Interim Secretariat for the Rotterdam Convention on the Prior Informed Consent Procedure for Certain Hazardous Chemicals and Pesticides in International Trade

UNEP

FORM FOR NOTIFICATION OF FINAL REGULATORY ACTION TO BAN OR SEVERELY RESTRICT A CHEMICAL

IMPORTANT: See instructions before filling in the form

COUNTRY: SWITZERLAND

PART I: PROPERTIES, IDENTIFICATION AND USES

1.	**IDENTITY OF CHEMICAL**	
1.1	**Common name**	Carbon Tetrachloride
1.2	**Chemical name according to an internationally recognized nomenclature (e.g. IUPAC), where such nomenclature exists**	Carbon Tetrachloride Tetrachloromethane (CAS chemical name)
1.3	**Trade names and names of preparations**	Carbona, carbon chloride, ENT 4,705, ENT 27164, Tetrachloromethane, Tetrachlorocarbon, Tetra, Carbon Tet, Methane tetrachloride, Perchloromethane, Benzinoform, Fasciolin, Flukoids, Freon 10, Halon 104, Necatorina, Necatorine, R 10, RCRA Waste Number U211, Tetrachloormetaan, Tetrafinol, Tetraform, Tetrasol, UN 1846, Univerm, Vermoestricid
1.4	**Code numbers**	
1.4.1	**CAS number**	56-23-5
1.4.2	**Harmonized System customs code**	2903.14.00
1.4.3	**Other numbers (specify the numbering system)**	FG4900000 (RTECS No.), 602-008-00-5 (EC No.)

1.5	**Indication regarding previous notification on this chemical, if any**	
1.5.1	θ This is a first time notification of final regulatory action on this chemical.	

PLEASE RETURN THE COMPLETED FORM TO:

Interim Secretariat for the Rotterdam Convention
Plant Protection Service
Plant Production and Protection Division, FAO
Viale delle Terme di Caracalla
00100 Rome, Italy

Tel: (+39 06) 5705 3441
Fax: (+39 06) 5705 6347
E-mail: pic@fao.org

OR

Interim Secretariat for the Rotterdam Convention
UNEP Chemicals

11-13, Chemin des Anémones
CH – 1219 Châtelaine, Geneva, Switzerland

Tel: (+41 22) 917 8183
Fax: (+41 22) 797 3460
E-mail: pic@unep.ch

1.5.2	✓ This is a modification of a previous notification of final regulatory action on this chemical. The sections modified are: _____ ✓ This notification replaces all previously submitted notifications on this chemical.
	Date of issue of the previous notification: ___1993_____

1.6 Information on hazard classification where the chemical is subject to classification requirements	
International classification systems	**Hazard class**
IARC	2B (The agent (mixture) is possibly carcinogenic to humans. The exposure circumstance entails exposures that are possibly carcinogenic to humans. Inadequate evidence in humans, sufficient evidence in experimental animals for the carcinogenicity of carbon tetrachloride)
IMDG	Marine pollutant
UN Vienna Convention for the Protection of the Ozone Layer & Montreal Protocol on Substances that Deplete the Ozone Layer	Ozone-depleting potential: 1.1
UN hazard class (pack group)	6.1: poisonous substance (II), 1846 (UN No.)
Other classification systems	**Hazard class**
EU classification	Symbol: T (toxic), N (environmentally dangerous substance) R23/24/25 toxic by inhalation, in contact with skin and if swallowed R40 limited evidence of a carcinogenic effect R48 toxic: danger of serious damage to health by prolonged exposure through inhalation R52/53 harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment R59 dangerous for the ozone layer
US EPA	probable human carcinogen of low carcinogenic hazard
Switzerland	Poison Class 1* (prohibited for general use)

1.7 Use or uses of the chemical	
1.7.1	✓ **Pesticide**
	Describe the uses of the chemical as a pesticide in your country:
	Carbon Tetrachloride has been used as an insecticide.
1.7.2	✓ **Industrial**
	Describe the industrial uses of the chemical in your country:

Carbon Tetrachloride has been used as a solvent.

1.8 Properties

1.8.1 Description of physico-chemical properties of the chemical

Carbon tetrachloride (empirical formula: CCl_4; molecular mass 153.8) is a colourless liquid with a characteristic sweet non-irritant odour. Carbon tetrachloride has a boiling point of 76.5°C, and a melting point of -23°C. Relative density: 1.59 g/ml; solubility in water 0.1 g/l at 20°C (poor); miscible with most aliphatic solvents; vapour pressure: 12.2 kPa at 20°C; relative vapour density: 5.3; relative density of the vapour/air-mixture at 20°C: 1.5; Henry's law constant at 24.8°C $2.3x10^{-2}$ atm/m^3/mol. Log P_{ow} 2.64; log K_{oc} 2.04. Carbon tetrachloride is non-flammable and stable in the presence of air and light.

UNEP/ILO/WHO (1998) International Programme on Chemical Safety, Health and Safety Guide No. 108: Carbon Tetrachloride (http://www.inchem.org/documents/hsg/hsg/hsg108.htm)
WHO (1999) International Programme on Chemical Safety, Environmental Health Criteria No. 208: Carbon Tetrachloride (http://www.inchem.org/documents/ehc/ehc/ehc208.htm)

1.8.2 Description of toxicological properties of the chemical

Mouse LD_{50} (ip) 3350-4676 mg/kg body weight
Rat LD_{50} (oral) 10054 mg/kg body weight
Rat LD_{50} (ip) 3029-6603 mg/kg body weight
Rat 100% death rate after inhalation of 121600 mg/m^3 for 2.2 h or 46700 mg/m^3 for 8 h
Guinea pig and rabbit LD_{50} (dermal) >15 g/kg body weight
Carbon tetrachloride accumulates in fat, bone marrow, white matter of brain, spinal cord and nerves, liver, kidney, salivary glands, and gastrointestinal mucosa (mouse, inhalation); in liver, kidney, brain, muscle, and blood (rat, oral); in brain, heart, liver, and blood (Beagle dogs, inhalation).
Carbon tetrachloride is metabolized by CYP2E1 and CYP2B1/2B2 to a trichloromethyl radical, which may undergo reductive or oxidative biotransformation. The trichloromethyl radical may react with molecular oxygen, resulting in the formation of trichloromethylperoxyl radicals. This radical may react with lipids, causing lipid peroxidation along with the production of 4-hydroxyalkenals. It is also presumed that the trichloromethylperoxyl radical will react further to produce phosgene, which may further react with tissue macromolecules or with water, finally producing hydrochloric acid and carbon dioxide.
Major effects in mice resulting from carbon tetrachloride exposure by acute oral exposure are changes in liver enzyme levels and other liver effects such as decreases in protein, glucose, phospholipids, DNA, RNA concentrations, increases in triglycerides, glycogen and free and esterified cholesterol concentrations. Centrilobular necrosis was seen in the low-dose (32 mg/kg body weight, 15-327 h) and mild midzonal necrosis in the mid- (797 mg/kg body weight) and high-dose (2391 mg/kg body weight) group. Rats showed dose-related increases in liver and serum alanine aminotransferase (ALAT), liver tyrosine transaminase and alkaline phosphatase activities after a single oral dose of 797-3188 mg/kg body weight. Centrilobular hepatocellular necrosis was observed in 2/4 monkeys 24 h after oral administration of a single dose of carbon tetrachloride.
Exposure by inhalation produced Clara cell lesions in mice (0.46 or 0.92 mmol/litre air for 1 h, 1.84 mmol/litre air for 12 min., and 3.68 mmol/litre air for 2 min.) Rats showed increases in aspartate aminotransferase (ASAT), ALAT, sorbitol dehydrogenase (SDH) and glutamate dehydrogenase activities in the serum 24 h post-inhalation exposure at >3404 mg/m^3 air for 4 h.
Long-term exposure (1.3 ml/kg body weight of 50% CCl_4, sc) resulted in severe cirrhosis in Sprague-Dawley rats (they did not develop carcinomas), and hepatocellular carcinoma and hyperplastic nodules in 8/13 Osborne-Mendel rats and in 12/15 Japanese rats. BDF1 mice which were exposed up to 801.25 mg/m^3 CCl_4 (2 years, whole body), showed a significant decrease in survival, changes in hematology, blood biochemistry, and urinalysis, changes in liver, kidney, spleen at ≥160.25 mg/m^3 CCl_4.
Carbon tetrachloride can be considered a reproductive toxicant; it is however not embryotoxic or teratogenic.
Findings from mutagenicity studies are equivocal; positive findings such as strand-breakage and

aneuploidy may be the consequence of nuclear protein or DNA damage induced secondarily to CCl_4 toxicity.

Carbon tetrachloride was shown to be immunotoxic in $B6C3F_1$ female mice, resulting in a suppression of both humoral and cell-mediated immune functions. The T-cell dependent antibody formation against sheep red blood cell was shown to be a very sensitive parameter. CCl_4 was toxic at all doses (25-5000 mg/kg body weight) tested, independent of the route (ip or oral). Rats showed no immunotoxic effects up to concentrations of 40 mg/kg body weight. T-cell dependent immune processes seem to be more sensitive than B-cells.

Controlled studies in humans showed no adverse effects of carbon tetrachloride. Cases of poisonings have resulted from accidental, mostly of CCl_4 vapours, or suicidal ingestion. In humans it seems to be toxic to liver and kidney. Concentrations of 64.1-512.8 mg/m^3 for 3-4 hours have no adverse effects, at higher concentrations nausea, headache, vomiting, rapid pulse, rapid respiration, sleepiness, dizziness, unconsciousness and immediate death occur. The lethal oral dose (1.5 to 355 ml CCl_4) varies widely due to individual differences, actual doses are, however, often difficult to ascertain. Non-cancer epidemiology shows significant effects (ALAT, ASAT, alkaline phosphatase, γ-GT, glutamate dehydrogenase and others) in workers exposed to air CCl_4 concentrations of ≥6.4 mg/m^3. Cancer epidemiology has not established an association between CCl_4 exposure and increased risk of mortality, neoplasia or liver disease.

UNEP/ILO/WHO (1998) International Programme on Chemical Safety, Health and Safety Guide No. 108: Carbon Tetrachloride (http://www.inchem.org/documents/hsg/hsg/hsg108.htm)
WHO (1999) International Programme on Chemical Safety, Environmental Health Criteria No. 208: Carbon Tetrachloride (http://www.inchem.org/documents/ehc/ehc/ehc208.htm)
International Agency for Research on Cancer (IARC) – Summaries & Evaluations (1999), Carbon Tetrachloride (Group 2B) (http://www.inchem.org/documents/iarc/vol71/011-carbontetrac.html)

1.8.3 Description of ecotoxicological properties of the chemical

Carbon tetrachoride is low to moderately toxic to bacteria (however: methanogenic bacteria IC_{50} 6.4 mg/l), protozoa and algae.

Aquatic invertebrates: *Daphnia magna* acute LC_{50} (24/48 h, static) 28 - >770 mg/l; no effect on the development of sea urchins.

Aquatic vertebrates: Golden Orfe (*Leuciscus idus melanotus*) LC_{50} (48 h) 13 to 472 mg/l; Dab (*Limanda limanda*) LC_{50} 50 mg/l

Rainbow trout (*Oncorhynchus mykiss*) no effects at 1-80 mg/l for up to 336 h under semi-static renewal

Common bullfrog (*Rana catesbiana*) LC_{50} 0.92 mg/l

CCL_4 seems to be more toxic to embryo-larval stages of fish and amphibians than adults. *O. mykiss* LC_{50} (27 d) 1.97 mg/l; most sensitive species: *R. catesbiana* 1% incidence of teratic larvae at 60 µg/l, 17% at 7.8 mg/l.

Earthworms (*Eisenia foetida*) LC_{50} 160 µg/cm^2 on filter paper in glass vials.

Ozone depletion

Carbon tetrachloride is subject to the UN Vienna Convention for the Protection of the Ozone Layer & Montreal Protocol on Substances that Deplete the Ozone Layer and is listed in Annex B, Group II. This means that by January 1, 1996 Switzerland will have reduced its carbon tetrachloride production and consumption by 100% (with possible essential use exemptions). Global atmospheric emissions of carbon tetrachloride in 1996 were estimated as 41,000 tonnes, of which some 26,000 tonnes originate from carbon tetrachloride production in Article 5(1) Parties (developing countries) and Countries with Economies in Transition. Emissions of carbon tetrachloride can be technically and economically reduced from both feed stock and process agent uses, although in some cases, alternatives to carbon tetrachloride use may not be available. Emissions from carbon tetrachloride used as a final product are estimated to be in the range of 11500 to 12400 metric tonnes. Doubts exist, however, as to the validity of the reported data. But it seems that industrialized countries have phased out production and consumption of carbon tetrachloride. In Article 5(1) countries and Countries with Economies in Transition there is significant trade, eg India imports 17000-20000 metric tonnes/year. The global lifetime of carbon tetrachloride is currently estimated to be 23-42 years. Global surface mixing ratios (tropospheric concentrations) have decreased since about 1990; mixing ratios in 2000 were between 95-100 ppt.

Health effects: potential health effects of ozone depletion are the result of elevated levels of ambient UV-B radiation. UV-B radiation is a risk factor for certain types of cataract, squamous cell carcinoma, it contributes to the formation of basal cell carcinoma and cutaneous melanoma and possibly to immune suppression. Since the risk of increased UV-B radiation is largely dependent on human behavior, it is difficult to quantify. Further complications stem from the emerging possibilities regarding interactions between ozone depletion and global climate change. A study in Punta Arenas, Chile, showed a relationship between episodes of ozone depletion, increased terrestrial UV-B radiation and sunburn during the spring months. The Antarctic "ozone hole" passes over Punta Arenas each spring and a rise in the number of sunburn cases after sudden ozone depletion, coinciding with Sunday outdoor recreational activities could be documented. In the skin UV-B radiation causes specific DNA damage, leads to the generation of reactive oxygen species, point mutations, DNA deletions and micronuclei. Basal cell carcinoma, squamous cell carcinoma, and cutaneous melanoma are all related to UV exposure. Concerning cutaneous melanoma, exposure in childhood seems to be a far higher risk factor than chronic exposure in adulthood. Solar UV radiation also seems to be a risk factor in the development of non-Hodgkin´s Lymphoma and chronic lymphocytic leukemia.

Environmental effects: Environmental damage to terrestrial or aquatic ecosystems due to increased UV-B radiation is difficult to observe or to quantify. With respect to terrestrial ecosystems, meta-analysis of >60 studies showed enhancement in some plant characteristics (plant height, leaf area, and shoot mass) while most studies reported decreases in these characteristics. There are also reports of studies where solar UV-B promoted plant growth. A potentially important phenomenon is that small effects of UV-B radiation might accumulate to produce larger effects in subsequent years in perennial plants. This is, however, being discussed controversially since cumulative effects, e.g. in subarctic heath perennials, were apparent for some traits of some species but not for others. Futhermore cumulative effects disappeared over a longer period of time. High UV-B may also affect genetic stability of plants causing long-term heritable effects, with a high frequency of deleterious mutations, such as the activation of "mutatator transposons" in maize. With respect to insect herbivory, enhanced UV-B radiation seems to lead to reduced herbivory and/or insect growth, mostly mediated through the host plant. Concerning aquatic ecosystems there is general consensus that solar UV negatively affects aquatic organisms. Reductions in productivity, impaired reproduction and development, and increased mutation rate have been shown for phytoplankton, macroalgae, fish eggs and larvae, zooplankton and primary and secondary consumers exposed to UV radiation. Decreases in biomass productivity due to enhanced UV-B radiation are relayed through all levels of the food web; quantitation of such effects is, however, difficult to perform. Species interactions and ecosystem dynamics are difficult to evaluate, model and predict. Feedback mechanisms between aquatic ecosystems, physical factors and atmospheric and oceanic circulation have significant impact on primary productivity and ecosystem integrity, but are not well understood and difficult to predict. Bacterioplankton does not seem to be very sensitive to enhanced UV-B radiation and cyanobacteria have been shown to be able to protect themselves with mycosporine-like amino acids, scytonemin, carotenoids, superoxide dismutase, and migration to habitats with reduced radiation. Phytoplankton communities have been shown to be quite sensitive to solar ambient UV. UV impairs photosynthesis, nitrogen metabolism, bleaches photosynthetic pigments, and induces DNA damage. There are, however, efficient repair and protection mechanisms in phytoplankter, including the xanthophyll cycle in photosynthesis, screening pigment

production, synthesis of antioxidants and DNA repair. Studies in Patagonia, Argentina, which is occasionally under the influence of the Antarctic "ozone hole", showed that photosynthetic inhibition in phytoplankton varies considerably between different environments and depends on the optical depth of the water column. Macroalgae and seagrass are important biomass producers, are exploited commercially and form habitats for larval stages of fish, shrimp and other crustaceans. Both long- and short-term epxosure to solar radiation inhibits growth in adult stages of several species of macroalgae. Susceptibility to UV is, however, highly variable among species which results in a specific depth distribution. UV exposure is considered to be a major stress factor for zooplankton, resulting in vertical migration into lower and darker water layers as well as in the production of UV-protective pigments such as melanin and carotenoids and mycosporine-like amino acids. Both the Arctic and Antarctic ecosystems may be affected by increased ambient UV-B radiation. The effects of increased UV-B radiation may, however, be masked by other climatic effects. It has for example been shown that large spatial and temporal interannual variability in cloud cover may augment or reduce increases in UV-B radiation. Arctic marine phytoplankton may be more sensitive to increased UV radiation than its Antarctic counterpart. In both ecosystems, however, a shift has been observed in species composition to diatom-dominated assemblages, which are capable of synthesizing UV screening compounds. Furthermore, results indicate that currently measured UV levels do not affect high Arctic macroalgal communities.

UNEP/ILO/WHO (1998) International Programme on Chemical Safety, Health and Safety Guide No. 108: Carbon Tetrachloride (http://www.inchem.org/documents/hsg/hsg/hsg108.htm)
WHO (1999) International Programme on Chemical Safety, Environmental Health Criteria No. 208: Carbon Tetrachloride (http://www.inchem.org/documents/ehc/ehc/ehc208.htm)
UNEP (2000) The Montreal Protocol on Substances that Deplete the Ozone Layer as adjusted and/or amended in London 1990, Copenhagen 1992, Vienna 1995, Montreal 1997, Beijing 1999 (ISBN: 92-807-1888-6) (http://www.unep.org/ozone)
UNEP, Ozone Secretariat (1999) Synthesis of the Reports of the Scientific, Environmental Effects, and Technology and Economic Assessment Panels of the Montreal Protocol: A Decade of Assessments for Decision Makers Regarding the Protection of the Ozone Layer: 1988-1999
Bundesministerium für wirtschaftliche Zusammenarbeit und Entwicklung & Deutsche Gesellschaft für Technische Zusammenarbeit (GTZ) GmbH (2001) Production and Consumption of Ozone Depleting Substances 1986-1999
UNEP (2003) Montreal Protocol on Substances that Deplete the Ozone Layer, Environmental Efects of Ozone Depletion and its Interactions with Climate Change: 2002 Assessment (http://www.unep.org/ozone)

PART II: FINAL REGULATORY ACTION

2.	**FINAL REGULATORY ACTION**
2.1	**The chemical is: ✓ banned** **OR** 3 **severely restricted**
2.2	**Information specific to the final regulatory action**
2.2.1	**Summary of the final regulatory action**

Carbon tetrachloride is listed as an ozone depleting substance in Annex 3.4, Number 1 of the Ordinance relating to Environmentally Hazardous Substances.
The use, production, import and export of ozone depleting substances (as well as simple mixtures and products containing ozone depleting substances if they are in containers used solely to transport or store these substances,) is prohibited.
Exception: recycled ozone depleting substances which are not chemically changed by the process
Exception: manufacture of products or articles which may be supplied or imported in accordance with the provisions of Annexes 4.9 (compressed gas containers), 4.11 (plastics), 4.14 (solvents), 4.15 (refrigerants), and 4.16 (extinguishing agents). This applies only to imports / exports from / to States which adhere to the provisions of the Montreal Protocol of 16 September 1987, and its amendments of 29 June 1990, 25 November 1992, 17 September 1997, and 3 December 1999

2.2.2	**Reference to the regulatory document**

	Ordinance relating to Environmentally Hazardous Substances (Ordinance on Substances, Osubst) of 9 June 1986; update 3 June 2003; Annex 3.4 (SR 814.013) http://www.admin.ch/ch/d/sr/c814_013.html (German) http://www.admin.ch/ch/f/rs/c814_013.html (French) http://www.admin.ch/ch/i/rs/c814_013.html (Italian)
2.2.3	**Date of entry into force of the final regulatory action** 14.08.1991

2.3	**Was the final regulatory action based on a risk or hazard evaluation?**	✓**Yes** θ **No**
	If yes, give information on such evaluation The hazard/risk evaluation was based on the Montreal Protocol which Switzerland has ratified and is thus obliged to enforce nationally.	
	Reference to the relevant documentation There is no specific document.	

2.4	**Reasons for the final regulatory action**	
2.4.1	**Is the reason for the final regulatory action relevant to the human health?**	✓**Yes** θ **No**
	If yes, give summary of the known hazards and risks presented by the chemical to human health, including the health of consumers and workers	
	Reference to the relevant documentation	
	Expected effect of the final regulatory action The reduction in CarbonTetrachloride emission, together with the reduction in emissions of other ozone depleting substances, is expected to reduce the risk of increase UV radiation due to depletion of stratospheric ozone ("ozone hole").	

2.4.2	**Is the reason for the final regulatory action relevant to the environment?**	✓**Yes** θ **No**
	If yes, give summary of the known hazards and risks to the environment	

Reference to the relevant documentation

Expected effect of the final regulatory action

The reduction in CarbonTetrachloride emission, together with the reduction in emissions of other ozone depleting substances, is expected to reduce the risk of increase UV radiation due to depletion of stratospheric ozone ("ozone hole").

2.5 Category or categories where the final regulatory action has been taken

2.5.1 Final regulatory action has been taken for the chemical category ✓ **Industrial**

Use or uses prohibited by the final regulatory action

Ozone depleting substances shall not be used.
The following is prohibited:

a. the manufacture of ozone depleting substances; this prohibition shall not apply to the manufacture of ozone depleting substances by means of recycling used ozone depleting substances, if ozone depleting substances are not chemically changed by this process;
b. the import and export of ozone depleting substances; this prohibition shall not apply to imports from States and exports to States which adhere to the provisions of the Montreal Protocol of 16 September 1987 (SR 814.021) to phase out Ozone Depleting Substances (hereinafter Protocol), approved by Switzerland;
c. the import of products and articles containing ozone depleting substances; except for products and articles, which may be imported in accordance with the provisions of Annexes 4.9, 4.11, 4.14, 4.15 and 4.16;
d. the import of products and articles containing ozone depleting substances or manufactured using ozone depleting substances and listed in an appendix to the Protocol ; subject to Letter c, this prohibition shall not apply to imports from States which adhere to the provisions of the Protocol approved by Switzerland

Use or uses that remain allowed

Exceptions exist for the following purposes:

a. to manufacture products or articles which may be supplied or imported in accordance with the provisions of Number 22 and Annexes 4.9, 4.11, 4.14, 4.15 and 4.16;
b. for use as intermediate products for further chemical conversion;
c. for research purposes;
d. pest control with a permit under Article 35 of the Ordinance on Toxic Substances of 19 September 1983 (SR 813.01)

The Federal Agency may authorize limited exemptions for other uses, provided that:

a. according to the state of the art, no replacement is available for ozone depleting substances or for the products and articles manufactured using ozone depleting substances, and
b. no more than the minimum amount of ozone depleting substances necessary for the desired purpose is used

2.5.2	**Final regulatory action has been taken for the chemical category**	✓ **Pesticide**
	Formulation(s) and use or uses prohibited by the final regulatory action	
	All uses and formulations are prohibited.	
	Formulation(s) and use or uses that remain allowed	

2.5.3 **Estimated quantity of the chemical produced, imported, exported and used, where available.**		
	Quantity per year (MT)	**Year**
Produced		
Imported		
Exported		
Used		

2.6	**Indication, to the extent possible, of the likely relevance of the final regulatory action to other states and regions**

2.7	**Other relevant information that may cover:**
2.7.1	**Assessment of socio-economic effects of the final regulatory action**

2.7.2	**Information on alternatives and their relative risks**
2.7.3	**Relevant additional information**

PART III : GOVERNMENT AUTHORITIES

Ministry/Department and authority responsible for issuing/enforcing the final regulatory action	
Institution	Federal Department of Environment, Transport, Energy and Communications
Address	Parliament Building North 3000 Berne, Switzerland
Telephone	+41 31 3225512 (General Secretary)
Telefax	+41 31 3242692 (General Secretary)
E-mail address	
Designated National Authority	
Institution	Swiss Agency for the Environment, Forests and Landscape Hazardous Substances, Soil and Biotechnology Division
Address	3003 Berne Switzerland
Name of person in charge	Prof. Dr. Georg Karlaganis
Position of person in charge	Head
Telephone	+41 31 3226955
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Date, signature of DNA and official seal: 09 September 2004

Swiss Agency for the
Environment, Forests
and Landscape
Substances, Soil and
Biotechnology Division
CH-3003 Bern