppm)

The four female cats, four guinea pigs and ten rats behaved calmly, too, during the whole duration of the test (exposure time 820 hours), showed no striking symptoms, slightly increased in weight on the average, and survived with the exception of two rats which died after 742 and 708 test hours respectively of a proven pulmonary infection. The investigation of urine, blood counts and osmotic impedance of erythrocytes, which was made as in series I prior to the start of the test, at half-time and at the end of the tests, showed as the only divergence a slight and uniform decrease of the erythrocyte and haemoglobin values for three out of four cats by 9 to 14 % toward the end of the tests, a phenomenon known in cats living in captivity, and isolated traces of protein in the rat urines. Guinea pigs and rats did not show the first-mentioned phenomenon. Blood counts, differential blood counts and osmotic impedance were within the normal variation range. The three liver function tests made on each of the four cats showed normal bromsulphthalein excretion in all the cases. The blood showed only the oxyhaemoglobin band and not that of the methaemoglobin, and had a normal color. The section and histological investigation of the organs of all the experimental animals showed again in the liver of the cats a slight diffuse fatty infiltration and an isolated slight cloudy swelling in the tubular epithelia of the rats. The organs of the guinea pigs showed no striking findings. The neuropathological investigation of brain sections of cats, rats and guinea pigs revealed slight and non-specific changes of the Purkinje cells, which, however, were considered as agonal or post-mortem changes.

Series III (5 ppm) /Subseries IIIa (5 ppm)

The 10 rats fell sick, showing poisoning symptoms after a total of 23 to 30 hours inhalation and died during the fifth and sixth tests, after a total of 27 to 36 hours inhalation. All the animals showed a strong hyperemia of the organs, in three of the cases a clear pulmonary edema. The urine at the end of the 4th day of test contained traces of albumen in 7 out of 10 cases. The colour of the blood withdrawn from the aorta immediately after the death of the three cats, three guinea pigs and five rats had a brownish shade and, in the spectrophotometrical investigation, showed only the oxyhaemoglobin band, a fact, however, which does not exclude the possibility of a slight methaemoglobin formation. The histological investigation of the organs of all the animals showed strong blood congestion in the capillaries, congestion in the liver with large vesicular-angular cells; a slight diffuse fatty infiltration was found in rats and guinea pigs, but not in cats. The neurohistological investigation of the brains showed for all the rats a striking dilatation of the perivascular areas, vacuolisation in the nuclei of the ganglion cells and decaying Purkinje cells, respectively.

Series IV

(combination tests) The combination tests showed that 102 hours pretreatment with 1 ppm phosphine (1.4 mg/m³) did not result, in the case of rats, in an injury in the sense of a reduction of the exposure time required for lethal exit. The acute HABER-FLURY Effect

Product [$W = c \text{ (mg/m}^3) \cdot t \text{ (min)}$] for rats not pretreated was, under identical test conditions 18,942 $\text{mg/m}^3 \cdot \text{min}$, for rats pretreated with 1 ppm phosphine 19,040 $\text{mg/m}^3 \cdot \text{min}$.

Conclusion of the author

The experiments were intended to furnish information as to whether or not the MAC presently adopted for phosphine (0.1 ppm = 0.15 mg/m^3 air) could be taken as a realistic one. On the basis of experiments, the author's opinion is that below the limiting range of 5 ppm phosphine, even an exposure of any duration will, in all probability, not result in a chronic poisoning of the experimental animals. The experiments on 3 species of animals have shown that chronic exposure to 1 and 2.5 ppm phosphine does not result in any recognisable disturbance of blood formation, i.e. in shifts of haemoglobin values, erythrocyte and leucocyte counts, and in no disturbance of the osmotic impedance of the erythrocytes. The subacute phosphine poisoning at 5 ppm, however, was accompanied, in some animals, by a decrease of about 50 % in the haemoglobin and erythrocyte counts. Neither during the first three test series, however, nor during the ultimate acute tests on several animal species the authors could establish a noticeable formation of methaemoglobin.

Comment of RMS

According to the state of knowledge the toxicity of calcium phosphide is solely caused by the release of phosphine (PH_3). Therefore, toxicities of phosphine and metal phosphides can be considered together and effects of calcium phosphide can be extrapolated from studies with other metal phosphides and phosphine. The data presented above are taken from literature. They allow conclusions on the effects of the compound. However, the studies do not fully comply with current standards for the testing of plant protection products and should be used as additional information for the risk assessment.

B.9.3.1 Summary of terrestrial vertebrate acute and reproductive studies

B.9.3.1.1 Effects of calcium phosphide and the product

Toxicity data was generated with rodents. Details of mammalian toxicity studies with POLYTANOL or calcium phosphide are provided in the chapter B.6 (Toxicology and metabolism). Results of the individual acute and long-term studies are summarised in Table B.9.3-1.

Table B.9.3-1: Summary of acute and long-term toxicity data for calcium phosphide and the product

Type of study	Animal species	Test substance	Endpoint	Reference
Acute oral	rats	Product Polytanol (17.6 % as)	LD_{50} 72.32 mg Polytanol/kg body weight	Rao, K. Venugopala (1999) IIA 5.2.1 (TOX2000-105)
Acute Inhalation (4 h)	rats	Product Polytanol (17.6 % as)	LD_{50} 0.090 mg PH_3/L	Rao, K. Venugopala (2001) IIA 5.2.3 (TOX2002-822)

Type of study	Animal species	Test substance	Endpoint	Reference
Acute Inhalation (4 h)	rats	Metabolite PH ₃	0.015 mg/L air / 11 ppm 2.8 mg PH ₃ /kg bw	Waritz & Brown (1975) IIA 5.2.3 (TOX2002-828)
2-years dietary study	rats	Metabolite PH ₃ generated from AIP	No adverse effects at average residual phosphine levels 5 ppb in diet (2000 ppm PH ₃ during diet fumigation)	Telle et al. (1985) IIA 5.5 (TOX2002-831)
2-generation inhalation study	rats	Metabolite PH ₃	NOAEC 3 ppm (air) (1.13 mg PH ₃ /kg bw/day)	Newton et al. (1999) IIA 5.5 (TOX2002-189)
2-generation dietary reproduction study	rats	Metabolite PH ₃	No adverse effects fed with diet fumigated with 2000 ppm PH ₃	Cabrol et al. (1986) IIA 5.6.1 (TOX2005-189)

B.9.3.2 Other studies

B.9.3.2.1 Supervised cage or field trials

As demonstrated in the risk assessment below and discussed above, a risk to non-target animals is restricted to rare and individual cases, which is why TER values covering the risk for whole communities must be expected above the thresholds of 10 (TER_a and TER_{st}) and 5 (TER_{lt}), respectively, as stated in Annex VI of Directive 91/414/EEC. Therefore, such studies are not considered to be required.

B.9.3.2.2 Acceptance of bait, granules or treated seeds by terrestrial vertebrates

Please refer to the risk assessment for terrestrial vertebrates and arguments from above.

B.9.3.3 Summary of results used in the risk assessment

The special conditions of use minimise the possibility that non-target terrestrial vertebrates other than birds come into contact with calcium phosphide or phosphine gas. Therefore, a risk assessment is not necessary (see below).

B.9.3.4 Risk assessment

Reference number: IIIA 10.2
Author: Köhler, U.; AVS 2005-30
Title: Effects on terrestrial vertebrates other than birds.
Date: February 8, 2002
DocID: Report no.: AP-ZAA-050927-00
Guidelines: Not applicable
GLP: Not applicable
Validity: Validation not possible, expert statement

Findings:

Other routes of exposure: Since Polytanol is applied underground as a rodenticide, other routes of exposure are not considered relevant.

Statement

Due to the GAP use of POLYTANOL, placing the product underground into target animal runs and tunnels, no short- or long-term risk from dietary exposure is indicated for terrestrial vertebrates. With the exception of rodents, an acute risk arising from consumption of POLYTANOL can also be excluded due to the low attractiveness and the hardness of the granule. Only a theoretical risk arising from spilled material cannot totally excluded, but was shown to be very unlikely, because of the disagreeable, garlic smell of the product. The risk for secondary poisoning was demonstrated to be negligible, and no risk is given from other routes of exposure.

Conclusion:

Due to the specific application method of calcium phosphide containing formulations as gassing products on agricultural land the hazard of non target mammals, reptiles and amphibians can principally not be excluded. These products are applied as pellets or tablets to the underground tunnels of the target organisms (*Arvicola terrestris*, *Talpa europaea* (mole)). Other organisms using these tunnels as a part of their habitat (*Mustela nivalis* (least weasel)) or living in similar holes in the same habitat (*Spermophilus* (gopher), *Cricetus cricetus* (hamster)) are potentially endangered. Exposure by spills is only possible for a short-time, since calcium phosphide reacts quickly with water (humid soil, air moisture) to phosphine and apatite, calcium phosphate and calcium carbonate. The evolved phosphine gas is heavier than air and will mainly remain and spread in the burrows. In the unlikely case that gas is escaping the burrows via uncovered holes phosphine will remain close to the ground and the strong smell of garlic, ammonia and carbide will drive off any bird or other animals coming near. A primary poisoning of animals living outside the tunnel system is therefore considered unlikely. Organisms inhabiting burrows, e.g. badgers, foxes and adders could be affected; therefore areas where these animals can be expected must not be treated. Under certain registrations this is a condition of approval.

However, during the recommended use of the calcium phosphide containing formulation POLYTANOL any relevant exposure of terrestrial vertebrates outside the burrows is not expected. It is therefore concluded that there is no unacceptable risk from the proposed use of POLYTANOL to non-target terrestrial vertebrates.

B.9.3.4.1 Bioaccumulation and food chain behaviour

The partition coefficient usually used for a first estimation of the bioaccumulation potential of an active substance cannot be determined. The compound decomposes rapidly in n-octanol/water into calcium hydroxide and phosphine. The only degradation product that may be relevant for bioaccumulation is gaseous phosphine. Phosphine residues are expected to be rapidly degraded in tissue. Thus, a potential for bioaccumulation is not expected. Furthermore, from the toxicological section (B.6) there is no indication for bioaccumulation of phosphine in organisms. Compared to oral or inhalative uptake dermal absorption can be neglected.

Conclusion:

The risk of secondary poisoning for terrestrial vertebrates is negligible during the recommended use of POLYTONAL. Poisoned voles contain only minimal quantities of phosphine. After inhalation by the voles, phosphine is rapidly oxidised to the non-hazardous phosphite and phosphate.

B.9.4 Effects on bees

B.9.4.1 Acute toxicity (Annex IIA 8.3.1, Annex IIIA 10.4)

B.9.4.1.1 Acute oral and contact toxicity of technical aluminium-phosphide (Annex II A; 8.3.1)

Not required.

B.9.4.1.2 Acute oral and contact toxicity of formulated aluminium-phosphide to honeybees (Annex III A; 10.4)

Not required.

B.9.4.2 Bee brood feeding test (Annex IIA 8.3.1.2)

Not required as the test substance is not an IGR.

B.9.4.3 Residue test (Annex IIIA 10.4.2)

Not required.

B.9.4.4 Cage test (Annex IIIA 10.4.3)

Not required.

B.9.4.5 Field test (Annex IIIA 10.4.4)

Not required.

B.9.4.6 Tunnel test (Annex IIIA 10.5.5)

Not required.

B.9.4.7 Risk assessment for honeybees

Bees will not be exposed when calcium-phosphide is used in the field for control of *Arvicola terrestris* and *Talpa europaea*. Therefore no data are required.

B.9.5 Effects on other arthropods species (Annex IIA 8.3.2; Annex IIIA 10.5)

Justification for non-submission

The submission of data or the performance of a test on effects on other non-target terrestrial arthropods of calcium phosphide is not considered to be required, since calcium phosphide is a rodenticide which is laid out only on discrete sites in tunnels, and is not a subject to broadcast or widespread application to soil.

B.9.5.1 Acute toxicity (Annex IIA 8.3.2; Annex IIIA 10.5.1)

Not required for reasons given in B.9.5 above.

B.9.5.1.1 Laboratory tests

Not required for reasons given in B.9.5 above.

B.9.5.2 Extended studies (Annex IIA 8.3.2; Annex IIIA 10.5.1)

B.9.5.2.1 Extended laboratory studies

Not required for reasons given in B.9.5 above.

B.9.5.2.2 Field tests

Not required for reasons given in B.9.5 above.

B.9.5.3 Summary of toxicity data on arthropods other than bees

The special conditions of use exclude the possibility that non-target arthropods come into contact with calcium phosphide or phosphine gas. Therefore, toxicity studies are not necessary for reasons given in B.9.5 above.

B.9.5.4 Risk assessment

The special conditions of use exclude the possibility that non-target arthropods come into contact with calcium phosphide or phosphine gas. Therefore, a risk assessment is not necessary for reasons given in B.9.5 above.

Conclusion:

On the basis of experimental data no risk assessment can be performed. Nevertheless, the risk is assumed to be acceptable for the following reasons: No risk is expected, as fumigation takes place only in the burrows of the target-animals and the fumigated area is very small compared to the field area.

B.9.6 Effects on earthworms (Annex IIA 8.4; Annex IIIA 10.6.1)

Since fumigation takes place only in the burrows of the target-animals, the fumigated area is very small compared to the whole field. Additionally phosphine diffusion into the soil will not be very high and the gas is transformed with a very short half-life into non-toxic phosphates. Due to the reasons mentioned above only a very small quantity of earthworms will be possibly affected from fumigation and possible mortality will be equalised very quickly (eggs of the earthworm, which are laid inside a cocoon, will no doubt survive). Therefore the submission of data or the performance of a test on acute toxicity to earthworms is not considered to be necessary and has never been required for registration of metal phosphide-containing products in any country. Nevertheless, an acute toxicity test with the product POLYTANOL was performed.

B.9.6.1 Acute toxicity (Annex IIA 8.4.1; Annex IIIA 10.6.1.1)**PRODUCT POLYTANOL**

Reference number: IIA 8.4.1
Author: Grunert, B.; ARW 2005-25
Title: Toxizität für Regenwürmer.
Date: 1991
DocID: Report no. 91 20 43 031, unpublished report, BioChem GmbH, Karlsruhe, Germany
Guidelines: OECD 207 (1984)
GLP: Yes
Validity: Acceptable

Materials and methods

Test substance: Polytanol, Charge: 1411/April/1991 purity: technical, calcium phosphide as 28 %, manufacturer: Chemische Fabrik Wülfel, Hannover, Germany Test organisms: 10 adult earthworms (*Eisenia foetida*) Test substrate concentration: 10 % Sphagnumtorf, 20 % Kaolinitkreide, 69 % Industriequarzsand, 1 % Calciumcarbonat; test substrate: Moisture: 28 %; test substrate (dry weight): 500 g; Vehicel: no; concentration of Polytanol: 100 mg/kg test substrate (dry weight), 400 mg/kg test substrate (dry weight)

Findings:**Table B.9.6-1: Results of the acute toxicity to earthworms: Mortality in earthworms**

Concentration	Control day 1	Control day 7	Control day 14
100 mg/kg (container 1)	0	0	0
100 mg/kg (container 2)	0	0	0
400 mg/kg (container 1)	0	0	0
400 mg/kg (container 2)	0	0	0

Conclusion:

During the 14-day test period with a concentration of 100 mg/kg and 400 mg/kg POLYTANOL no mortality in earthworms was observed. No pathological symptoms or changes in behaviour were observed in surviving worms. Even when only two replicates were applied, the validity is accepted since no effect occurred.

B.9.6.2 Sublethal effects (Annex IIA 8.4.2; Annex IIIA 10.6.1.2)**Justification for non-submission**

The submission of data or the performance of a test on sub-lethal effects on earthworms is not considered to be required due to the reasons mentioned above (2.6).

B.9.6.3 Field study (Annex IIIA 10.6.1.3)

Not performed, not required.

B.9.6.4 Summary of toxicity data on earthworms

No acute toxicity up to a concentration of 400 mg POLYTANOL per kg soil (112 mg as/kg) occurred for earthworms.

B.9.6.5 Risk assessment**Exposure**

Since fumigation takes place only in the burrows of the target-animals, the fumigated area is very small compared to the whole field. Phosphine diffusion into the soil will not be very high and the gas is transformed with a very short half-life into non-toxic phosphates.

Risk assessment

Only a very small quantity of earthworms will be affected from fumigation and mortality will be equalised very quickly.

Conclusion:

During the recommended use of POLYTANOL any relevant exposure of earthworms outside the burrows is not expected. It is concluded that there is no unacceptable risk from the proposed use of POLYTANOL to the earthworm population.

B.9.7 Effects on other soil non-target macro-organisms (Annex IIIA 10.6.2)

PRODUCT POLYTANOL

Not performed, not required.

B.9.8 Effects on soil non-target micro-organisms (Annex IIA 8.5; Annex IIIA 10.7)

B.9.8.1 Laboratory testing

CALCIUM PHOSPHIDE TECHNICAL

Reference number: IIA 8.5, IIIA 10.7.1
Author: Dresbach, C.; BMF 2005-14
Title: Berichterstattung über die Auswirkungen von Polytanol auf die Aktivität der Bodenmikroflora.
Date: 1990
DocID: Landwirtschaftliche Untersuchungs- und Forschungsanstalt, Bonn, Germany, unpublished report, September 17, 1990
Guideline: BBA VI, 1-1 (1987)
GLP: No, but conform to GLP
Validity: Not acceptable

Material and methods

Test substance: Calcium phosphide technical, as 28 % Ca_3P_2 ; application rate: 5 g/m² (66,5 mg/kg of dry soil); test soils: 3 replicates/treatment of 2 mm (soil 1) and 5 mm (soil 2) sieved natural soils from agricultural fields: soil 1: silty sand (4.8 % loam, 6.9 % silt, 88.3 % sand), pH 6.0, 1 % humus content; soil 2: loamy silt (8.8 % loam, 59.4 % silt, 31.8 % sand), pH 6.5, 2.4 % humus content; control: untreated soils; reference substance: Aretit flüssig applied at 16 µL/kg (soil 1) and 40 µL/ha (soil 2); test conditions: 22 °C room temperature, soils adjusted to 30 % (soil 1) and 60 (soil 2) moisture holding capacity; test procedure: single application by mixing; duration: 28 days and 70 days; test method/analytical verification: (i) dehydrogenase activity: conversion of triphenyltetrazoliumchloride to triphenylformazane (TPF); (ii) nitrogen turnover: determination of ammonium, nitrite and nitrate concentrations.

Findings:

Dehydrogenase activity

As summarised in tabular format below, the test substance had no marked effect on the metabolic activity of the microbial biomass in both soils.

Table B.9.8-1: Effect of the test substance on dehydrogenase activity in soil 1 (dehydrogenase activity \pm SD [mg TPF/100 g soil dry matter equivalent])

Test group	Day 0	Day 7	Day 14	Day 28	Day 42	Day 56	Day 70
Control	4.27 \pm 0.26	5.00 \pm 0.20	3.91 \pm 0.30	4.17 \pm 0.32	4.32 \pm 0.14	3.81 \pm 0.29	3.09 \pm 0.23
Test substance	. / .	3.72 \pm 0.18	3.12 \pm 0.43	3.27 \pm 0.16	3.14 \pm 0.13	3.03 \pm 0.12	1.97 \pm 0.18
Test substance to control (%)	. / .	-25,6	-20,2	-21,58	-27,31	-20,47	-36,25
Reference substance	2.47 \pm 0.17	1.96 \pm 0.06	1.23 \pm 0.29	1.53 \pm 0.03	1.36 \pm 0.21	0.93 \pm 0.19	0.61 \pm 0.10
Reference substance to control (%)	- 42,15	-60,80	-68,54	-63,31	-68,52	-75,59	-80,26

Table B.9.8-2: Effect of the test substance on dehydrogenase activity in soil 2 (dehydrogenase activity \pm SD [mg TPF/100 g soil dry matter equivalent])

Test group	Day 0	Day 7	Day 14	Day 28	Day 42	Day 56	Day 70
Control	8.89 \pm 0.84	11.55 \pm 0.88	10.27 \pm 1.44	11.48 \pm 0.35	11.81 \pm 0.30	8.35 \pm 0.21	5.36 \pm 0.50
Test substance	. / .	11.20 \pm 1.17	9.49 \pm 0.13	10.04 \pm 0.22	10.83 \pm 0.15	7.72 \pm 0.06	5.35 \pm 0.19
Test substance to control (%)	. / .	-3,03	-7,59	-12,54	-8,30	-7,54	-0,19
Reference substance	7.43 \pm 0.27	8.42 \pm 0.87	6.95 \pm 0.34	7.02 \pm 0.71	7.32 \pm 0.71	5.37 \pm 0.08	2.81 \pm 0.18
Reference substance to control (%)	- 16,42	-27,10	-32,23	-38,85	-38,02	-35,69	-47,57

Nitrogen turnover

The test substance had no remarkable effect on nitrogen turnover in both soils. The rapid decrease of ammonium-nitrogen within the first 14 days after study initiation was accompanied by a subsequent increase in soil nitrite/nitrate concentration. All deviations were at or below 15 % as compared to the control. For further information please refer to the following tables.

Table B.9.8-3: Effects on nitrogen turnover in soil 1 (content of inorganic nitrogen [mg/100 g dry soil])

Test group	Nitrogen compound	0 days	14 days	28 days
Control	A	0.22 ± 0.04	0.14 ± 0.03	0.03 ± 0.00
	B	2.40 ± 0.02	3.82 ± 0.22	3.67 ± 0.19
	C	2.62 ± 0.04	3.96 ± 0.21	3.70 ± 0.19
Test substance	A	./.	0.35 ± 0.03	0.03 ± 0.00
	B		3.58 ± 0.03	4.33 ± 0.41
	C		3.93 ± 0.46	4.36 ± 0.40
Test substance to control (%)	A	./.	+ 150,0	±0,00
	B		- 6,28	- 17,98
	C		- 0,76	+ 17,84
Reference substance	A	0.24 ± 0.03	0.86 ± 0.37	0.13 ± 0.01
	B	2.29 ± 0.10	4.18 ± 0.49	5.65 ± 0.12
	C	2.53 ± 0.08	5.04 ± 0.19	5.78 ± 0.12
Reference substance to control (%)	A	+ 9,09	+ 514,29	+ 333,33
	B	- 4,48	+ 9,42	+ 53,95
	C	- 3,44	+ 27,27	+ 56,22

A = ammonium nitrogen, B = nitrate and nitrite nitrogen, C = A + B

Table B.9.8-4: Effects on nitrogen turnover in soil 2 (content of inorganic nitrogen [mg/100 g dry soil])

Test group	Nitrogen compound	0 days	14 days	28 days
Control	A	0.10 ± 0.02	0.12 ± 0.04	0.03 ± 0.00
	B	7.87 ± 0.02	9.08 ± 0.18	8.41 ± 0.73
	C	7.97 ± 0.00	9.20 ± 0.16	8.44 ± 0.73
Test substance	A	./.	0.19 ± 0.01	0.03 ± 0.00
	B		8.97 ± 0.32	8.96 ± 1.62
	C		9.16 ± 0.31	8.99 ± 1.61
Test substance to control (%)	A	./.	+58,33	±0,00
	B		- 1,21	+ 6,54
	C		- 0,43	+ 6,52
Reference substance	A	0.47 ± 0.09	0.38 ± 0.08	0.13 ± 0.02
	B	7.39 ± 0.10	10.46 ± 0.18	9.59 ± 0.02
	C	7.86 ± 0.09	10.84 ± 0.20	9.72 ± 0.21
Reference substance to control (%)	A	+ 370,00	+ 216,67	+ 333,33
	B	- 6,10	+ 15,20	+ 14,03
	C	- 1,38	+ 17,83	+ 15,17

A = ammonium nitrogen, B = nitrate and nitrite nitrogen, C = A + B

Conclusion:

The validity of the study is considered to be not acceptable. The moisture of soil 1 during the test was too low (30 % of Water Holding Capacity). According to the BBA Guideline the moisture has to be within the range of 40 – 60 % of WHC. The continuous inhibition of the dehydrogenase activity of 20 – 36 % in the course of the test duration of 100 days could be explained by microbial stress due to dryness and is not necessarily an effect of the test item (no relevant effect in soil 2 with 60 % WHC). Therefore, the results of the dehydrogenase study with soil 1 is not plausible. Nitrogen turnover was not influenced by calcium phosphide technical at a concentration of 66.5 mg/kg of dry soil equivalent to a rate of 5 g/m² hypothetically applied to the soil.

The applied concentration is clearly above the maximum amount that theoretically may be introduced into soil by the envisaged use for calcium phosphide. Hence, soil non-target micro-organisms are not considered to be adversely affected by the use of calcium phosphide as a rodenticide. Therefore, no further information is required.

B.9.8.2 Additional testing

No additional testing is triggered.

B.9.8.3 Summary of results and risk assessment

Table B.9.8-5: Summary of ecotoxicological results with calcium phosphide

Test objectives	Test system	Results
Soil micro-organisms		
Soil micro-organisms	dehydrogenase activity nitrogen turn over	no irreversible negative effects

Overall summary and conclusion

The results of the assessment for the product POLYTANOL with the active substance calcium phosphide evolving the biologically active component phosphine gas indicate that no adverse impact on soil non-target micro-organisms and their function in soil systems is expected from the recommended use of POLYTANOL.

B.9.9 Effects on other non-target organisms (flora and fauna) believed to be at risk (Annex IIA 8.6)

ACTIVE SUBSTANCE and FORMULATION

Justification for non-submission

Fauna

All available information on studies with non-target-organisms is presented above. Further studies with calcium phosphide on primary and secondary poisoning are not considered to be required due to the following reasons:

Primary poisoning

Calcium phosphide containing products are used against animals living in underground tunnel systems. The correct way of applying these products is to place the product in the tunnel system and afterwards close the hole to the tunnel system with a stone, grass or a piece of paper. It is therefore excluded that an animal would swallow the product. According to the current state of knowledge the toxicity of calcium phosphide is solely caused by release of phosphine (PH_3). The evolved phosphine gas is heavier than air and will mainly remain and spread in the burrows. In the unlikely case that gas is escaping the burrows via uncovered holes phosphine will remain close to the ground and the strong smell of garlic, ammonia and carbide will drive off any animal coming near. A primary poisoning of animals living outside the tunnel system is therefore considered unlikely.

Organisms which co-inhabit the tunnel systems could be affected, but all studies with the species in question are presented in the chapters above or, if not, reasons for non-submission are given.

Secondary poisoning

Carnivorous and scavenging terrestrial vertebrates are identified theoretically at risk from consumption of intoxicated target animals. However, adverse or lethal effects on these species are considered to be of minor relevance since phosphine does not accumulate in the target animals but is metabolised to non-toxic phosphates rapidly.

Flora

Calcium phosphide containing products are not intended for direct application to growing crops. The application of the products in underground tunnel systems excludes direct contact with plants. The decomposition products of calcium phosphide are approx. 80 - 90 % hydroxyl-apatite, approx. 5 % calcium phosphate, approx. 5 % polyphosphoric acids and approx. 5 % calcium hydroxide and calcium carbonate. These complexes are widely found in nature. In tests with polytanol on cress, no harmful effect was determined, indeed better plant growth was observed (fertilising effect). The evolved phosphine gas will spread and remain in the burrows; the only imaginable way for uptake should therefore be through the roots, which will be minimal. The phosphine gas is finally transformed with a very short half-life into phosphorous compounds (phosphates), which are not toxic but even fertilising. Phytotoxic effects during or after the use of metal phosphides in underground tunnel systems have never been observed and reported and it can be therefore concluded that the use of metal phosphides causes no risk for growing plants, hence tests are not considered to be required.

Conclusion:

During the recommended use of POLYTANOL any relevant exposure of non-target organisms (flora and fauna) - beside these organisms which co-inhabit the tunnel systems - is not expected. It is concluded that there is no unacceptable risk from the proposed use of POLYTANOL to non-target populations.

B.9.10 Effects on biological methods of sewage treatment (Annex IIA 8.7)

PHOSPHINE

Reference number: IIA 8.7

Author: Glindemann, D. et al.; WAT 2003-238

Title: Spontaneous emission of phosphine from animal slurry treatment processing.

Date: 1995

DocId: Zentr. Hyg. Umweltmed. 198, pp.49-56, 1995

Guideline: Not applicable

GLP: No

Validity: Validation not possible; literature

Materials and methods

Gas samples from different sources of animal slurry were analysed regarding their phosphine emission. Dimethyl disulfide (DMDS) concentrations were determined to detect any correlation between evolution of both gases. Trace analysis of phosphine was carried out by gas chromatography with a thermoionic nitrogen phosphorous detector. Concentration of DMDS was determined with a gas chromatograph equipped with flame photometric detection in sulphur modus. Observed plants were: Cattle slurry and biogas processing, pig slurry and biogas processing, pig slurry and simple storage processing.

Findings:

The maximum concentration was detected in putrefaction gas with 14621 ppt (v/v). The correlation of phosphine and DMDS concentrations indicates that primary lytic processes play a role in the liberation of phosphine. Fluxes and concentrations in open basins are significantly higher during summer than in winter.

Comment of the notifier

During sewage treatment as well as from the anaerobic biosphere (i.e., marsh gas, aquatic sediments) phosphine is released into atmosphere at low levels. Since the amount in manure is higher than in feed, phosphine residues in feed can be excluded as an explanation for this phenomenon. Obviously, fermentation processes are not effected negatively by the released phosphine amounts.

Conclusion:

The detection of phosphine in gas samples from mainly anaerobic stages on animal slurry processing or in gas samples from the anaerobic biosphere cannot be used as argument that there is no toxicity to microorganisms in waste water treatment plants.

Justification:

- I. Although biotic processes were studied, phosphine may be of abiotic origin. Therefore the line of argument "detection in biotic processes - therefore, formation by microorganisms - therefore un toxic" cannot be accepted.
- II. If phosphine is of biotic origin in the cited studies, a formation at anaerobic conditions has to be assumed. In contrast, the degradation process in waste water treatment plants is usually aerobic. The sensitivity of an aerobic microbial population concerning phosphine may be different to the sensitivity of a microbial population present at anaerobic conditions.
- III. It is well known that microbial processes can be inhibited by microbial degradation products (e.g. nitrification: nitrite at critical concentrations inhibits formation of nitrite from ammonium). Therefore, the observation of a biotic formation of substances cannot be used as indicator of "no toxicity".

Reference number: IIA 8.7

Author: Glindemann, D. et al.; WAT 2003-237

Title: Free phosphine from the anaerobic biosphere.

Date: 1996

DocId: Environ. Sci. Pollut. Res. 3, pp. 17-19, 1996

Guideline: Not stated

GLP: No

Validity: Validation not possible; literature

Materials and methods

Gas samples from different sources supposed to be phosphine emitters were analysed: Biogas from a digester or fermentation process, putrefaction gas from open basins or tanks, landfill gas, interstitial gas from the anaerobic part of solid waste and marsh gas from methanogenesis in aquatic sediments. Trace analysis in the samples was carried out by gas chromatography.

Findings:

Maximum emission concentrations of phosphine in the closed working atmosphere near the sources was 41 ppt (domestic sewage plant, sludge drying process, near centrifuge). Phosphine is universally present in a variety of gases emitted from the biosphere. Landfills liberated the highest concentrations and fluxes. Putrefaction processes in open basins also liberated high concentrations but low fluxes. German digester gas contained about 6 orders of magnitude less than Hungarian samples. This could be due to the more efficient phosphate elimination procedure in German sewage plants.

POLYTANOL (CALCIUM PHOSPHIDE)

Reference number:	IIA 8.7
Author:	Köhler, U.; WAT 2005-46
Title:	Auswirkungen auf die biologische Abwasseraufbereitung.
Date:	1999
DocId:	Report no.: AP-ZA 004441-00
Guideline:	Not stated
GLP:	No
Validity:	Not possible to validate; expert statement

Statement

Effects on biological effluent treatment:

Polytanol pellets must not be used in immediate proximity to water. Application is subterranean, the decomposition products are calcium hydroxide, calcium phosphite and calcium phosphate. These complexes are partly water soluble and can only enter the water cycle by leaching. The concentration is then very low. Extinguishing water must not be used in accidents or fire with Polytanol, so that the effluent water is not contaminated with hydrolysis products. Should calcium phosphide, in “worst case” enter effluent, pH is shifted. If anaerobic or aerobic phosphorus elimination takes place in biological treatment plant, neutralisation of the effluent should occur. Furthermore, calcium hydroxide is used as a phosphate additive, in the form of calcium phosphate, in treatment plants. Calcium salts are important as nutrient medium for micro-organisms in biological processes. We therefore do not consider testing according to GLP necessary.

Overall conclusion

During the recommended use of POLYTANOL any relevant exposure of biological waste water treatment organisms is not expected. It is concluded that there is no unacceptable risk from the proposed use of POLYTANOL to the efficiency of sewage treatment plants.

B.9.11 References relied on

Annex point/ reference number	Author(s)	Year	Title source (where different from company) report no. GLP or GEP status (where relevant), published or not BVL registration number	Data protection claimed Y/N	Owner
AIIA-8.1.1	Blaxland, J.D., Gordon, R.F.	1945	Zinc phosphide poisoning in poultry. The veterinary Journal, 101, 108-110 not GLP, published AVS2007-63	N	-
AIIA-8.1.1	Klimmer, O.R.	1969	Contribution to the study of the action of phosphine (PH ₃) the question of the so-called chronic phosphine poisoning. Arch.Toxi-kol., 24, 1969, 56-59 not GLP, published AVS2005-24	N	-
AIIA-8.1.1	Shivanandappa, T.	1979	Rodenticidal Poisoning of non target animals: Acute oral toxicity of zinc phosphide to poultry. Bull.Environm.Contam., 23, 452-455 not GLP, published AVS2007-65	N	-
AIIA-8.1.1	WHO	1988	Environmental Health Criteria 73 - Phosphine and selected metal phosphides. Chemical Safety, 56-59 not GLP, published AVS2005-25	N	-
AIIA-8.1.1 AIIA-8.1.4	Ikeda, S.	1971	The toxic effect of Zn ₃ P ₂ (zinc phosphide) on penned quail, Coturnix coturnix japonica. not GLP, published AVS2002-21	N	
AIIA-8.1.2	Anonymous	1978	A Zinc phosphide dietary LC ₅₀ study against bobwhite quail and mallard ducks. Dept. of Interior, Fish and Wildlife service, 1- 10 not GLP, published AVS2006-179	N	-
AIIA-8.1.2	Daum, R.J., Killcreas, W.	1966	Two computer programs for probit analysis. Bull. Entomol.Soc.Am, 12, 4, 1966, 365-369 not GLP, published AVS2007-64	N	-
AIIA-8.1.2	Hill, E.F., Ca- mardese, M.B.	1986	Lethal dietary toxicities of environmental con- taminants and pesticides to Coturnix. USDI Fish Wildl Service, Fish Wildlife Tech- nical Report , 2, 1986 not GLP, published AVS96-00078	N	-

¹ Only notifier listed

Annex point/ reference number	Author(s)	Year	Title source (where different from company) report no. GLP or GEP status (where relevant), published or not BVL registration number	Data protection claimed Y/N	Owner ¹
AIIA-8.1.2	Matschke, G.H.	1978	A Zinc phosphide dietary LC ₅₀ study against bobwhite quail. Dept. of Interior, Fish and Wildlife service, 1-10 not GLP, published AVS2006-180	N	-
AIIA-8.1.3	Hill, E.F. et al.	1975	Lethal dietary toxicities of environmental pollutants to birds. not GLP, published AVS96-0007975	N	CFW
AIIA-8.2.1; AIIIA-10.2.1	Anonymus	2005	Health and safety Data Sheet acc. 91/155 and 93/112 EEC, Detia sachet-roll. not GLP, unpublished WAT2007-183	N	CFW
AIIA-8.2.1	WHO	1988	Environmental Health Criteria 73-Phosphine and selected metal phosphides. Chemical Safety not GLP, published WAT2005-45	N	-
AIIA-8.7	Glindemann, D., Bergmann, A.	1995	Spontaneous emission of phosphine from animal slurry treatment processing. Zentr.Hyg Umweltmed., 198, 49-56 not GLP, published WAT2003-238	N	-
AIIA-8.7	Glindemann, D. et al	1996	Free phosphine from the anaerobic biosphere. Environ.Sci.Pollut., 3, 17-19 not GLP, published WAT2003-237	N	-
AIIA-8.7	Köhler, U.	1999	Effects on biological effluent treatment. not GLP, unpublished WAT2005-46	N	CFW
AIIIA-10.1	Köhler, U.	2002	Risk assessments for birds. not GLP, unpublished AVS2005-29	N	CFW
AIIIA-10.2	Köhler, U.	2002	Effects on aquatic organisms. not GLP, unpublished WAT2005-60	N	CFW
AIIIA-10.3	Köhler, U.	2002	Effects on terrestrial vertebrates other than birds. not GLP, unpublished AVS2005-30	N	CFW

Annex point/ reference number	Author(s)	Year	Title source (where different from company) report no. GLP or GEP status (where relevant), published or not BVL registration number	Data protection claimed Y/N	Owner ¹
AIIIA-10.6.1	Grunert, B.	1991	Toxizität für Regenwürmer von Polytanol. 91 20 43 031 GLP, unpublished ARW2005-25	Y	CFW

Codes of owner

CFW: Chemische Fabrik Wülfel

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