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TOXICOLOGICAL EVALUATIONS

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TOXICOLOGICAL EVALUATION

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Chloroformic acid butyl ester

No. 160

CAS No. 592-34-7

BG Chemie
Berufsgenossenschaft der chemischen Industrie

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Chloroformic acid butyl ester

Apart from the evaluation of chloroformic acid butyl ester (No 160), there are also TOXICOLOGICAL EVALUATIONS of chloroformic acid methyl ester (No. 36), chloroformic acid ethyl ester (No. 77) and chloroformic acid propyl ester (No. 159), which may be consulted for comparison.

1 Summary and assessment

Chloroformic acid butyl ester is of low toxicity upon acute oral administration (LD_{50} rat oral for a solution of the chemical in oil: 2610 mg/kg body weight). LD_{50} determinations using aqueous formulations of the chemical gave lower oral LD_{50} values of approx. 1325 and approx. 2120 mg/kg body weight for the rat. Chloroformic acid butyl ester is clearly toxic following inhalation; a 1-hour exposure to 200 ppm (approx. 1134 mg/m³) was lethal to 4 out of 10 rats while in inhalation hazard tests all rats died following exposure for 3 or 10 minutes to atmosphere which was enriched or saturated with vapour at 20 °C, respectively. Dyspnoea, apathy, abnormal position, reeling, spastic gait, rough coat, diarrhoea, cyanosis and poor general condition were reported as signs of toxicity following oral administration. The signs of toxicity seen upon inhalation exposure included severe mucous membrane irritation, gasping and dyspnoea caused by the agent's corrosive effects. Necropsy findings also, in particular, included changes that were attributable to the chemical's corrosive properties, with inhalation exposure being associated with considerable congestion, emphysema and oedema of the lungs with hydrothorax and oral administration of lethal doses resulting in white intestinal mucosa (necrosis) and bloody sloughing in the glandular stomach and forestomach (corrosive gastritis). In addition, findings following oral administration of lethal doses also included acute atrial dilatation and acute congestive hyperaemia of the heart. Upon oral administration of sublethal doses > 681 mg/kg body weight, the rats' forestomachs were hardened and thickened, exhibiting some evaginations, and the forestomach mucosa had removable encrustations.

The toxic effects of chloroformic acid butyl ester (98.9% pure) following subacute inhalation exposure were investigated in Sprague-Dawley rats in an inhalation study that met the requirements set forth in OECD guideline

No. 412. In the preliminary 5-day range-finding study, all animals survived daily 6-hour whole-body exposure to chloroformic acid butyl ester at nominal levels of 0 (controls), 15, 50 or 150 mg/m³ (analysed levels of 0 (controls), 16, 55 and 158 mg/m³, respectively). Clinical signs of toxicity observed in rats from the top two concentration groups included dose-dependent sneezing, rubbing the snout with paws, closed or half-closed eyes, rapid breathing, licking the inside of the mouth and, between exposures, sniffing and noisy respiration. Rats in the top concentration group additionally exhibited prone position, failure to react to acoustic stimuli and, after the first exposure, hypoactivity. The males of the top concentration group lost weight over the entire study period, while the top-dose females showed initial body weight loss followed by body weight gain at a markedly reduced rate. Body weight gains of animals from the intermediate concentration group were also retarded. Food consumption was reduced in a concentration-dependent manner at all dose levels. Initially reduced, water consumption in animals from the top concentration group was increased at the end of the study. Lung weights were increased in males and females from the top concentration group and in females from the intermediate concentration group, and the lungs of the top-dose animals and of one male from the intermediate concentration group failed to collapse upon opening of the thoracic cavity. In the 28-day study, all animals survived whole-body exposure to chloroformic acid butyl ester at nominal levels of 0 (controls), 3, 15 or 30 mg/m³ (analysed levels of 0 (controls), 2.8, 10.0 and 28.2 mg/m³, respectively). The only clinical sign of toxicity observed was piloerection, which occurred in the top concentration group. Toxicologically relevant findings as compared with controls were confined to the top concentration group. Lung weights were increased, this finding being significant in the males only, and histological examination of the lungs revealed pathological changes of the carina tracheae in the form of minimal focal epithelial hyperplasia in 1 out of 5 males and 3 out of 5 females together with minimal focal crowding of epithelial cells in a further 3 out of 5 males from that concentration group. Chloroformic acid butyl ester had a *no observed adverse effect level* (NOAEL) of 10 mg/m³ in both sexes in the 28-day inhalation study described.

Chloroformic acid butyl ester has a severely irritating and corrosive effect on the skin and eyes of rabbits. Even a 5-minute skin exposure causes slight necrosis.

Conducted as a preincubation test, the Salmonella/microsome test on *Salmonella typhimurium* strains TA 98, TA 100, TA 1535 and TA 1537 revealed no mutagenic potential for chloroformic acid butyl ester either in the absence or presence of metabolic activation. In the absence of metabolic activation, chloroformic acid butyl ester is not clastogenic in the chromosome aberration test in V79 cells of the Chinese hamster. A nonreproducible increase in aberration rate seen at the highest test concentration in the presence of metabolic activation was evaluated as resulting from the strong cytotoxic activity of that concentration on the cells' genetic material rather than being an indication of the genotoxic potential of the agent as such. Concentration levels of chloroformic acid butyl ester which reduced cell survival rates by more than 50% but not below 30% did not induce chromosome aberrations.

In the Federal Republic of Germany, the Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area ("MAK-Kommission") of the Deutsche Forschungsgemeinschaft has established a MAK value (maximum workplace concentration) for chloroacetic acid butyl ester on the suggestion of BG Chemie. It was set in the List of MAK- and BAT Values 2004 at 0,2 ml/m³ (ppm, equivalent to 1,1 mg/m³). Furthermore, chloroacetic acid butyl ester has been assigned to pregnancy risk group C, i.e. substances for which "there is no reason to fear a risk of damage to the embryo or foetus when MAK and BAT values are observed".

2 Name of substance

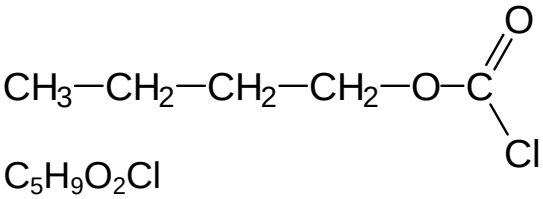
2.1	Usual name	Chloroformic acid butyl ester
2.2	IUPAC name	Chloroformic acid n-butyl ester
2.3	CAS No.	592-34-7
2.4	EINECS No.	209-750-5

3 Synonyms, common and trade names

Butoxycarbonyl chloride
Butylchlorformiat
n-Butylchlorformiat
n-Butylchlorkohlensäureester
Butyl chlorocarbonate
Butyl chloroformate
n-Butyl chloroformate
Carbonochloridic acid, butyl ester
Chlorameisensäurebutylester
Chlorameisensäure-n-butylester
Chloroformic acid butyl ester
Formic acid, chloro-, butyl ester

4 Structural and molecular formulae

4.1 Structural formula



4.2 Molecular formula $C_5H_9O_2Cl$

5 Physical and chemical properties

5.1	Molecular mass, g/mol	136.58	
5.2	Melting point, °C	< –70	(BASF, 1999)
5.3	Boiling point, °C	35 (at 17 hPa)	(Böhm, 2001)
		44 (at 26.7 hPa)	(Damle, 1992)
		77.6 (at 133 hPa)	(Damle, 1992)
		138	(Bayer, 2001)
		138–144.5 (at 1013 hPa)	(BASF, 1991)
		142 (at 1013 hPa)	(Böhm, 2001; Lide and Frederikse, 1997)
5.4	Vapour pressure, hPa	7 (at 20 °C)	(BASF, 1999)
		ca. 15 (at 20 °C)	(Bayer, 2001)
		36 (at 50 °C)	(BASF, 1991)
		ca. 53 (at 50 °C)	(Bayer, 2001)
		ca. 64 (at 55 °C)	(Bayer, 2001)

5.5	Density, g/cm³	1.0513 (at 20 °C) (Böhm, 2001) ca. 1.053 (at 20 °C) (Bayer, 2001) 1.0585 (at 20 °C) (Damle, 1992) 1.06 (at 20 °C) (BASF, 1999) 1.074 (at 25 °C) (Lide and Frederikse, 1997)
5.6	Solubility in water	Does not mix, hydrolysis (Bayer, 2001) Insoluble, hydrolysis (Damle, 1992) Poorly soluble, undergoes gradual hydrolysis to 1-butanol, hydrochloric acid and carbon dioxide (BASF, 1991)
5.7	Solubility in organic solvents	Miscible with ether, soluble in acetone, slightly soluble in tetrachloromethane (Lide and Frederikse, 1997) Soluble in ethanol (BASF, 1988 b)
5.8	Solubility in fat	No information available
5.9	pH value	Acidic (BASF, 1999; Bayer, 2001)
5.10	Conversion factor	1 ml/m³ (ppm) ≙ 5.67 mg/m³ 1 mg/m³ ≙ 0.18 ml/m³ (ppm) (at 1013 hPa and 25 °C)

6 Uses

Versatile intermediate used in the chemical industry, particularly in the manufacture of peroxydicarbonates and pesticides (BASF, 1988 a; Böhm, 2001; Damle, 1992).

7 Experimental results

7.1 Toxicokinetics and metabolism

No information available.

7.2 Acute and subacute toxicity

Acute toxicity

The acute toxicity data for chloroformic acid butyl ester following oral, inhalation and intraperitoneal administration are summarised in Table 1.

Beginning of Table 1

Table 1. Acute toxicity studies of chloroformic acid butyl ester						
Species, strain, sex[1]	Route	Dose (mg/ kg body weight or mg/m³)	Purity	Effect	Observation period	Reference
Rat	oral	ca. 1325 (1250 mm³), administered as a 10% aqueous tragacanth gum emulsion	n. d.	LD_{50}; dyspnoea, apathy; necropsy findings: corrosion of the gastrointestinal tract, adhesive inflammatory processes in the stomach wall	14 days	BASF, 1970
Rat	oral	ca. 2120 (2000 mm³), administered as a 20% aqueous tragacanth gum emulsion	n. d.	LD_{50}; dyspnoea, apathy; necropsy findings: corrosion of the gastrointestinal tract, adhesive inflammatory processes in the stomach wall	14 days	BASF, 1970
Rat, Sprague-Dawley, male, female	oral	2610, administered as an up to 63.2% formulation in olive oil	n. d.	LD_{50}; ≥ 1000 mg/kg body weight: dyspnoea, apathy, abnormal position, reeling, spastic gait, rough coat, diarrhoea, cyanosis and poor general condition; necropsy findings in the animals that died at dose levels of 2150 mg/kg body weight and above: acute atrial dilatation and acute congestive hyperaemia of the heart, acute emphysema of the lung, bloody sloughing in the glandular stomach and the forestomach (corrosive gastritis) and white intestinal mucosa (necrosis); terminal necropsy was without findings at dose levels ≤ 681 mg/kg body weight, but at higher dose levels fore-stomachs were hardened and thickened with some evaginations and the forestomach mucosa had removable encrustations	14 days	BASF, 1980
Rat	inhala-tion	atmosphere enriched or saturated with vapour at 20 °C, 3 minutes	n. d.	mortality: 12/12; vigorous escape behaviour, severe mucous membrane irritation, gasping; necropsy: considerable congestion and oedema of the lungs with hydrothorax	n. d.	BASF, 1970
Rat	inhala-tion	atmosphere enriched or saturated with vapour at 20 °C, 10 minutes	n. d.	mortality: 6/6; vigorous escape behaviour, severe mucous membrane irritation, gasping; necropsy findings: considerable congestion and oedema of the lungs with hydrothorax	n. d.	BASF, 1970
Rat	inhala-tion	ca. 1134 (200 ppm), one hour	n. d.	mortality: 4/10; dyspnoea; necropsy findings: pulmonary emphysema	n. d.	BASF, 1970
Mouse	intrape-ritoneal	ca. 13 (12.5 mm³), administered as a 0.5% aqueous tragacanth gum emulsion	n. d.	LD_{50}; dyspnoea, tremor, lying on the abdomen; necropsy findings: adhesions in the abdominal cavity	14 days	BASF, 1970

Table 1. Acute toxicity studies of chloroformic acid butyl ester						
Species, strain, sex[1]	Route	Dose (mg/ kg body weight or mg/m³)	Purity	Effect	Observation period	Reference
Mouse	intraperitoneal	ca. 53 (50 mm³), administered as a 0.1% aqueous tragacanth gum emulsion	n. d.	LD_{50}; dyspnoea, tremor, lying on the abdomen; necropsy findings: adhesions in the abdominal cavity	14 days	BASF, 1970
[1] where specified n. d. no data						

End of Table 1

When administered orally to rats, chloroformic acid butyl ester formulated in olive oil was found to have an LD_{50} value of 2610 mg/kg body weight, and thus proved to be of low toxicity (BASF, 1980). Upon inhalation, chloroformic acid butyl ester was clearly toxic. A 1-hour exposure to 200 ppm (approx. 1134 mg/m³) was lethal to 4 out of 10 rats while in inhalation hazard tests all rats died following exposure for 3 or 10 minutes to atmosphere which was enriched or saturated with vapour at 20 °C, respectively (BASF, 1970). Dyspnoea, apathy, abnormal position, reeling, spastic gait, rough coat, diarrhoea, cyanosis and poor general condition were described as signs of toxicity following oral administration (BASF, 1970, 1980). The signs of toxicity seen upon inhalation exposure included severe mucous membrane irritation, gasping and dyspnoea caused by the agent's corrosive effects (BASF, 1970). Necropsy findings also, in particular, included changes that were attributable to the chemical's corrosive properties, with inhalation exposure being associated with considerable congestion, emphysema and oedema of the lungs with hydrothorax and oral administration of lethal doses resulting in white intestinal mucosa (necrosis) and bloody sloughing in the glandular stomach and forestomach (corrosive gastritis). In addition, findings following oral administration of lethal doses also included acute atrial dilatation and acute congestive hyperaemia of the heart. Upon oral administration of sublethal doses > 681 mg/kg body weight, the rats' forestomachs were hardened and thickened, exhibiting some evaginations, and the forestomach mucosa had removable encrustations (BASF, 1970, 1980).

Chloroformic acid butyl ester undergoes hydrolysis in aqueous formulations (see also Section 5.6). Therefore, the oral LD_{50} values determined as approx. 1325 and 2120 mg/kg body weight in rats given aqueous tragacanth gum emulsions of chloroformic acid butyl ester and the intraperitoneal LD_{50}

values of 13 and 53 mg/kg body weight obtained for mice (BASF, 1970) are suitable only to a limited extent for the assessment of the chemical's acute toxicity, as the possibility can not be precluded that the test substance contained in those formulations was partially hydrolysed.

Subacute toxicity

The toxic effects of chloroformic acid butyl ester (98.9% pure) following subacute administration were investigated in Sprague-Dawley rats in an inhalation study that met the requirements set forth in OECD guideline No. 412. The exposure levels used in the 28-day study were determined on the basis of a preliminary range-finding study in which groups of 5 males and 5 females underwent whole-body exposure to chloroformic acid butyl ester at 0 (controls), 15, 50 or 150 mg/m³ for 6 hours/day on 5 consecutive days. The analysed concentration levels were 0 (controls), 16, 55 and 158 mg/m³. None of the animals died. The clinical signs of toxicity observed in the animals of the intermediate and top concentration groups included dose-dependent sneezing, rubbing of the snout with paws, closed or half-closed eyes, rapid breathing, licking the inside of the mouth and, between exposures, sniffing and noisy respiration. In addition, animals of the top concentration group exhibited prone position, lack of reaction to acoustic stimuli and, after the first exposure, hypoactivity. The males in the top dose group lost weight over the entire study period while the top-dose females showed initial body weight loss followed by body weight gain at a markedly reduced rate. The males and females in the intermediate dose group also exhibited retarded body weight gain. Food consumption was reduced in a concentration-dependent manner at all dose levels. Initially reduced, water consumption in the top dose group was increased at the end of the study. The males and females in the top dose group and the intermediate-dose females were found to have increased lung weights. The lungs of the top-dose animals and of one male from the intermediate dose group failed to collapse upon opening of the thoracic cavity (HRC, 1990).

In the subsequent 28-day study, groups of 5 males and 5 females with mean initial body weights of 155.4 and 130.9 g, respectively, underwent whole-body exposure to chloroformic acid butyl ester at nominal concentration levels of 0 (controls), 3, 15 or 30 mg/m³ for 6 hours/day on 5 days/week over a period of 4 weeks. The analysed concentration levels

were 0 (controls), 2.8, 10.0 and 28.2 mg/m³. None of the animals died in this study, either. The only clinical sign of toxicity observed in animals exposed to chloroformic acid butyl ester was piloerection, which was noted during exposure of the top concentration group. Minimal, concentration-independent differences in body weight gain and food consumption and in the haematology and blood biochemistry parameters investigated that were noted in animals treated with chloroformic acid butyl ester relative to controls were considered by the authors not to be of toxicological relevance. In all concentration groups, macroscopic examination was without remarkable findings. Only the males had significantly increased lung weights in the top concentration group. Histological examination of the lungs revealed pathological changes of the carina tracheae in the form of minimal focal epithelial hyperplasia in 1 out of 5 males and 3 out of 5 females together with minimal focal crowding of epithelial cells in a further 3 out of 5 males from the top concentration group. The *no observed adverse effect level* (NOAEL) for chloroformic acid butyl ester was 10 mg/m³ in both sexes (HRC, 1990).

7.3 Skin and mucous membrane effects

The acute skin irritancy of undiluted chloroformic acid butyl ester (purity not specified) was tested in rabbits. The chemical was applied to the dorsal skin for 1, 5 or 15 minutes or 20 hours, or to the skin of the ear for 20 hours. The observation period was 8 days. As shown in Table 2 below, the treated skin exhibited severe irritation and corrosion which was dependent upon the duration of exposure to chloroformic acid butyl ester. Necrosis occurred when the duration of exposure was ≥ 5 minutes (BASF, 1970).

Table 2. Irritant effects of chloroformic acid butyl ester on the rabbit skin and their dependence on the duration of exposure (based on BASF, 1970)

Application site	Duration of exposure	Findings after 24 hours	Findings after 8 days
Back	1 minute	severe reddening of the area of exposure and beyond	severe reddening, severe scaling
Back	5 minutes	severe reddening of the area of exposure and beyond, mild oedema	slight necrosis, slight reddening
Back	15 minutes	severe reddening of the area of exposure and beyond, mild oedema	slight necrosis, severe reddening
Back	20 hours	severe reddening of the area of exposure and beyond, severe oedema	severe necrosis, severe reddening
Ear	20 hours	slight necrosis, severe reddening, severe oedema	very severe necrosis

Chloroformic acid butyl ester exhibited corrosive effects to the eye in a rabbit study. One hour following instillation of 50 µl chloroformic acid butyl ester (purity not specified) into the rabbit eye, severe reddening, very severe oedema and severe clouding of the eye were observed, and 24 hours upon instillation there was severe oedema and bleeding as well persistent severe clouding. At 8 days, corrosive effects were even more manifest, with slight reddening, severe clouding, staphyloma, injected blood vessels and scars on the eyelids being observed. Negative controls treated with sodium chloride were without abnormal findings at all scheduled examinations (BASF, 1970).

7.4 Sensitisation

No information available.

7.5 Subchronic and chronic toxicity

No information available.

7.6 Genotoxicity

7.6.1 In vitro

In the Salmonella/microsome test carried out in accordance with OECD guideline No. 471, chloroformic acid butyl ester (purity > 99%) was devoid of mutagenicity both in the absence and presence of metabolic activation (S-9 mix from Aroclor 1254-induced rat liver). The study was conducted as a preincubation test in which *Salmonella typhimurium* strains TA 98, TA 100 and TA 1537 were incubated with chloroformic acid butyl ester at concentrations ranging from 0.0005 to 0.5 µl/plate, *Salmonella typhimurium* strain TA 1535 being exposed to chloroformic acid butyl ester levels of 0.0005 to 5.0 µl/plate. Ethanol served as the solvent. In the presence of S-9 mix concentration levels > 0.2 µl/plate exhibited bacteriotoxicity while in the absence of metabolic activation even lower concentrations > 0.005 µl/plate were toxic. Incubation of *Salmonella typhimurium* strains with chloroformic acid butyl ester did not result in any significant increase in revertant counts. Tests employing the positive controls 2-aminoanthracene,

N-methyl-N'-nitro-N-nitrosoguanidine, 4-nitro-o-phenylenediamine, 9-aminoacridine chloride monohydrate and dimethylcarbamyl chloride gave the expected results (BASF, 1988 b).

The chromosome-damaging effect of chloroformic acid butyl ester (98.9% pure) was assessed in a chromosome aberration assay conducted in accordance with OECD guideline No. 473 in V79 cells of the Chinese hamster both with and without metabolic activation (S-9 mix from Aroclor 1254-induced rat liver). The cells were incubated with chloroformic acid butyl ester at concentration levels of 0 (controls), 10 and 50 (for the 18-hour fixation only), 100, 200, 300 or 420 µg/ml for 4 hours and fixed 7, 18 or 28 hours after the start of incubation. A preliminary toxicity test had demonstrated that 420 µg/ml reduced the colony-forming ability, measured in terms of plating efficiency (PE), to 0% and 10.4% of the solvent control (acetone) in the absence and presence of S-9 mix, respectively. Reduced mitotic indices observed in the actual chromosome aberration assay in cultures incubated with 420 µg/ml both in the absence (all fixation intervals) and presence (fixation at 7 and 28 hours) of S-9 mix also demonstrated the cytotoxicity of that concentration. The mitotic indices determined for cultures treated with that concentration and fixed after 7 (± S-9 mix) or 28 hours (without S-9 mix only) were 44.1, 22.6 and 0.4% of the solvent controls, respectively, and were thus reduced to the extent that no further evaluation of the cultures was carried out. Per concentration level and fixation interval, 200 metaphases from two parallel cultures were scored for structural chromosome aberrations. Aberration rates observed in the absence of metabolic activation were not increased over untreated, solvent or historical controls at any of the fixation intervals. In the presence of metabolic activation, cultures treated with 420 µg/ml and fixed at 28 hours had a relative mitotic index of 87.1% and a PE value of 10.4%, and the aberration rate not including the gaps was significantly increased to 7.5% as compared with 1.5% in the solvent control. Chromatid exchanges were observed in 2.5% of the cells (0% in the solvent control). This positive result could not be reproduced in an independent second assay in which V79 cells were incubated with 350 or 420 µg/ml in the presence of S-9 mix, fixed at 28 hours and scored for structural chromosome aberrations. Both concentrations exhibited cytotoxicity, the respective mitotic indices being 70.5 and 69.1% and the respective PE values being 6.8 and 0% relative to the solvent control. Neither test showed any increase in the incidence of

polyploid metaphases. Additional cytotoxicity studies ("monolayer mass cultures") demonstrated the strong, concentration-dependent cytotoxic activity of chloroformic acid butyl ester. Respective cell survival rates at 28 hours after the start of incubation with chloroformic acid butyl ester at concentration levels of 350, 380 or 420 µg/ml were 84, 31 and 27% relative to control in the absence of metabolic activation, but only 26, 20 and 15% in the presence of metabolic activation. Discussing their results, the authors suggested that the increased aberration rate seen upon exposure to chloroformic acid butyl ester at 420 µg/ml in the first experiment was attributable to the strong cytotoxic effect of that concentration on the cells' genetic material rather than being an indication of the genotoxic potential of the test agent as such. Concentration levels of chloroformic acid butyl ester which reduced cell survival rates by more than 50% but not below 30% did not induce chromosome aberrations. Ethylmethane sulphonate (without S-9 mix) and cyclophosphamide (with S-9 mix) as positive controls induced chromosome changes as expected (CCR, 1990).

7.6.2 In vivo

No information available.

7.7 Carcinogenicity

No information available.

7.8 Reproductive toxicity

No information available.

7.9 Effects on the immune system

No information available.

7.10 Neurotoxicity

No information available.

7.11 Other effects

No information available.

8 Experience in humans

No information available.

9 Classifications and threshold limit values

In the Federal Republic of Germany, the Commission for the Investigation of Health Hazards of Chemical Compounds in the Work Area ("MAK-Kommission") of the Deutsche Forschungsgemeinschaft has established a MAK value (maximum workplace concentration) for chloroacetic acid butyl ester on the suggestion of BG Chemie. It was set in the List of MAK- and BAT Values 2004 at 0,2 ml/m³ (ppm, equivalent to 1,1 mg/m³). Furthermore, chloroacetic acid butyl ester has been assigned to pregnancy risk group C, i.e. substances for which "there is no reason to fear a risk of damage to the embryo or foetus when MAK and BAT values are observed" (DFG, 2004; Greim, 2003).

Australia and the United Kingdom have set the threshold limit value for chloroformic acid butyl ester at 5.6 mg/m³ (BIA, 2004).

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