

# **Addendum to the risk profile of Dicofol**

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Final Report

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A COMPANY OF



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## 1 INTRODUCTION

This report is an addendum to the report 'Risk profile and summary report for dicofol; Dossier prepared for the third meeting of the UN-ECE Ad hoc Expert Group on POPs' (Rasenberg 2003), further referred to as original report. This original report for dicofol was first sent in by the Netherlands d.d. 19 November 2003 when it submitted dicofol for inclusion into the POP Protocol. Somewhat later the Dutch submission was suspended for consideration on request of the Netherlands and on the 9<sup>th</sup> of September 2005 the Netherlands withdrew its original submission. At the same time the Netherlands requested the Executive Body of the Convention on Long-range Transboundary Air Pollution for a technical review of dicofol to see if it is a POP. The original report on dicofol was again used to support this proposal. As the report was published in 2003, new information may have become available since, that is important for the evaluation of dicofol. Thus, the aim of this addendum is to update the original report. Only new information published in the period 2003 – 2005 is presented in the addendum. This implies that the original report and addendum should be assessed together when evaluating dicofol. To facilitate this process, the same lay-out as in the original report is followed. Occasionally, already presented data is also discussed, when this provides new information or is important for the interpretation of conclusions.

## 2 CHEMICAL IDENTITY

No new information

## 3 POP CHARACTERISTICS

### 3.1 Potential for long-range atmospheric transport

Mantseva et al (2004) developed a multicompartiment transport model for the evaluation of long-range atmospheric transport and deposition of POPs. Based on this model assessment a transport distance in Europe of 1650 km is calculated for dicofol. The residence time of dicofol in the atmosphere ( $T_{1/2}^{air}$ ) estimated on the basis of model calculations of its atmospheric transport totals to three days. For more details see Vulykh et al (2005).

We are not aware of monitoring studies in remote areas in which dicofol was included.

Conclusion: The conclusion drawn in the original report (Rasenberg 2003) remains unchanged: Dicofol meets the criterion for long-range transport.

### 3.2 Persistence

Since the persistence of dicofol is an important issue, the literature presented in the original report (Rasenberg 2003) is studied again into detail.

#### *Criteria*

Based on the executive body decision 1998/2, persistence is evident if under test conditions with water, soil or sediment the half life time of a substance is:

- 2 months in water;
- 6 months in soil or sediment.

Or alternatively, on the basis of monitoring data the substance appears to be sufficiently persistent to be of concern within the scope of the protocol.

#### *Test data*

Degradation in water is primarily by hydrolysis. The rate is strongly pH dependent. At pH 5 the p'p-isomer of dicofol has a DT50 of 85 days, at pH 7 the DT50 is 4 days. Given the steep relationship between hydrolysis rate and pH in the range 5 – 7 this implies that in situations where pH > 5.5 (including marine water) the DT50 for hydrolysis of dicofol will be < 2 months and dicofol is not persistent. In situations where a pH of 5 or less occurs dicofol is persistent. A pH of <5 occurs in acid lakes as for example lakes in peat mores in Scotland, Ireland and Scandinavia. It is known that about 10% of the lakes in Norway in 1995 had a pH of < 5.

Dissipation studies from soil were conducted in New York, Florida and California after application on strawberry and cotton, citrus grove and apple orchards. Neither dicofol nor its residues moved below 6 inches and volatilisation could be excluded. The half – life for the parent ranged up to 2 months. The overall half lives of dicofol residues in soils varied between 2 – 4 months (USEPA REDs database on Dicofol 115-32-2 Oct 2000).

Due to its high Koc value, dicofol will also be removed from water by adsorption to sediment. Also Xue et al (2005) show that dicofol is for 90% sorbed to sediment. In sediment anaerobic degradation will occur. A half-life < 30 days has been observed (Verschueren 1997)

In soil and sediment under aerobic or anaerobic conditions biodegradation of dicofol occurs with a half-life that is significantly shorter than 6 months. Thus, dicofol is not persistent in these matrices.

Degradation products of dicofol are dichlorobenzophenon (DCBP) that degrades relatively fast to the corresponding alcohol or carboxylates molecules (DCBH and DCBA) and these products degraded further at different rates. For DCBA the half-life in sediment is < 6 months and for DCBH the half-life in sediment is > 6 months.

Conclusion: According to the criteria for degradation dicofol is persistent in water with a pH < 5 (acid lakes). Dicofol is not persistent in water with a pH > 5.5 (including marine water), soil and sediment.

### **3.3 Bioaccumulation**

No new data

### **3.4 Toxicity and Ecotoxicity**

#### *Toxicity to aquatic organism*

From the OSPAR document on dicofol (OSPAR 2002, summarizing data from various sources), the following ranges on the aquatic toxicity of dicofol can be retrieved, see Table 3.4.

**Table 3.4: Aquatic toxicity ranges of dicofol (OSPAR 2002, summarizing data from various sources)**

	Acute toxicity (mg/l)	Chronic toxicity (mg/l)
Algae	0.07 – 0.5	
Daphnia	0.08 – 3.8	0.13
Fish	0.05 – 1.14	0.0045 – 0.019

Conclusion: The data from the OSPAR document are in the same order as in the original report and confirm that dicofol is very toxic to aquatic organisms.

#### *Toxicity to birds*

In addition to the data presented in the original report, eggshell thinning was observed also in captive American kestrels (Wiemeyer et al 2001). The lowest observed dietary effect concentration for eggshell thinning was 3 µg/g and the no/observed adverse effect concentration was 1 µg/g. This is slightly lower than the NOEC of 2.5 ppm (2.5 µg/g) for eggshell thinning in ducks as reported in the original report.

According to the OSPAR document on dicofol (OSPAR 2002), the pattern and magnitude of dicofol on eggshell thinning was similar as observed with p,p'-DDE. Schwarzbach et al (1988, cited in OSPAR 2002) showed that dicofol was not metabolised to DDE in birds and therefore that the adverse effect is based on dicofol itself. Dicofol metabolites have less effect on egg shell thinning than dicofol.

Conclusion: The new information confirms that dicofol causes eggshell thinning.

#### *Endocrine disruption*

Dicofol was shown to possess an estrogenic activity in an in vitro test with the α- and β-estrogenic receptor (Kojima et al 2004). Its potency was 1 x 10<sup>6</sup> lower than 17β-estradiol and a factor 10-100 lower than o,p'-DDT.

Lavado et al (2004) showed that dicofol can inhibit testosterone glucuronidation in an in vitro test system (carp liver microsomes). They hypothesised that when this inhibition would occur in a complete organism, the excretion of testosterone is also inhibited. A similar mode of action was observed for the well known androgenic compound tributyltin.

Also Thibaut and Porte (2004) showed that dicofol interfered with the synthesis of sexhormones in fish microsomes.

According to the study references submitted by the manufacturer, no effects on reproductive hormone function in rats was found for dicofol (Hoberman 1997, cited by OSPAR 2002).

Lind et al (2004) hypothesized that observed abnormalities in alligators from Lake Apopka, Florida (contaminated with DDT-like compounds and dicofol) were caused partly by the anti-androgenic and estrogenic actions of the various pesticides in the animals. The wild living alligators studied were exposed to a complex mixture of chemicals, many of which have the potential to interact with the alligator estrogen receptors. *o,p'*-DDT is generally considered estrogenic, but the metabolite *p,p'*-DDE has varying effects: It has been reported to be estrogenic or to have no estrogenic action in reptiles. However, the authors did not disclose a causal relationship between the observed effects in the alligators and any component in the mixture of chemicals they were exposed to.

**Conclusion:** In vitro studies show the estrogenic potential of dicofol as well as the potential to inhibit testosterone glucuronidation. The observed effects have not yet been confirmed by in vivo studies. A notice on contradicting data is made: for rats no effects on reproductive hormone function was found for dicofol.

### 3.5 Conclusion on POP characteristics of Dicofol

The conclusions on the POP characteristics of dicofol according to the UN-ECE criteria are presented in Table 3.5.

**Table 3.5: POP characteristics of dicofol according to the UN-ECE POP criteria**

Criterion	Meets the criterion (Yes/No)	Remark
Potential long-range atmospheric transport	Yes	No monitoring data available
Persistence in water, soil and sediment	Yes	Dicofol is only persistent in acid water (pH < 5).
Bioaccumulation	Yes	Experimental BCF > 5,000
Toxicity and ecotoxicity	Yes	Dicofol is moderately toxic to mammals. The substance is not carcinogenic. In wildlife it is reported to be reprotoxic. In birds, dicofol may reduce the eggshell quality. Dicofol is very toxic for the aquatic environment based on the acute toxicity tests. Dicofol shows endocrine disrupting effects in <i>in vitro</i> tests.

It is concluded that dicofol meet the UN-ECE POP criteria. For the criterion persistence, however, it should be noted that dicofol is only persistent in acid water with a pH < 5 (acid lakes as for example lakes in peat mores in Scotland, Ireland and Scandinavia). Dicofol is not persistent in water with a pH > 5.5 (including marine water), soil and sediment.

Important is also the issue whether dicofol should be considered as an endocrine disruptor. In vitro studies did show the estrogenic potential and the potential to endocrine disruption of dicofol but more research is needed to conclude whether dicofol exerts endocrine disruption in organisms.

## 4 EXTENT OF RELEASE TO THE ENVIRONMENT

### 4.1 Production

For the Czech Republic information has come forward: Dicofol is presently not produced in the Czech Republic (Hlinova 2005).

### 4.2 Uses

For the Czech Republic information has come forward: Dicofol as a pesticide has not been used in the Czech Republic since 31.1.2001 (Hlinova, 2005).

Dicofol is one of the major acaricides used in China. In 2002, an estimated total of 2750 tonnes of technical dicofol was used, almost half of it for the production of cotton (Qiu et al 2005). This technical dicofol was contaminated with 10 – 34% of DDT like compounds.

In Europe, the use of dicofol is not only allowed in Belgium, Luxembourg, Italy, France and Spain (original report) but also in Croatia and Portugal.

### 4.3 Emissions and pathways to the environment

The calculated emission of dicofol in UNECE-Europe for the year 2000 was 32 tonnes/year (Denier van der Gon et al 2005). This value is based on data from individual countries and expert estimates where detailed data are missing. The only emission source is through agriculture.

## 5 ENVIRONMENTAL LEVELS AND BIOAVAILABILITY

Dicofol has not yet been included in any monitoring programme focussing on remote and/or arctic areas. As a result, no studies on the presence of dicofol in these areas are available at this moment.

### **Presence of dicofol in countries that do not allow the use of dicofol**

No data available

### **Presence of dicofol in countries that allow the use of dicofol**

Dicofol has been monitored in surface and groundwater in France. The 90 percentile concentration was 10 ng/l in surface water and groundwater (520 and 359 samples, respectively) (annex of the original report).

In studies of Domagalski (1996) and of Pereira et al (1996) for the catchment area of the Californian San Joaquin river where dicofol was used in significant amounts, concentrations of up to 2.5 ng/l were found in water during the growing season, 23.7 ng/l in sediments and 97 ng/g in the tissue of the clam *Corbicula fluminea*.

In an agricultural area of Spain (Hernandez et al 1991) and in Greece (Angelidis et al 1996) dicofol was detected in river sediment at 2.2 µg/kg.

In a US study (Bender 2001) for an area with intensive use of dicofol 2 % of the water and sediment samples contained residues of the p,p' isomer above the reporting level of 5 ng/l water respectively 0.1 mg/kg sediment.

Aquatic invertebrates contained residues over 0.1 mg/kg in 7.2 % of the samples and fish contained residues over 0.05 – 0.1 mg/kg in 71 % of the samples.

Samples of small mammals, terrestrial invertebrates, reptiles/amphibians, earthworms and birds all contained concentrations between 0.1 and 3.9 mg/kg.

Dicofol was observed in organisms captured or collected near areas of high dicofol use. Whiptail lizard carcasses from Texas contained a maximum of 12 mg/kg dicofol and an average concentration of 0.87 mg/kg (cited in Wiemeyer et al 2001). In Florida screech owl from areas of intensive dicofol use 1.8 mg/kg dicofol, average 0.20 was measured (cited in Wiemeyer et al 2001).

In China, many studies have been carried out on the presence of DDT-like compounds as a result of the use of dicofol (Chen et al 2005; Xu et al 2005a, b; Wan et al 2005). A ratio of o,p'-DDT/p,p'-DDT of 7 indicates the recent use of dicofol (Qiu et al 2005). Nevertheless, only in few studies dicofol itself was included in the measurement programme. Xue et al (2005) measured dicofol in water and sediment of the Beijing Guanting reservoir, which is known to be heavily contaminated due to runoff from non-point sources and direct dumping of waste in the river. Concentrations ranged from not detected – 2.60 ng/l in water (average 0.91 ng/l), not detected – 28.5 ng/l in pore water (average 4 ng/l) and not detected – 0.06 mg/kg in sediment (average 0.05 mg/kg).

#### Conclusion

The above mentioned observations of residues in sediment and animals in the direct proximity of areas with intensive use are not indicative for a long-term persistence. For areas where no dicofol is used no data are available.

## 6 SOCIO-ECONOMIC FACTORS

Dicofol is one of the major acaricides used in China. In 2002 and 2003 dicofol produced by 7 different manufacturers was contaminated with 10 – 34% of DDT like compounds (Qiu et al 2005). This observation was done around the time two standards were implemented in China, requiring the DDT impurity to be no more than 0.5% of technical dicofol or no more than 0.1% of formulated dicofol containing 20% dicofol. The present situation on dicofol impurities in China is unknown.

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