Committee for Risk Assessment

RAC

Opinion

proposing harmonised classification and labelling
at Community level of

Benzenamine, 2-chloro-6-nitro-3-phenoxy- (Aclonifen)

ECHA/RAC/CLH-O-0000001543-79-03/F

Adopted

14 September 2011

Annankatu 18 | P.O. Box 400 | 00121 Helsinki | Finland
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ECHA/RAC/CLH-O-0000001543-79-03/F

OPINION OF THE COMMITTEE FOR RISK ASSESSMENT ON A DOSSIER PROPOSING HARMONISED CLASSIFICATION AND LABELLING AT COMMUNITY LEVEL

In accordance with Article 37(4) of the Regulation (EC) No 1272/2008 (CLP Regulation), the Committee for Risk Assessment (RAC) has adopted an opinion on the proposal for harmonised classification and labelling of

1. Chemical name: Benzenamine, 2-chloro-6-nitro-3-phenoxy- (Aclonifen)

2. EC No.: 277-704-1

3. CAS No.: 74070-46-5

The proposal was submitted by ***Germany***
and received by RAC on **17 January 2011**

The proposed harmonised classification

	Regulation (EC) No 1272/2008	Directive 67/548/EEC
Current entry in Annex VI of CLP Regulation (EC) No 1272/2008	Aquatic Acute 1 – H400 Aquatic Chronic 1 – H410	N; R50/53
Proposal by dossier submitter for consideration by RAC	Carc. 2 – H351 Skin Sens. 1 – H317 M-factor 100	Carc. Cat. 3; R40 R43 C≥0.25% N; R50/53 0.025%≤C<0.25% N; R51/53 0.0025%≤C<0.025% R52/53
Resulting harmonised classification (future entry in Annex VI of CLP Regulation) as proposed by dossier submitter	Carc. 2 – H351 Skin Sens. 1 – H317 Aquatic Acute 1 – H400 Aquatic Chronic 1 – H410 M-factor 100	Carc. Cat. 3; R40 R43 N; R50/53 C≥0.25% N; R50/53 0.025%≤C<0.25% N; R51/53 0.0025%≤C<0.025% R52/53

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PROCESS FOR ADOPTION OF THE OPINION

Germany has submitted a CLH dossier containing a proposal together with the justification and background information documented in a CLH report. The CLH report was made publicly available in accordance with the requirements of the CLP Regulation at ***http://echa.europa.eu/consultations/harmonised_cl/harmon_cl_prev_cons_en.asp*** on 17 January 2011. Parties concerned and MSCAs were invited to submit comments and contributions by 03 March 2011.

ADOPTION OF THE OPINION OF RAC

Rapporteur, appointed by RAC: ***Marja Pronk***
Co-rapporteur, appointed by RAC: ***Riitta Leinonen***

The opinion takes into account the comments of MSCAs and parties concerned provided in accordance with Article 37(4) of the CLP Regulation.

The RAC opinion on the proposed harmonised classification and labelling has been reached on 14 September 2011**,** in accordance with Article 37(4) of the CLP Regulation, giving parties concerned the opportunity to comment. Comments received are compiled in Annex 2.

The RAC Opinion was adopted by **consensus**.

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OPINION OF RAC

The RAC adopted the opinion that **Aclonifen** should be classified and labelled as follows:

Classification & Labelling in accordance with the CLP Regulation

Index No	International Chemical Identification	EC No	CAS No	Classification		Labelling			Specific Conc. Limits, M-factors	Notes
				Hazard Class and Category Code(s)	Hazard statement Code(s)	Pictogram, Signal Word Code(s)	Hazard statement Code(s)	Suppl. Hazard statement Code(s)		
612-120-00-6	Aclonifen (ISO) 2-chloro-6-nitro-3-phenoxyaniline	277-704-1	74070-46-5	Carc. 2 Skin. Sens. 1A Aquatic Acute 1 Aquatic Chronic 1	H351 H317 H400 H410	GHS08 GHS07 GHS09 Wng	H351 H317 H410		M = 100 (Acute) M = 10 (Chronic)	

Classification & Labelling in accordance with Directive 67/548/EEC:

Index No	International Chemical Identification	EC No	CAS No	Classification	Labelling	Concentration Limits	Notes
612-120-00-6	Aclonifen (ISO) 2-chloro-6-nitro-3-phenoxyaniline	277-704-1	74070-46-5	Carc. Cat. 3; R40 R43 N; 50/53	Xn, N R: 40-43-50/53 S: (2-)36/37-60-61	C≥0.1% R43 C≥0.25% N; R50/53 0.025%≤C<0.25% N; R51/53 0.0025%≤C<0.025% R52/53	

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SCIENTIFIC GROUNDS FOR THE OPINION

In 2008, aclonifen was included as active substance in Annex I of the Plant Protection Products Directive (91/414/EEC). This opinion on harmonised classification and labelling relates to all hazard classes.

1. HUMAN HEALTH HAZARD ASSESSMENT

1.1 Acute toxicity and Specific Target Organ Toxicity – Single Exposure (STOT-SE)

1.1.1 Dossier submitter

Aclonifen is of very low acute toxicity by the oral (LD_{50} > 5000 mg/kg bw), dermal (LD_{50} > 5000 mg/kg bw) and inhalation route (LC_{50} > 5.06 mg/L) in the rat and also by the oral route in the mouse (LD_{50} > 5000 mg/kg bw). No classification is required.

1.1.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for acute toxicity (or for specific target organ toxicity upon single exposure), which was not questioned during public consultation.

For assessment of oral acute toxicity one rat and one mice study, both with a reported LD_{50} of > 5000 mg/kg bw, are available. The LD_{50} is above the threshold value of 2000 mg/kg bw for both Acute Tox. 4 – H302 (CLP) and Xn; R22 (DSD).
For assessment of dermal acute toxicity one rat study with a reported LD_{50} of >5000 mg/kg bw is available. This LD_{50} is above the threshold value of 2000 mg/kg bw for both Acute Tox. 4 – H312 (CLP) and Xn; R21 (DSD).
For assessment of inhalation acute toxicity one rat study with a reported LC_{50} of >5.06 mg/L/4hr is available. This LC_{50} is above the threshold value of 5 mg/L/4hr for both Acute Tox. 4 – H332 (CLP) and Xn; R20 (DSD).

In the acute toxicity studies only slight clinical effects were observed, which were transient in nature. These effects do not fulfil the CLP criteria to classify for STOT-SE.

Based on the available data, RAC supported the conclusion of the dossier submitter that aclonifen should not be classified for acute oral, dermal or inhalation toxicity. RAC also concluded that aclonifen should not be classified for STOT-SE.

1.2 Irritation

1.2.1 Dossier submitter

Very slight dermal and no ocular irritation was noted after application of aclonifen to the skin and eye of rabbits. Therefore no classification for irritation is required.

1.2.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for irritation, which was not questioned during public consultation.

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For assessment of skin irritation a rabbit study is available. In this study, some slight, transient irritation was observed, with mean scores for erythema, and eschar formation or oedema formation below the threshold value of 2.3 for Skin Irrit. 2 – H315 (CLP) or 2 for Xi; R38 (DSD).

For assessment of eye irritation a rabbit study is available. In this study, no effects on the cornea, iris or conjunctiva were observed (all scores 0).

No data are available for respiratory tract irritation.

Based on the data available, RAC supported the conclusion of the dossier submitter that aclonifen should not be classified for irritation.

1.3 Corrosivity

1.3.1 Dossier submitter

In skin and eye irritation studies there was no evidence for a corrosive action of aclonifen.

1.3.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for corrosion, which was not questioned during public consultation.

In skin and eye irritation studies there was no evidence for a corrosive action of aclonifen. RAC therefore concluded that aclonifen does not fulfil the criteria for classification as Skin Corr. 1B – H314 (CLP) or C; R34 (DSD).

1.4 Sensitisation

1.4.1 Dossier submitter

While in a Buehler test negative results were obtained, aclonifen caused delayed contact hypersensitivity in guinea pigs in a Magnusson & Kligman skin sensitisation test. With the exception of one animal all induced guinea pigs (95 %) showed a skin reaction after challenge. Based on these data a classification as R43 “Irritant; May cause sensitisation by skin contact” is required.

According to Directive 67/548/EEC:

R43 (Irritant; May cause sensitisation by skin contact)

According to Regulation (EC) No 1272/2008:

Skin Sens. 1; H317 (May cause an allergic skin reaction)

1.4.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter to classify aclonifen for skin sensitisation with Skin Sens. 1 – H317 (CLP) or R43 (DSD). This classification proposal was not questioned during public consultation, but a sub-categorisation under CLP was asked for, in accordance with the 2nd ATP.

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For assessment of skin sensitisation 2 guinea pig studies are available. In the Buehler test, 0% of the animals showed a positive response. However, in the GPMT test, 95% of the test animals showed a positive response, compared to 0% of the controls. This is above the threshold of 30% for Skin Sens. 1 – H317 (CLP) or R43 (DSD). As the response is also above the threshold of 60% at an intradermal induction dose of 1%, aclonifen can be considered a strong sensitiser, leading to sub- category 1A under CLP according to the 2nd ATP, as well as the setting of a specific concentration limit (SCL) of 0.1% under the DSD (in line with the generic concentration limit for the sub-category 1A under CLP according to the 2nd ATP).

No data are available for respiratory sensitisation.

Based on the data available, RAC supported the proposal of the dossier submitter to classify aclonifen for skin sensitisation. The appropriate classification is:

Skin Sens. 1A – H317: May cause an allergic skin reaction (CLP, taking into account the 2nd ATP)

R43: May cause sensitisation by skin contact (DSD)

SCL: 0.1%

1.5 Repeated dose toxicity

1.5.1 Dossier submitter

Liver and kidney have been identified as the main target organs. Toxic effects in these organs appear to be related to concentrations that overwhelm metabolic and/or excretional capacities. No classification for repeated dose toxicity is required.

1.5.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for repeated dose toxicity, which was not questioned during public consultation.

For assessment of oral repeated dose toxicity, five studies were available, among which three 90-day studies in rat. Liver and kidney have been identified as the main target organs in rats and mice. In rats, the thyroid was also affected, in mice the ovaries. The lowest NOAEL and LOAEL in the oral repeated dose studies were 3.6 and 35.4 mg/kg bw/day, respectively, in a 90-day rat study. At this LOAEL, some hyperplasia and hypertrophy was noted, without clear effects on organ weights or associated blood and urine parameters. These effects are not considered “significant and/or severe toxicity” in the sense of classification. The next higher LOAEL is 258 mg/kg bw/day. Although at this dose level (and comparable dose levels in the other studies) more significant effects were observed, the dose level is clearly above the 90-day guidance value of 100 mg/kg bw/d for STOT RE 2 – H373 (CLP) and of 50 mg/kg bw/d for Xn; R48/22 (DSD).

For the assessment of dermal repeated dose toxicity, one 28-day study in rats was available. The LOAEL in this study is 1000 mg/kg bw/day. This is clearly above the guidance value of 600 mg/kg bw/ (recalculated for 28 days) for STOT RE 2 – H373 (CLP) and of 300 mg/kg bw/d (recalculated for 28 days) for Xn; R48/21 (DSD).

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No studies were available for repeated dose inhalation toxicity.

Based on the data available, RAC supported the conclusion of the dossier submitter that aclonifen should not be classified for repeated dose toxicity.

1.6 Mutagenicity

1.6.1 Dossier submitter

Aclonifen did not induce gene mutations in procaryotes or mammalian cell cultures, chromosome aberrations in cultured human lymphocytes or *in vivo* in bone marrow cells from NMRI mice, nor did it lead to DNA damage in mammalian cells in the *in vitro* UDS assay. Aclonifen (or metabolites) does not bind to DNA *in vivo*, but has been shown to interact with chromatin proteins (specific interaction partners were not identified). Therefore, it may produce epigenetic changes on chromosomes and on gene expression. Taken together, the results demonstrate that aclonifen is not genotoxic and is unlikely to present a genotoxic hazard to humans. Classification for genotoxicity is not required.

1.6.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for mutagenicity, which was not questioned during public consultation.

For assessment of mutagenicity, several *in vitro* (bacterial and mammalian cell assays) and one *in vivo* study were available. All studies were negative with regard to mutagenicity. Consequently, RAC supported the conclusion by the dossier submitter that no classification for mutagenicity is necessary.

1.7 Carcinogenicity

1.7.1 Dossier submitter

In the carcinogenicity study in mice, urinary bladder tumours were found in two males and one female at the highest dose (7000 ppm). Taking into account the lack of genotoxicity and that the kidney is responsible for the excretion of a major part of the dose, these tumours are attributed to the continuous irritation of the tissue at high doses of aclonifen. A similar mechanism can be excluded with respect to the astrocytomas seen in four out of sixty female rats in the high dose group. According to the toxicokinetic data aclonifen/metabolite levels in male and female rat brains are low, even at time points with the highest blood and plasma concentrations; unless astrocytes have a mechanism of concentrating the test substance or unless the blood-brain barrier becomes leaky with age or prolonged treatment, very little exposure should occur. In addition, male rats experience higher blood, plasma and brain levels of aclonifen-related material than females and should therefore be at a larger risk for a tumourigenic effect on astrocytes. Thus no mechanistic explanation could be found. However, due to the rarity of this tumour type in control groups, the finding in female rats remains a concern and is considered as limited evidence of carcinogenicity. Consequently, a classification of aclonifen as a carcinogen is proposed.

According to Directive 67/548/EEC:

Carc. Cat. 3 R40 (Harmful; Limited evidence of a carcinogenic effect)

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According to Regulation (EC) No 1272/2008:

Carc. 2; H351 (Suspected of causing cancer)

1.7.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter to classify aclonifen for carcinogenicity as Carc. 2 – H351 (CLP) or Carc. Cat. 3; R40 (DSD), based on a low incidence of unusual brain tumours in female rats. There was support for this proposal during public consultation, aside from one Industry association that referred to a position paper. In this position paper (dated February 2006), Industry commented during the peer review consultation of the aclonifen DAR on a similar proposal for classification by the RMS Germany, and considered the brain tumours observed in high dose females to be unlikely related to the administration of aclonifen. The Industry comments however did not change the opinion of EFSA: EFSA concluded in their final opinion of 2008 that the brain tumours remained of concern, and therefore kept the classification proposal for Carc. Cat. 3; R40.

For assessment of the carcinogenic potential of aclonifen, three combined toxicity/carcinogenicity studies are available, two in rats and one in mice. In the study in mice, urinary bladder tumours were found in two males and one female at the highest dose (7000 ppm). Neither in males or in females the incidence was statistically significantly increased, nor was there a positive trend. Moreover, at 7000 ppm also chronic inflammation was observed in the urinary bladder, as well as transitional cell hyperplasia. Upon review of the urinary bladder histological lesions, signs suggestive of crystal formation were noted that had not been reported in the original study. In rats, where the kidney is responsible for the excretion of a major part of the aclonifen dose, at high doses of 5000 ppm crystals have been observed in urine and aggregated brownish deposits in kidney and urinary bladder. If these data on urinary excretion are applicable for mice as well, crystal formation can be expected at high doses where the urinary concentration of test substance derived material approaches or exceeds the limit of solubility in aqueous media. Taking further into account that aclonifen is not genotoxic, the urinary bladder tumours likely resulted from a persistent irritation/inflammation of the tissue following crystal formation at high doses of aclonifen. All in all, RAC concluded that the urinary bladder tumours observed at 7000 ppm are not relevant for classification.

In one of the rat studies, a slightly higher incidence of thyroid C-cell carcinomas was seen in females without a dose-response relationship. This finding was not confirmed upon two separate histological re-evaluations of the thyroid sections, nor was there evidence of an oncogenic effect on the thyroid in the second rat study. Therefore RAC considered the finding probably unrelated to aclonifen treatment.

In the second rat study, an increased incidence (positive trend) of astrocytomas was observed in brains of females of the high dose group. The incidence in the high dose females (4/60, as compared to 0/60 for the controls) was above the reported historical control incidences. Also in the high dose males, where the incidence of astrocytomas (2/60) was not statistically significantly increased compared to the controls (1/60), the incidence was slightly above the reported historical control incidences. There is no mechanistic explanation for the astrocytoma findings. The toxicokinetic data on aclonifen indicate that aclonifen/metabolite levels in male and female rat brains are low. So, unless astrocytes have a mechanism of concentrating the test substance or unless the blood-brain barrier becomes leaky with age or prolonged treatment, very little exposure should occur. In addition, male rats experience

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higher blood, plasma and brain levels of aclonifen-related material than females and should therefore be at a larger risk for a tumourigenic effect on astrocytes. But that was not the case in this study. Due to the rarity of this tumour type and the absence of a mechanistic explanation, the finding in female rats remains a concern and is considered as limited evidence of carcinogenicity. Consequently, RAC supported the proposal of the dossier submitter to classify aclonifen for carcinogenicity. The appropriate classification is:

Carc. 2 – H351: Suspected of causing cancer
Carc. Cat. 3; R40: Limited evidence of a carcinogenic effect

1.8 Reproductive toxicity

1.8.1 Dossier submitter

Aclonifen did not affect reproduction and influenced developmental parameters only at a dose that also induced systemic effects in the dams. The decrease in the number of corpora lutea observed in the 28-day mouse study at a dose of 12 g/kg bw/day is not considered a specific effect on reproduction. As no specific impairments of fertility and embryo-foetal development have been observed a classification for fertility effects or developmental toxicity is not required.

1.8.2 RAC opinion

The evaluation by RAC relates to the proposal of the dossier submitter not to classify aclonifen for reproductive toxicity, which was not questioned during public consultation.

For assessment of the reproductive toxic potential of aclonifen, a 2-generation study in rats and two developmental toxicity studies (one in rat, one in rabbit) are available. Aclonifen did not affect reproductive parameters in the rat, nor was it teratogenic in the rat and rabbit. The only effect observed in these studies was a reduction in foetal (minus 7%) and pup (up to 22%) body weight in the rat developmental toxicity and 2-generation study, respectively, at doses that also induced maternal toxicity (reduced body weight gain of 10% and 12-18%, respectively). The decrease in the number of corpora lutea observed in the 28-day mouse study at 50000 ppm (approximately 12 g/kg bw/d) is not considered a specific effect on reproduction when occurring at such a high dose level. As no specific impairments of fertility and embryo-foetal development have been observed, RAC supported the conclusion of the dossier submitter that aclonifen should not be classified for fertility effects or developmental toxicity.

2. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

2.1 Explosivity

2.1.1 Dossier submitter

Aclonifen (technical) is not explosive in the sense of EEC method A14.

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2.1.2 RAC opinion

Given that aclonifen is not sensitive to heat, shock or friction, RAC concluded that aclonifen does not need to be classified as explosive.

2.2 Flammability

2.2.1 Dossier submitter

Aclonifen (technical) is not highly flammable in the sense of EEC method A10.

2.2.2 RAC opinion

On contact by the hot wire, technical aclonifen melted, but no flame was observed. Technical aclonifen melted at about 85 °C, no autoinflammation occurred.
RAC concluded that aclonifen does not need to be classified as flammable.

2.3 Oxidising potential

2.3.1 Dossier submitter

Aclonifen (technical) has no oxidising properties in the sense of EEC method A17.

2.3.2 RAC opinion

A mixture of 40/60 % aclonifen/cellulose gave reproducibly higher burning rates than BaNO3/cellulose. When cellulose was replaced by silica, the flame rapidly extinguished. Under nitrogen the test mixture did not burn.
RAC concluded that aclonifen does not need to be classified as oxidising.

3. ENVIRONMENTAL HAZARD ASSESSMENT

3.1 Hazard to the Aquatic Environment

3.1.1 Dossier submitter

In aquatic toxicity studies, ErC_{50} values for algae and aquatic plants and LC_{50} value for fish were obtained at aclonifen concentrations < 1 mg/L. Aclonifen is not readily biodegradable according to the Sturm test (OECD 301B) and the simulation tests (EU (=EEC) 95/36/EC (1995) and SETAC 1.1 (1995). Aclonifen has a log Kow of 4.37. The experimentally derived steady state BCF of 2896 and kinetic BCF of 2248 are above the trigger of 100 (criterion for bioaccumulating potential conform Directive 67/548/EEC) and of 500 (criterion for bioaccumulating potential conform Regulation (EC) No 1272/2008). Aclonifen therefore fulfils the criteria for classification with

N; R50/53 (according to Directive 67/548/EEC); and as
aquatic environmental hazard acute category 1, H400 and aquatic environmental hazard chronic category 1, H410 (according to Regulation (EC) No 1272/2008).

Based on the toxicity data for *Desmodesmus subspicatus* (ErC_{50} 0.0069 mg/L) the following specific concentration limits should be applied:

Concentration	Classification
C ≥ 0.25%	N; R50/53

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0.025% ≤ C < 0.25% N; R51-53
0.0025% ≤ C < 0.025% R52-53

where C is the concentration of aclonifen in the preparation.

The M-factor for aclonifen is 100. This value is based on ErC_{50} value of 0.0069 mg/L obtained for the algae *Desmodesmus subspicatus* in a 96-h static study.

3.1.2 RAC opinion

The evaluation by RAC relates to the classification proposal of the dossier submitter to keep unchanged the existing harmonised classification for aquatic acute and chronic toxicity, but to add an M-factor of 100 and corresponding SCLs. This classification proposal was not questioned during public consultation, except for the M-factor where comments suggested M-factors of 100 and 10 for the short-term and long-term hazard category, respectively. RAC concluded the following.

Aclonifen is hydrolytically stable. Aclonifen was found to be not readily biodegradable within 28 days in the Sturm test (OECD guideline 301B). In a water/sediment study aclonifen is metabolised at a moderate rate (DT_{50s} of 11.2 and 17.3 days) but there was neglible mineralisation. In a soil degradation study the DT_{50s} for aclonifen ranged from 41.9 days to 93.6 days. Mineralisation was negligible or very low. There is no information on the degradation products in either study. Aclonifen has a log Kow of 4.37. In a BCF study, a BCF value of 2896 was obtained based on plateau total radioactive residue in whole fish and average total radioactive residue in water, whereas a BCF value of 2248 was obtained based on uptake and elimination rate constants.

Aclonifen shows a high acute toxicity to algae (ErC_{50} = 0.0069 mg/L) and aquatic plants (ErC_{50} = 0.012 mg/L). The acute toxicity of aclonifen to fish and invertebrates is in the mg/L range with an LC_{50} = 0.67 mg/L to fish and an EC_{50} = 1.2 mg/L to invertebrates. The lowest toxicity values in chronic studies were a 35-day NOEC to fish of 0.005 mg/L, a 21-day NOEC to *Daphnia* of 0.016 mg/L, a 96-h NOEC to algae of 0.0025 mg/l, and a 14-day NOErC to the aquatic plant *Lemna* of 0.0012 mg/L.

According to the CLP Regulation the aquatic plant growth inhibition tests are normally considered as chronic tests but the EC_{50s} are treated as acute values for classification purposes.

Conclusion of environmental classification according to Regulation (EC) No 1272/ 2008, taking into account the 2nd ATP

In aquatic toxicity studies, ErC_{50} values for algae and aquatic plants and LC_{50} value for fish were obtained at aclonifen concentrations < 1 mg/L. The chronic toxicity values for the three trophic levels vary from 0.0012 to 0.016 mg/L aclonifen, and are below the cut-off value of 0.1 mg/L. Aclonifen is not rapidly biodegradable. The experimentally derived steady state BCF of 2896 and kinetic BCF of 2248 are above the trigger of 500. Aclonifen therefore fulfils the criteria for classification as hazardous to the aquatic environment, acute category 1, H400 and chronic category 1, H410.

The M-factor for aclonifen for the short-term hazard category is 100. This value is based on ErC_{50} value of 0.0069 mg/L obtained for the algae *Desmodesmus subspicatus* in a 96-h static study.

The M-factor for long-term hazard is 10, based on the NOErC to *Lemna gibba* of 0.0012 mg/L.

Aquatic Acute 1 – H400: Very toxic to aquatic life
Aquatic Chronic 1 – H410: Very toxic to aquatic life with long lasting effects
M-factor acute 100; M-factor chronic 10

Conclusion of environmental classification according to Directive 67/548/EEC

In aquatic toxicity studies, ErC_{50} values for algae and aquatic plants and LC_{50} value for fish were obtained at aclonifen concentrations < 1 mg/L. Aclonifen is not readily biodegradable. Aclonifen has a log Kow of 4.37. The experimentally derived steady state BCF of 2896 and kinetic BCF of 2248 are above the trigger of 100. Aclonifen therefore fulfils the criteria for classification with N; R50/53.

Based on the toxicity data for *Desmodesmus subspicatus* (ErC50 0.0069 mg/L) the following specific concentration limits should be applied:

Concentration	Classification
$C \geq 0.25\%$	N; R50/53
$0.025\% \leq C < 0.25\%$	N; R51/53
$0.0025\% \leq C < 0.025\%$	R52/53

where C is the concentration of aclonifen in the preparation.

N; R50/53: Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.		
SCLs	$C \geq 0.25\%$	N; R50/53
	$0.025\% \leq C < 0.25\%$	N; R51/53
	$0.0025\% \leq C < 0.025\%$	R52/53

Additional information

The Background Document, attached as Annex 1, gives the detailed scientific grounds for the Opinion.

ANNEXES:

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Annex 1 Background Document (BD)[1]

Annex 2 Comments received on the CLH report, response to comments provided by the dossier submitter and rapporteurs' comments (excl. confidential information)

[1] The Background Document (BD) supporting the opinion contains scientific justifications for the CLH proposal. The BD is based on the CLH report prepared by a dossier submitter. The original CLH report may need to be changed as a result of the comments and contributions received during the public consultation(s) and the comments by and discussions in the Committees.

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