INFORMATION

Cases of Poisoning Reported by Physicians

2005

BfR

Risiken erkennen – Gesundheit schützen

Cases of Poisoning Reported by Physicians in 2005
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Cases of Poisoning Reported by Physicians 2005

Centre for Documentation and Assessment of Poisonings
at the Federal Institute for Risk Assessment – 12th Report (2005)

A. Hahn, H. Michalak, K. Begemann, K. Preußner, R. Burger, H. Meyer

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1 Introduction

1.1 Legal basis and activities of the Centre
With the Chemicals Act (ChemG), legislation in the Federal Republic of Germany has provided a basis "to protect humans and the environment from harmful effects of dangerous substances and preparations, particularly to make them recognizable, to avert and to prevent the development of such effects" (according to §1).

For a realistic assessment of risks for human health, importance has been increasingly given to the knowledge of data on human toxicology that can be obtained from the evaluation of cases of poisoning in humans. This is why legislation has introduced compulsory notification of poisonings by attending physicians from 1 August 1990, by the first amendment to the ChemG (§16e).

A physician who is consulted for treatment or evaluation of sequelae of diseases caused by chemical substances or products is obliged to submit essential data on poisonings to the Centre for Documentation and Assessment of Poisonings at the Federal Institute for Risk Assessment (BfR).

According to the Chemicals Act, illnesses or suspected poisonings associated with the following substances are notifiable:

- Chemical substances and products used in the household, e.g. detergents and cleansing agents, hobby and DIY articles;
- Cosmetics;
- Pest control products;
- Plant protection products;
- Wood preservatives;
- Chemicals used at the workplace;
- Harmful chemical substances found in the environment; and
- Plants / animals.

Within the meaning of the Chemicals Act, the term of poisoning designates all cases in which health impairment has occurred, including suspected cases of poisoning. Under the Act, also the poison information and treatment centres (Poison Control Centres, PCC) were subjected to compulsory reporting of their knowledge (of general importance) gained in the context of their activities.

1.2 Processing of reports received
Reports received on health impairment associated with chemicals are subjected to an assessment procedure resulting in the rating of a possible causal relationship between the toxicant and the manifestations observed, as well as other conclusions. Such relationship may be classified as "possible", "probable", "confirmed", "absent" or "cannot be assessed". The rules applied in the assessment of individual cases have been described in detail in earlier annual reports.

The estimation of toxic risks in humans is based on differentiated analyses and evaluation of the data on cases. For these purposes, the data on cases in humans are continuously documented in the form of case data sets and case reports. Information on identified risks is passed on to the responsible ministries, manufacturers and industrial associations in the form of rapid communications or annual summarizing reports by means of the product information system PRINS (see Chapter 2.3). At the same time, the responsible manufacturers or distributors are requested to submit information on the measures envisaged by them to improve product safety.

The BfR publishes annual reports on the knowledge gained from the cases of poisoning reported by physicians. These publications are available on request by writing to Pressestelle, Bundesinstitut für Risikobewertung, Thielallee

88-92, D-14195 Berlin, Germany, and they have also been published as electronic documents on the internet (www.bfr.bund.de).

In Fig. 1, these tasks and procedures are shown in graphical form.

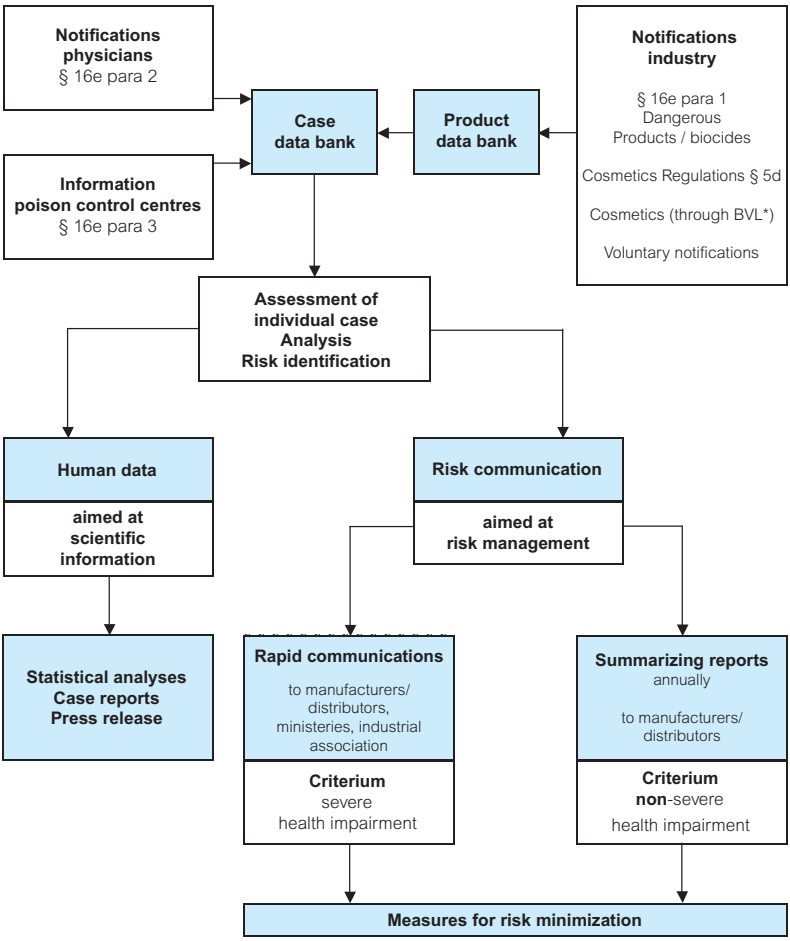


Fig. 1: Terms of reference of the Centre for Documentation and Assessment of Poisonings

*BVL: Federal Office for Consumer Protection and Food Safety

1.3 Product data bank (poison information data bank)

1.3.1 Figures

Until the end of 2005, 205 949 documents on individual products had been recorded in the poison information data bank maintained by the BfR, which can be accessed by the Poison Control Centres (PCC) in Germany, thus supporting their activities in providing consultation and treatment in cases of poisoning. The structure of the data bank and the different types of product data sets have been described in detail in earlier reports. In 2005, the number of reports on products received by the Centre for Documentation and Assessment of Poisonings at the BfR increased by 20 462 documents (Fig. 2).

1.3.2 Collaboration between the BfR, industry and reporting Poison Control Centres

The major part of product data on dangerous preparations and biocidal products as well as of the voluntary reports by manufacturers and distributors received by the BfR is still submitted on paper forms. For cosmetics, more than 90 % of product data are submitted in electronic form. Since 2005, the recording of reports on cosmetics has been assigned to the Federal Office for Consumer Protection and Food Safety (BVL), which resulted from the subdivision of the former Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV) into the two successor institutions, BfR and BVL. From the BVL, the data are returned to the BfR at monthly intervals and from there, transmitted to the PCCs together with the other product data.

This has resulted in the regrettable fact that the principle of product data processing being in the hands of a single institution was abandoned. Six months of experience have already shown data sets (e.g. addresses) to diverge.

Until the end of the year 2005, 1 500 product formulations were received by the BfR through the electronic information procedure (EMIL) elaborated in cooperation with the Poison Control Centres and the German Cosmetics, Toiletries, Perfumes, and Detergents Association

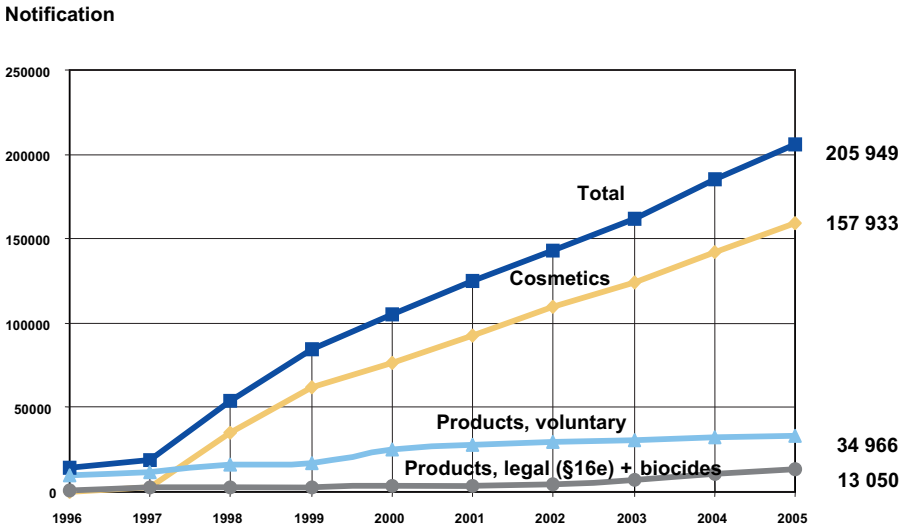


Fig. 2: Notifications on products received since 1996 and transmission of information to the German Poison Control Centres

(Industrieverband Körperpflege und Waschmittel e.V. – IKW), a system which is guided by the notification procedure for cosmetics. Within the framework of EMIL, the data are transmitted by floppy disk and email. The envisaged end point of electronic data transmission is online data recording and data transmission in the framework of a central poison information data bank.

Of the dangerous preparations and biocidal products notifiable under §16e para 1 of the Chemicals Act, 13 050 data sets have been transmitted to the Poison Control Centres so far. 5 277 of these refer to dangerous preparations and 7 773, to biocides.

Within a period of three years, no more than about 350 notifications of products were received by the centres in electronic form through the product reporting system for voluntary notifications designed in the context of the TDI research project (TDI – Toxicological Documentation and Information Network) coordinated by the GIZ-Nord Poison Centre (covering the northern federal Länder of Bremen, Hamburg, Lower Saxony and Schleswig-Holstein). In contrast, more than 2 700 voluntary notifications were received and processed by the BfR in the last three years.

This relatively low number of notifications has raised doubt whether the considerable effort made to establish this additional reporting variant has been worth the efforts made. Furthermore, it has to be taken into account that a considerable part of voluntary reports submitted to individual PCCs is not made available in the data network because transmission of such data has been excluded in bilateral agreements made between PCCs and reporting manufacturers. As a consequence, the research project has failed to meet its envisaged objectives such as an exchange of product information between the PCCs and a division of labour. Experience made in the last six years has shown that obviously, central data processing at the BfR and subsequent transmission of formulations to the PCCs is a more reasonable and effective way to provide the PCCs with product information for the provision of emergency poisoning management advice. The research project has ended in June 2006. The corresponding results will be discussed in the next annual report.

2 Case reports by physicians

2.1 Evaluation of reports

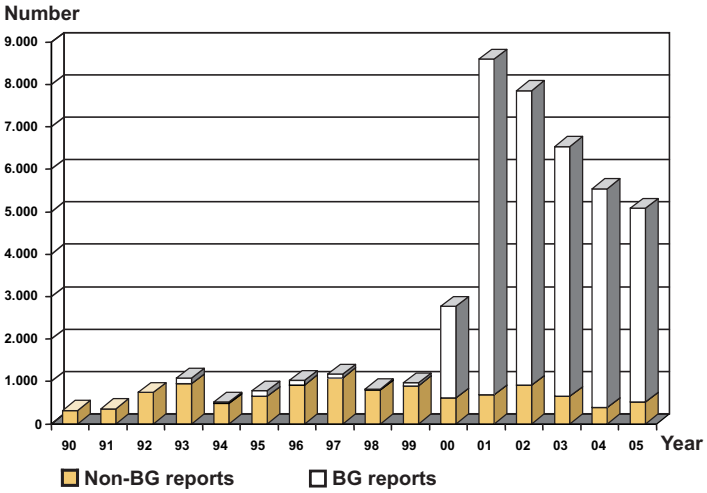


Fig. 3: Cases reported (BG reports 100 % = 4 573; non-BG reports 100 % = 509)
BG: Berufsgenossenschaften – institutions for statutory accident insurance and prevention for trade and industry in Germany

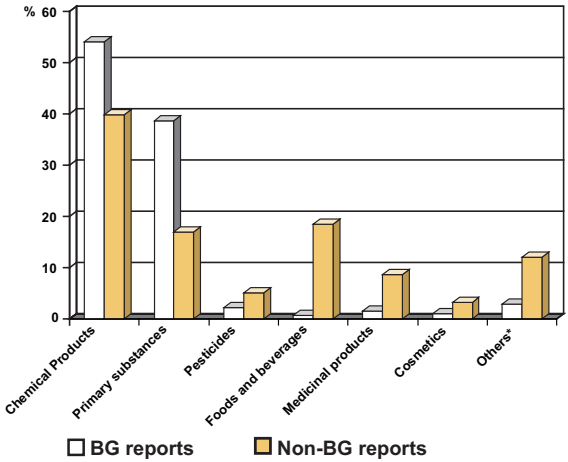
During the period between 1 August 1990, i.e. the beginning of the compulsory notification, and 31 December 2005, altogether 44 154 reports on cases of health impairment, poisoning or suspected cases of poisoning were received by the BfR. In 2005, i.e. the reporting year considered, 5 082 notifications were received (Fig. 3).

The increase in the number of notifications received in 2000 was due to an agreement with the Berufsgenossenschaften. According to this agreement, all notifications on cases of acute health impairment after contact with chemicals or chemical products are directly reported by the Berufsgenossenschaften to the BfR.

2.2 Reports on cases of poisoning in 2005

2.2.1 Origin

In 2005, 4 573 cases, i.e. 90 % of all cases notified, were reported by the Berufsgenossenschaften. The remaining 509 notifications (10 %) were essentially submitted by hospitals and medical practitioners. Single notifications were also received from pharmacies, Poison Control Centres, the Arzneimittelkommission der Deutschen Ärzteschaft (Drug Commission of the German Medical Profession), the Arzneimittelkommission der Deutschen Apotheker (Drug Commission of the German Pharmacists) and others.



* Others: Agrochemicals, industrial accidents, fungi, animals, veterinary medicines, weapons, others

Fig. 4: Spectrum of cases reported (BG reports 100 % = 4 573; non-BG reports 100 % = 509)

2.2.2 Spectrum of cases reported

Fig. 4 provides a synoptic view of the spectrum of product groups involved in the cases reported. Among the total of cases reported by the Berufsgenossenschaften, those of poisoning by chemical products and primary substances have remained in top position. All other product groups played a minor role, with shares of 2 % each, or less.

As expected, the spectrum of substances and products involved in cases of poisoning is different in the reports received from the Berufsgenossenschaften and in those received from hospitals and medical practitioners. Also among the latter, notifications related to chemical products ranked first in the reporting year. These were followed, at a clear distance, by the group of foods and beverages. Next in the ranking were cases of health impairment caused by primary substances being subject to compulsory notification like chemical products, and medicinal products that were reported although not being subject to compulsory notification.

For a detailed list of toxicants in tabular form see Annex. In this table, the cases reported in

	BG reports (100 % = 4 573 reports)		Non-BG reports (100 % = 509 reports)	
Chemical products	54.1 %	(2 472 cases)	39.9 %	(203 cases)
Primary substances	38.6 %	(1 766 cases)	16.9 %	(86 cases)
Pesticides	2.2 %	(100 cases)	5.1 %	(26 cases)
Foods and beverages	0.7 %	(31 cases)	18.5 %	(94 cases)
Medicinal products	1.6 %	(75 cases)	8.6 %	(44 cases)
Cosmetics	1.0 %	(48 cases)	3.3 %	(17 cases)
Others	2.9 %	(125 cases)	12.0 %	(61 cases)

Table 1: Spectrum of reports – synoptic view (repeat listing of cases possible)

2005 have been classified by product application groups (assignment of toxicants according to their intended use).

2.2.3 Causes of poisoning

The Berufsgenossenschaften almost exclusively reported cases of exposure to poisons in the context of occupational accidents (ca. 98 % of cases). The remaining 2 % of cases referred to accidents that had occurred during the common use of a product, or the cause of the accident was unknown.

Also among the reports submitted by hospitals and medical practitioners, accidents were the predominant cause of poisoning (46 %) followed by exposure during common use (25 %). Suicidal action was reported in no more than 2 % of cases. In the remaining cases, the cause was unknown.

On principle, acute poisoning takes a dominant position among the reports (Table 2). This is mainly due to the fact that the agreement on the transmission of reports on cases of poisoning by the Berufsgenossenschaften expressly provides for the submission of reports on acute cases of poisoning only.

2.2.4 Age structure and sex distribution

In 2005, the share of cases referring to adults among the total of cases reported was 96 %.

In the previous year, the reports submitted by the Berufsgenossenschaften also included four cases referring to children. These cases were attributed to accidents in schools or kindergartens. The share of cases in adults predominated also among the reports received from hospitals and medical practitioners. However, the share of children involved in these cases was as high as 33.6 % (Table 3).

	BG reports (100 % = 4 573 reports)		Non-BG reports (100 % = 509 reports)	
Acute	100 %	(4 572 cases)	78 %	(397 cases)
Chronic	0 %	(1 case)	12 %	(61 cases)
Unknown	0 %	(No case)	10 %	(51 case)

Table 2: Duration of exposure – synoptic view

	BG reports (100 % = 4 573 reports)		Non-BG reports (100 % = 509 reports)	
Children	0.1 %	(4 cases)	33.6 %	(171 case)
Adults	99.9 %	(4 569 cases)	61.9 %	(315 cases)
Unknown	0 %	(No case)	4.5 %	(23 cases)

Table 3: Age groups – synoptic view

	BG reports (100 % = 4 573 reports)		Non-BG reports (100 % = 509 reports)	
Male	63.0 %	(2 883 cases)	40.2 %	(206 cases)
Female	24.3 %	(1 113 cases)	38.9 %	(197 cases)
Unknown	12.6 %	(577 cases)	20.9 %	(106 cases)

Table 4: Sex distribution – synoptic view

2.2.5 Degree of severity of health impairment

Also in 2005, the majority of cases reported referred to minor health impairment only, both among the cases reported by the Berufsgenossenschaften and among those reported by hospitals and medical practitioners. Moderate and severe health impairment is more often reported by medical practitioners or physicians working in hospitals because they also receive reports on suicide attempts (Table 5).

The product groups involved most frequently with regard to the degree of severity of health effects have been listed in Table 6 for the cases reported by the Berufsgenossenschaften and in Table 7, for the cases reported by hospitals and medical practitioners. Of course, the toxicants reported from occupational environments were different from those reported from the private sphere because the availability of certain product groups differs, for example. One of the reasons for the high number of cases involving lamp oil may be seen in the specific ascertainment of such cases in collaboration with ESPED (Erhebungseinheit für seltene pädiatrische Erkrankungen in Deutschland – clinical registration unit for rare paediatric illnesses in Germany), an organization cooperating with almost all paediatric hospitals in Germany.

	BG reports (100 % = 4 573 reports)		Non-BG reports (100 % = 509 reports)	
None	1.0 %	(46 cases)	8.6 %	(44 cases)
Minor	94.3 %	(4 314 cases)	64.8 %	(330 cases)
Moderate	3.9 %	(178 cases)	20.6 %	(105 cases)
Severe	0.2 %	(10 cases)	3.1 %	(16 cases)
Cannot be assessed	0.5 %	(25 cases)	2.8 %	(14 cases)

Table 5: Degree of severity of health impairment – synoptic view

	Health impairment		
Product group	Minor (4 314 cases)	Moderate (178 cases)	Severe (10 cases)
Primary substances	1 641	92	8
Cleansing products	751	30	
Industrial cleansers	70	3	
All-purpose cleansers	52	1	
Descaling products	29	3	
Disinfectants/sterilizers	337	8	
Paints and related materials	194	5	
Paint thinners		1	
Glossy paints	44	3	
Building materials	142	13	1
Glues		1	
Sewage		1	
Galvanic cells	103		

Table 6: Product groups involved most frequently, by degree of severity of health impairment (BG reports 100 % = 4 573 reports)

	Health impairment		
Product group	Minor (330 cases)	Moderate (105 cases)	Severe (16 cases)
Foods and beverages	86	3	3
Food supplements	25	2	
Lamp oil	18	19	
Primary substances	62	15	3
Cleansing products	24	6	4
Office materials, chemical	14	22	
Fire lighting products	14	7	
Fungi	6	7	4

Table 7: Product groups involved most frequently, by degree of severity of health impairment (Non-BG reports 100 % = 509 reports)

2.2.6 Outcome of cases

For the notifications submitted by the Berufsgenossenschaften, the outcome has remained unknown in ca. 85 % of cases. The reason for this is that in the majority of cases, the report submitted corresponds to that by the so-called Durchgangsarzt ("transition doctor" appointed by the Berufsgenossenschaft). The reporting form is completed after the patient's first presentation. Therefore, such report does not contain any information on the course of the patient's illness. In selected cases, enquiries were made to obtain information on the course of illness. In the majority of cases on which information was available, patients had recovered completely.

Of the notifications submitted by hospitals and medical practitioners, patients recovered completely in 275 cases (54 %). In 220 cases (43.2 %), the outcome was unknown; in twelve cases (2.4 %), late sequelae could not be excluded or partial recovery was reported.

Gratifyingly, no more than two deaths were reported in 2005. The first case was that of an accidental ingestion of a product containing surfactants by an elderly male who died after aspiration as a result of cardiovascular arrest. This case has been described in a case report (see Chapter 3.4.7). The problem of "Elderly persons: a risk group for poisoning" was already discussed in detail in the annual report on Cases of Poisoning Reported by Physicians in 2001.

The second case was that of a suicide involving sodium selenite. The patient died from multiple organ failure (see case report in Chapter 3.4.1.2).

2.3 The product information system, PRINS

The notifications by physicians in cases of poisoning legally required under the Chemicals Act (§16e para 2) are regularly evaluated to protect consumers from health risks posed by chemicals and chemical products. Since 1994, the reporting physicians, the responsible ministries and the scientific community have been informed by annual reports on analyses of these notifications and the corresponding results. In the context of these notifications, the term, poisoning, is used to designate any health impairment associated with chemicals, including for example also allergies.

Since 1998, manufacturers and distributors of chemical products such as household chemicals and DIY products, cosmetics, plant protection and pest control products and correspon-

ding products for commercial use have been informed about cases of health impairment associated with their products that have become known to the BfR through case reports.

2.3.1 Rapid communications

If reports on **severe** health risks (except those related to suicides) are received by the BfR, it will provide for immediate information of the manufacturer/distributor of the chemical product involved as well as the competent industrial association/federal trade association and the responsible ministries, i.e. the Federal Ministry of Food, Agriculture and Consumer Protection, the Federal Ministry for the Environment, Nature Conservation and Nuclear Safety, and the Federal Ministry of Health as well as the Federal Office for Consumer Protection and Food Safety.

Between 1 January 1998 and 31 December 2005, 24 rapid communications were prepared and communicated. A synoptic view of these is given in Table 8. For explanations with regard to

Year	Product	Toxicologically relevant substance	Person exposed	Outcome	Proposal by BfR (P) and results (R)
1998	Impregnating agent	Fluorinated hydrocarbons	Adult	Death	P: Warnings for asthmatics R: Accepted
1998	Disinfectants/ sterilizers	Quaternary ammonium compounds (surfactants)	Elderly adult	Death	P: Information, labelling Ñcorrosive“ R: Accepted
1999	Toilet drain cleanser	Sodium hydroxide	Adult	Chemical burns	None
1999	Solvent	Petrol	Adult	Lung oedema	None
1999	Depilatory cream	Thioglycolic acid	Adult	Scars	None
1999	Disinfectants/ sterilizers	Quaternary ammonium compounds (surfactants)	Elderly adult	Death	P: Information, labelling
1999	Industrial cleansers	Sodium hydroxide	Infant	Chemical burns	P: Information
1999	Medicinal product	Dimenhydrinate	Infant	Death	P: Warnings
2000	Cream bath product	Surfactants	Elderly adult	Death	P: Information
2000	Lamp oil	Paraffins	Infant	Severe pneumonia	P: Partial ban R: Accepted, paraffin substitute
2001	Disinfectants/ sterilizers	Alkylamine (surfactant)	Elderly adult	Death	P: Information
2001	Tea (biodrug)	Atropa belladonna	Adolescent	Respiratory insufficiency	P: Information
2002	Lavatory cleansers	Surfactant	Elderly adult	Chemical burns	P: Information
2002	Mild detergent	Surfactant	Elderly adult	Death	P: Information
2003	Cleansing products	Surfactant	Elderly adult	Respiratory insufficiency	P: Information

Year	Product	Toxicologically relevant substance	Person exposed	Outcome	Proposal by BfR (P) and results (R)
2003	Food supplement	Proteins	Adult	Severe allergy	P: Information
2003	Fumigant	Sulfuryl difluoride	Adult	Death	P: Information
2003	Drain cleanser	Potassium hydroxide solution	Child	Severe chemical burns	P: Information
2003	Disinfectants/ sterilizers	Peracetic acid	Adult	Respiratory insufficiency	P: Information
2004	Garden torch	Paraffins, colourless	Infant	Respiratory insufficiency, death	P: Information, additional EU ban on colourless / unscented paraffins R: Accepted
2004	Oil lamp	Paraffins, colourless	Infant	Respiratory insufficiency, death	P: Information, additional EU ban on colourless / unscented paraffins R: Accepted
2005	Detergents	Surfactant	Elderly adult	Death	None
2005	Dishwasher cleanser for industrial use	Potassium hydroxide	Elderly adult	Severe chemical burns	None
2005	Breadseed poppy	Morphine	Infant	Respiratory insufficiency	P: guideline values / maximum levels and control, measures to reduce opiate levels R: Accepted

Table 8: Rapid communications 1 January 1998 – 31 December 2005

individual cases until 2004, reference is made to the 2002, 2003 and 2004 annual reports.

In the reporting year of 2005, three rapid communications were distributed.

Two of these referred to poisoning accidents suffered by elderly persons one of which had a lethal outcome as a result of detergent aspiration. In the other case involved, severe manifestations of chemical burns had developed after ingestion of a corrosive dishwasher cleanser for industrial use.

The third rapid communication referred to the case of an infant who had developed typical manifestations of opiate poisoning including somnolence and respiratory insufficiency after administration of a “calming beverage” containing poppy seeds for baking.

2.3.2 Summary reports

Reports referring to **non-severe** health impairment caused by chemical products in occupational or private environments are transmitted to the manufacturers/distributors in the first quarter of each new year in a summarized form. As recommended by manufacturers, also suicides and attempted suicides have been included in the summary reports since 2003 irrespective of the degree of severity of poisoning. Thus, manufacturers and distributors gain knowledge on

possible risks involved in the handling of their products. If they find the information provided to be insufficient, additional information can be obtained from the BfR. Much use has been made of this opportunity.

The information provided for the manufacturer/distributor is a contribution to increase product safety and thus, to improve consumer protection. There has been great interest in such information. For example, accident analyses have revealed that eye injuries caused by chemical products have frequently occurred in spite of wearing safety goggles. The safety data sheet should therefore draw attention to wearing "closely fitting safety goggles".

Summary reports for the year 2005 referred to 319 products and were transmitted to 142 manufacturers.

The table below (Table 9) provides a synoptic view of product application groups (minimum three listings) of the 2005 summary reports.

First level		Second level		Third level	
Agrochemicals	4				
Chemical products	274	Paints and related materials	5		
		Fire lighting products	8		
		Fuels, liquid	13	Lamp oil	12
		Office materials, chemical	11		
		Disinfectants/sterilizers	69		
		Glues	7		
		Cleansing products	116	All-purpose cleansers	5
				Oven cleansers	3
				Descaling products	9
				Dishwashing products, manual use	6
				Dishwashing products, machine use	3
				Industrial cleansers	28
				Rinsing additive for dishwashers	3
				Milking machine cleansers	10
				Furniture polishes	3
				Lavatory cleansers	10
Cosmetics/personal hygiene products	17	Hair care products	12	Depilatory products	12
		Skin care products	5	Creams / ointments	5
Pesticides	26	Herbicides	3		
		Insecticides	14	Phosphoric esters	8
				Pyrethroids	4

Table 9: Product groups frequently involved in 2005 summary reports

First level		Second level		Third level	
Agrochemicals	1	Fertilizers	1		
Chemical products	27	Fire lighting products	2		
		Fuels, liquid	6	Lamp oil	6
		Office materials, chemical	2		
		Disinfectants/sterilizers	2		
		Cleansing products	11	Oven cleansers	1
				Descaling products	1
				Glass cleansers	1
				Industrial cleansers	4
				Milking machine cleansers	2
				Carpet / cushion cleansers	1
		Textile, auxiliary products	1		
Cosmetics/personal hygiene products	2	Hair care products	1	Hair tonics	1
		Skin care products	1	Creams / ointments	1
Pesticides	2	Herbicides	1		
		Wood preservatives	1		
		Insecticides	1	Phosphoric esters	1

Table 10: Product groups associated with moderate health impairment listed in summary reports in 2005

Strikingly high numbers have been recorded for disinfectants (69) and cleansing products (116). Attention has to be drawn to the relatively high number of accidents involving milking machine cleansers (10).

Table 10 shows the numbers of moderate health disturbances associated with the respective product groups in 2005. 33 cases have been listed. It may be concluded that ca. 10 % of reports requiring summary reports to manufacturers referred to cases of severe health impairment. For five cases, the degree of severity of the health impairment could not be assessed despite further investigations. In four cases, no rapid communications were sent because either these referred to suicides or the degree of health damage had been primarily determined by an underlying disease instead of the product reported.

The BfR also performs cumulative data analyses of case reports. If trends become apparent, the manufacturers of the products concerned will be informed. In turn, manufacturers are requested by the BfR to communicate comparable data and trends that may serve to improve product safety.

3 Selected toxicological problems

3.1 Risk of aspiration carried by liquids for grill lighting and other petroleum distillates/ paraffins

As repeatedly reported in the past, liquid fuels containing paraffins and petroleum distillates that are used in ornamental oil lamps, garden torches, for grill lighting and fire-breathing carry a high risk potential. Due to their physicochemical properties, low-viscosity paraffinic hydrocarbons, if ingested, may enter the lungs (aspiration) and cause severe chemical pneumonia. Even very low quantities, often only a little sip, may result in severe health damage. In Germany, already five deaths in infants and young children have been recorded since 1990.

Most liquids for grill lighting or fire-breathing and also colourless lamp oils are prepared from petroleum distillates with straight-chain saturated aliphatic hydrocarbons (n-paraffins) having chain lengths between C_8 and C_{16}, and some of these contain low quantities (up to 10 %) of lower alcohols (e.g. isopropanol or isobutanol).

In Germany, the use of such petroleum distillates in coloured and/or scented lamp oils sold to the private end consumer has been banned since 1 January 1999 and in the EU, since 1 July 2000. However, the use of such low-viscosity paraffins is still permitted in colourless liquids such as those used in grill lighting aids, in colourless lamp oils etc. Again and again, severe health impairment has resulted from their ingestion. Observations made by different parties have indicated that the uncoloured and unscented products containing paraffins/petroleum distillates that are not subject to the ban are more aggressively advertised and sold, with increasing market shares, by wholesalers and retailers. A possible reason is that these products are a good fuel for garden torches and for the lighting of charcoal (grill lighting aids).

As a consequence of changes made to the formulations of coloured and/or scented lamp oils, e.g. the use of substitute substances, cases of severe health impairment or even with a lethal outcome have no longer been observed.

During the period between 1 January 1999 and 31 December 2005, the BfR received reports on 66 cases involving grill lighting aids and on 104 cases involving colourless lamp oils. Pneumonia was developed in 38 % of cases caused by grill lighting aids and in 45 % of cases caused by colourless lamp oils. Chemical pneumonia also resulted from the aspiration of pure kerosene and liquids for fire-breathing (see Table 11).

As in the past, the BfR continues to strongly advocate that the ban on the selling of coloured and/or scented lamp oils containing low-viscosity paraffinic hydrocarbons to the private end consumer be extended to other products such as grill lighting aids and colourless lamp oils.

	Total 1999–2005	Thereof pneumonia
Grill lighting aids	66	25 (38 %)
Colourless lamp oils	104	47 (45 %)
Kerosene	19	6 (32 %)
Liquid for fire-breathing	9	6 (67 %)

Table 11: Products containing low-viscosity paraffinic hydrocarbons vs. pneumonia cases

The Federal Ministry for the Environment, Nature Conservation and Nuclear Safety has again taken up this problem. Thus, Sigmar Gabriel, the Federal Minister for the Environment, has demanded more stringent regulations for the marketing of such chemicals on the EU level.

Fig. 5: Grill lighting aids

3.2 Health impairment due to poppy seeds
(See also risk assessment of 27 December 2005, www.bfr.bund.de)

Opium poppy (*Papaver somniferum*), also referred to as breadseed poppy, has been known as a medicinal plant since about 3000 BC. Today, its cultivation for opium and alkaloid production is no longer officially permitted in most countries except a few (e.g. India, Turkey, Greece).

Until recently, opium poppy was associated with narcotic drugs and morphine production for therapeutic purposes rather than with a possible health risk from the pure poppy seeds. In the last few years, however, it has repeatedly been pointed out that also the poppy seeds may involve health risks. The poppy seeds referred to are the ripe seeds of opium poppy (*Papaver somniferum* L.) Owing to their content of fatty oil (40–60 %) and protein (15–24 %), they are a popular source of food (for example in poppy-seed cake, desserts etc.). In poppy seeds, in contrast to other parts of the plant, the alkaloid-rich latex (milky juice) is not found at all or only in traces as a contaminant. Therefore, poppy seeds have not been included in the regulations under the Narcotics Act. The same plant variety is also used to obtain opium (dried milky juice from unripe pods) or opium alkaloids. In addition to the primary alkaloid, morphine, there are secondary alkaloids such as codeine, thebaine, noscapine (formerly referred to as narcotine) and papaverine.

The case of poisoning described in this Chapter was that of an infant who almost died due to poppy seeds intended for baking. Around the time when this incident happened, a television programme on edible poppy seeds was broadcasted in the Magazine, “Zeitspiegel” on Bavarian TV. The programme reported that considerable quantities of morphine had been detected in poppy seeds commercially available as a food and that such poppy seeds were being used for the preparation of narcotic drugs by drug addicts. It was stated that, from a scientific point of view, there was an urgent need to set maximum levels for the morphine levels in edible poppy seeds. As a consequence, the Federal Ministry for Consumer Protection, Food and Agriculture (BMVEL – now BMELV), in an effort to clarify the facts, commissioned the BfR to re-evaluate the risk potential involved. The problem to be considered included the quantities of morphine to be expected in edible poppy seeds and the health risks to be inferred from such quantities. Limitations of morphine levels in edible poppy seeds had been called for by the BfR already in the past.

Constituents of opium poppy
The percentage share of the alkaloids, morphine, codeine, narcotine, thebaine and papaverine found in the milky juice of opium poppy will vary depending on the species, soil conditions and climatic factors. Average levels detected in the dried milky juice (opium) are: 15 % morphine, 5 % narcotine, 1 % codeine, 1 % papaverine and 0.5 % thebaine. The oil-rich poppy

seeds are by nature almost free from alkaloids and contain only traces (0.005 %) of these substances. However, consumption of major amounts of bakery products containing poppy seeds may be sufficient for such traces to produce positive morphine findings in urine drug screening and thus, suggest a possible opiate abuse. For example, studies performed in the past have revealed that poppy seeds originating from different countries (i.e. the Netherlands, Australia, Hungary, Spain, the Czech Republic and Turkey) may vary considerably with regard to their opiate levels. Morphine levels varied between 2 and 251 µg/g, and codeine levels, between 0.4 and 57.1 µg/g, and no other opiates could be detected. It was assumed that 1–2 bread rolls contained 4 g of poppy seeds. In a study involving the administration of this quantity to test persons, positive urine findings of morphine were made after the consumption of poppy seeds from Spain and Australia. Poppy seeds from the Czech Republic, Turkey and the Netherlands resulted in negative urine findings.

Since the end of 2005, current analytical data on poppy seeds originating from the German market have been available to the BfR. Morphine levels detected in these seeds were between less than 10 µg/g and 50 µg/g or between more than 50 µg/g and less than 100 µg/g. However, in single cases, levels above 100 µg/g up to a maximum of 330 µg/g were detected. Evaluation has revealed that compared with data published in the past, morphine (and codeine) levels show a clearly increasing trend. Possible causes to be considered for the high opiate levels found in some types of poppy seed include the selection of less appropriate plant varieties, an unfavourable time of harvesting and a certain geographic origin. However, there is also good reason to assume that changes in harvesting techniques could play a role. For examples, newly introduced harvesting techniques lead to crushing or cutting of the seed pods. Thus, the poppy seeds may easily become contaminated with the milky juice from the poppy pods or with pod fragments during harvesting. As mentioned above, alkaloids are not contained naturally in the poppy seeds. However, the toxicologically active substances are contained in all other parts of the plant including the dried milky juice.

Risk potential

The risk potential carried by poppy seeds can be derived for example from the known pharmacological, pharmacokinetic and toxicological facts describing the dose-effect relationship for the morphine contained in these seeds. The most important manifestation observed in the clinical picture of poisoning is respiratory depression. It will develop in an insidious way by an increase in the stimulus threshold of the respiratory centre even in the absence of a feeling of dyspnoea, or sudden respiratory arrest will occur. Severe poisoning is typically characterized by the triad of respiratory depression, coma and miosis. With regard to the circulatory organs, manifestations will include episodes of bradycardia, arrhythmia and collapse. Less severe manifestations may include constipation, retention of urine, hypothermia, paleness or reddening of the skin (release of histamine). Morphine is mainly used for the treatment of severe and most severe pain. Also therapeutic morphine doses have to be expected to cause serious adverse effects in some cases, particularly an impairment of the mental and physical activity. Chronic administration of morphine will result in a development of tolerance and both mental and physical dependency. In animal experiments, morphine has shown effects indicating developmental and reproductive toxicity, and in a number of studies, also genotoxic effects.

So far, products containing poppy seeds have been consumed almost unscrupulously. On the basis of the recent analyses, however, a more serious warning has to be given that in unfavourable cases, production batches may be

Fig. 6: Poppy

contaminated and contain high morphine levels reaching or even exceeding the upper limit of the range for therapeutic single oral doses stated by the Federal Institute for Drugs and Medical Devices (BfArM). The common effective single dose recommended for adults is 7.6 to max. 45.6 mg of morphine, while the lowest effective single dose stated is 1.9 mg. On principle, considerable variation in individual sensitivity has to be assumed with regard to the therapeutically desirable and the adverse effects of morphine.

The BfR has now derived a "provisional maximum intake" for morphine. It is 6.3 µg morphine/kg body weight/day and determines the morphine intake which an individual (as related to body weight) should not exceed per day when consuming foods containing poppy seeds, whether in a single meal or distributed over the entire day. The calculation was based on the lowest effective dose stated for oral morphine medication, i.e. 1.9 mg (corresponding to 31.7 µg/kg body weight for an individual of 60 kg body weight). Even at such low doses, however, pharmacological effects due to other factors such as individual sensitivity cannot be excluded. Therefore, the guideline value determined for daily morphine intake is lower by a factor of 5 than the lowest effective dose stated for morphine.

Based on the provisional maximum daily intake and taking into account an estimate of the amounts consumed, a provisional guideline value for morphine levels in poppy seeds has been recommended. This value is 4 µg morphine/g poppy seeds.

Conclusions

The figures stated are only guideline values. The manufacturers involved are requested to make every effort to reduce the concentrations of all pharmacologically active opium alkaloids in poppy seeds to the lowest level technologically achievable. The BfR recommends to establish on this basis guideline values also for the other alkaloids found in poppy seeds such as codeine, noscapine, papaverine and thebaine. Until a change in the processing conditions of poppy seeds has been achieved, the BfR recommends to refrain from excessive consumption of foods containing high quantities of poppy seeds, particularly during pregnancy.

Case report

Severe health impairment in the case of a 6-week-old infant temporally related to the ingestion of boiled poppy seeds (as suggested by a baking book recipe)

The Poison Control Centre in the city of Erfurt communicated to the BfR information followed by a medical report and the results of toxicological examination referring to a case of severe health impairment in a 6-week-old female infant who had suffered from respiratory depression culminating in respiratory arrest. The infant had been given the strained milk of poppy seeds by her mother, who had intended to help her sleep through the night, following advice given in a baking book. In that baking book entitled "Backen ohne Schnickschnack" (No-frills baking), the following recommendation is given: "Grandmother told us that in Silesia, people used to give their babies the strained milk from poppy seeds. This made them sleep through the night."

The infant had been given 75 mL of strained milk from a mixture of 200 g poppy seeds boiled in 500 mL milk and with some honey added. According to the information given by the Poison Control Centre, the mother had fortunately used only half the quantity of poppy seeds recommended (amount stated in the recipe: 400 g poppy seeds).

Toxicological examinations revealed that the morphine level in the serum of the infant was still as high as 4.3 µg/L on the following day. The poppy seeds used contained 0.1 % morphine and 0.003 % codeine. The infant had to be respirated and administered an antidote. After 10 days, she could be discharged from hospital in a healthy condition.

Manifestations / course
After the mother had given her baby the infusion prepared from poppy seeds at about 23 h with the intention to have her sleep through the night, the little girl cried for a short period at about 2 h and subsequently fell asleep again. Shortly afterwards, her breath became irregular with a rasping sound. She was brought to hospital by an emergency ambulance.

On admission, the infant's general condition was critical. Her consciousness was clouded and she hardly responded to pain stimulus. Findings included an inspiratory stridor sound, her contracted pupils did not react to light, and her skin was of a pale, grey, cyanotic and marbled appearance. Increasing respiratory insufficiency was observed associated with a repeated dropping of oxygen saturation to a level of 67 % in the absence of oxygen supply. Due to imminent respiratory arrest, artificial respiration was performed using an oxygen mask. Aspiration could be excluded. Since opiate poisoning was suspected, an i.v. antidote treatment with naloxone was performed by administration of a total of six single doses, which resulted in a persistent effect, i.e. sufficient spontaneous respiration.

The suspected poisoning was confirmed on the same day by urine analysis revealing a morphine level of 18.2 µg/L and a codeine level of 317 µg/L. On the following day, the morphine level in the serum was 4.3 µg/L. The level of morphine in the urine had dropped to 627 µg/L and that of codeine, to less than 5 µg/L. In the further course, increased temperature and diarrhoea were observed. After 10 days, the child could be discharged in a good general condition and referred to outpatient care. The poppy seeds used were also subjected to analysis. The level of morphine detected was 0.1 %, and that of codeine, 0.003 %.

Notes
In the past, there had been frequent reports of cases of poisoning in infants after administration of "calming tea". As a rule, such tea contained poppy shells prepared from unripe or ripe poppy pods whose morphine levels were considered as harmless. As a consequence, trade in non-detoxified parts of the opium poppy plant has been banned. Ornamental poppy pods may be used by florists after morphine extraction.

For the treatment of the slowly increasing respiratory insufficiency, administration of naloxone (5–10 µg/kg b.w.) is indicated to neutralize the depressing effect of morphine derivatives on respiration. Because of the short half-life of 1–1.5 hours, administration of repeat doses (continuous infusion, if appropriate) may become necessary.

Evaluation of the case described
In the case described above, a causal relationship between the ingestion and the manifesta-

tions observed has been rated as confirmed on the basis of the information given as to the temporal relationship and in the absence of other causes for the manifestations developed.

Consequences of this case
Given the fact that in the above case, the morphine level detected in the poppy seeds was as high as 0.1 %, the superior government authorities of the German Länder were requested to consider appropriate monitoring measures. A rapid communication was circulated among the responsible ministries.

3.3 Chemical burns

3.3.1 General information
Chemical burns are caused by exposure to acids, alkalies or other chemicals having corrosive or colliquative effects. Body areas particularly at risk include the mucosae of the upper gastrointestinal tract, mainly the oesophagus, after oral ingestion of a corrosive substance.

Most corrosive poisons act very quickly. Therefore, chemical burning processes will become terminated within about 60–90 seconds. This is why rapid action is required. Based on the clinical course, there are essentially two types of chemical burns.

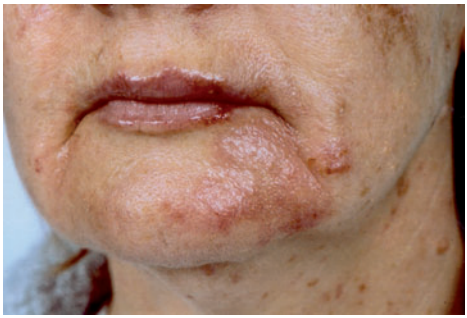


Fig. 7: Chemical burns caused by drain cleaner

Types of chemical burns
Coagulation necrosis
Acids will cause immediate precipitation of proteins resulting in coagulation necrosis, which will prevent further deep action and thus protect the underlying tissue. Sulfuric acid causes dehydration and strong heating of the tissue and thus may cause damage to all layers of the skin, mucous membranes and the eyes. Similarly, deep action is produced by hydrofluoric and nitric acids. Therefore, the extent of the damage can be assessed only after a certain latency period.

Colliquative necrosis
Exposure to alkalies will result in colliquative necrosis. Due to cell lysis, the chemical will rapidly penetrate the outer layers of the skin. Among alkaline burns those caused by lime are prominent. Exposure to unslaked lime (calcium oxide, also referred to as quicklime or burnt lime) will result in the formation of calcium hydroxide on the mucous membranes and in the eyes (lacrimal fluid). This is accompanied by heat formation, which may cause additional damage to the skin, the mucosa and the eyes.

Low concentrations of corrosive substances
Often, no clear differentiation of clinical manifestations is possible after exposure to low concentrations of corrosive substances. Both acid and alkaline substances will cause no more than various degrees of irritation of the skin or mucosa because the underlying tissue is affected to a minor degree only.

*Chemical burns of the **oesophagus** (chemical burns of the **skin**): Degrees of severity*
Oesophageal burns are classified by three degrees of severity.

First-degree burns are characterized by superficial lesions of the mucosa with erythema and oedema and without epithelial damage. In most cases they will heal without sequelae.

Second-degree burns are associated with minor erosions, ulcerations and fibrin exudation. They reach the submucosa and are associated with a loss of substance. Their healing process will include reactive granulation followed by scar formation.

Third-degree burns are characterized by deep ulceration reaching into the muscularis mucosae and being associated with bacterial penetration into the perioesophageal tissue and perforation followed by mediastinitis. This will result in the formation of stenosing scars impairing food intake. Scarred strictures on the skin may restrict movements so that patients need to undergo surgical correction in the further course.

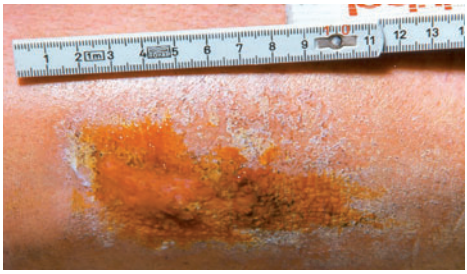


Fig. 8: Chemical burns caused by depilatory product

*Chemical burns of the **eye**: Degrees of severity*
Mild burns are characterized by keratolysis of the outer layer of the cornea. The edge of the cornea remains intact. From there, the cornea can regenerate. No corneal opacification will follow.

Moderate burns are characterized by damage both to the outer layer of the cornea and to the conjunctiva. Reddening is a favourable sign indicating that blood flow has remained intact.

Most severe chemical eye burns are characterized by a dense white opacification of the eyeball. In such cases, also deeper parts of the eye such as the iris, lens, vessels and sclera will be affected by irreparable damage. The eye affected will go blind.

3.3.2 Substances causing chemical burns

Typical substances causing chemical burns include acids, alkalies and other chemicals such as phenols, cresols, potassium permanganate, organotin compounds, bisulfides, hypochlorites, concentrated extracts of vegetal tanning agents (oak, camomile) and many others.

In cases of poisoning, it is impossible to establish any direct relationship between either the pH value of the aqueous solution or the acid dissociation constant (pKa value) and the clinical manifestations (degree of severity of the chemical burn). The corrosive potential of a substance will depend not only on the pH value but also on the concentration, on other substances ingested simultaneously, on the duration of exposure and the quantity ingested. Documented cases of poisoning have shown that under very unfavourable conditions, substances may cause changes of the skin in the sense of chemical or thermal burns even if this is none of their known primary chemical properties (e.g. kerosene or tributyl tin compounds if soaked clothes are not changed for an extended period).

In contrast to the industrial environment (workplace), the number of corrosive products used by the private consumer is relatively low. These include primarily lavatory and drain cleansers as well as cleansers and descaling products for dishwashers and coffee machines. In general, a tendency has been observed that the number of corrosive products being marketed is decreasing.

3.3.3 Routes of exposure and therapy

Immediate measures on the scene of the accident
Most corrosive poisons act very quickly. Therefore, immediate action is of essential importance. Less rapid action is developed by dry substances in granular form because these can act only after having become dissolved. After **oral** ingestion, the first measure to be taken by the person affected is to spit out the corrosive

substance. Particularly in the case of children and after ingestion of solid corrosive substances such as granular dishwasher cleansers, the patient's mouth should be wiped out with a damp cloth as soon as possible. To make use of the dilution effect, the patient should be prompted to immediately drink water or another (non-alcoholic, non-carbonated) fluid quickly available such as tea or juice. If some of the granules are stuck e.g. in one of the oesophageal sphincters, drinking of large amounts of fluid may also be effective some minutes later: the oesophagus becomes flushed.

Any administration of milk in cases of poisoning is of no practical benefit; rather, it is contraindicated. Since poisons are either fat-soluble or water-soluble, milk will constitute an ideal transport medium, and the toxicants will be absorbed with particular readiness. In children, milk will often have an additional emetic effect so that the corrosive substance passes the oesophagus twice, thus causing double damage. Therefore, vomiting must NEVER be induced. Also for a so-called neutralization, milk is of minor importance because its buffering capacity is minimal. Drinking of milk is only recommended in exceptional cases such as after accidental ingestion of fluorides, tetracyclines or iron since the poison types formed as a result are less readily absorbed. Also gastric lavage is not recommended. However, it may be indicated to remove the corrosive substance ingested through a feeding tube if in addition, there is a threatened risk of poisoning by absorption (e.g. hydrofluoric acid, formic acid).

On exposure of the **skin**, clothes soaked with the substance should be removed as quickly as possible. The skin areas affected by wetting should be rinsed immediately with plenty of water, ideally under running water or a shower. However, it should be avoided that patients become hypothermic.

If exposure to a corrosive substance has affected the **eyes**, intensive rinsing with water (for at least 10 minutes) should be performed. Aid measures will become more effective if a second helper is present. Effective rinsing requires ectropionizing of the eyelids, which is often difficult. Slipping of the fingers can be prevented by using a dry cloth or swabs. If this does not succeed, an analgetic effect and thus opening of the eyelid can be achieved by administering 2 % lidocain (contained in emergency medical kits) onto the edge of the lid. Ideally, the eye affected should be rinsed under running water with the eyelids open, which, however is impossible in most cases for technical reasons. As an alternative, a cloth soaked with water can be pressed out above the eye several times. After exposure of the eye to lime or cement, deeper penetration may be prevented by additional cautious mechanical cleaning of the conjunctiva, e.g. using the corner of a handkerchief. After first-aid measures in cases of chemical eye burns, an ophthalmologist should always be consulted for specialist examination and therapy.

Inhalation of a corrosive substance may on principle result in irritation or chemical burns of the upper airways. Some corrosive poisons also act as an irritant gas. Depending on water or lipid solubility of the substance, toxic lung oedema will develop with (e.g. nitrous gases from nitric acid) or without a latency period. Therefore, one package of a CFC-free corticoid spray should be administered prophylactically which is inhaled by the patient until it has become empty. If manifestations persist or a severe poisoning is assumed, the therapy should be continued and must never be terminated too early, particularly if irritant gases inducing symptoms with a latency period are involved. If first signs of lung oedema are observed, local glucocorticoid administration will no longer be sufficient and therefore, has to be replaced by systemic administration. All such cases require observation in a hospital in the further course. Subsequent therapy will depend on the manifestations observed.

*Subsequent procedure/**scheme for treatment of chemical burns** (after **accidental** ingestion)*
All cases of confirmed ingestion of a corrosive substance will require treatment. In cases of suspected ingestion, a chemical burn is suggested by at least one of the following manifestations observed within the first 24 hours after the incident: Signs of burn in the oral cavity or pharyngeal region, hypersalivation, retching, vomiting, retrosternal or epigastric pain and refusal to eat (in children, an indirect manifestation of pain) It has to be taken into account that although sings of burn may be absent in the mouth after ingestion of a corrosive fluid due to the rapid passage and short duration of exposure, considerable chemical burns may be present in the oesophagus. Patients who are initially symptom-free after suspected ingestion do not undergo treatment. However, they should be seen by a physician for control and observation. Immediate measures to be taken still on the scene of the accident may include pain relief, circulatory assistance, and intubation or tracheotomy.

Systemic administration of 3 mg/kg b.w. prednisolone is performed to prevent glottic oedema. Oesophagoscopy should be performed later when this can be done on a routine basis (as a rule, on the following morning) and should be carried out by an experienced team. This will ensure that the extent of damage suffered is correctly identified. Inspection is performed until the first sign of burn becomes visible. The stomach has to be inspected only after ingestion of strong acids, soldering fluid or potassium permanganate crystals in order to exclude lesions of the stomach wall or to remove remaining crystals to prevent them from penetrating into deeper regions. If findings are *negative*, the patient can be discharged without any further measures. Also *first-degree* chemical burns do not require any treatment or follow-up examination. In cases of *second-degree* chemical burns, administration of prednisolone at a dosage of 1 mg/kg b.w. is recommended until healing has been verified by endoscopy. In animal studies, prednisolone has reduced abnormal growth of fibroblasts. Therefore, it was assumed to be able to prevent the formation of oesophageal strictures in humans. However, this assumption has not been confirmed in follow-up examinations of patients after chemical burns. In cases of *third-degree* burns, steroid therapy is discontinued. Such lesions may require surgical intervention. In cases of severe chemical burns where a high risk of stricture formation is involved or oesophageal stenosis has developed after healing, treatment may consist in (early) bougienage, insertion of a permanent gastric tube, or percutaneous endoscopic gastrostomy (PEG).

Onward therapy will depend on the manifestations observed. It may include administration of antibiotics to control mediastinitis and secondary infections associated with gastric ulcers, of proton pump inhibitors to reduce the production of gastric acid, and dietetic measures to ensure food intake (liquid or mushy diet or parenteral nutrition depending on findings). After ingestion of large quantities of harmful substances, possible effects from absorption such as acidosis, haemolysis or renal failure have to be taken into account. Finally, follow-up endoscopy is required, with the appropriate time of such examination depending on the extent and severity of the initial findings. Contrast X-ray examinations should be performed after three to four weeks, in cases where apparent stenosis in the oesophagus or in the pyloric area has to be confirmed or excluded.

Suicidal chemical burns require much more active and aggressive treatment. In addition to severe local damage, also the systemic effect resulting from absorption has to be taken into account in such cases. For example, acids may cause acidosis, haemolysis and subsequent renal failure, hydrofluoric acid (see below) may cause hypocalcaemia followed by life-threatening cardiac arrhythmia. Large quantities ingest-

ed cannot be sufficiently neutralized by dilution so that continued coagulation and colliquation have to be expected resulting in severe damage to the stomach and the duodenum. Consequences may include perforation or later cicatricial deformation with associated stenosis formation. Septicaemia may develop even several days or weeks later.

Based on these considerations, gastric evacuation by means of a carefully introduced soft feeding tube, emergency endoscopy and early surgical intervention may be indicated in certain situations. Since almost all of these cases represent third-degree chemical burns, no prednisolone is administered on principle because of the high risk of perforation. In most of these cases, there is a poor prognosis. If the patient survives, severe permanent damage has to be expected.

3.3.4 A special case: Hydrofluoric acid burns

Due to its low dissociation and high lipid solubility, hydrofluoric acid has a strong ability to deeply penetrate into the tissue and is quickly and readily absorbed. This will result not only in severe local damage with the corrosive action continuing for hours or days and reaching deep layers of the tissue and bones, but also in manifestations due to absorption. These may occur after oral as well as after inhalational or dermal exposure. On ingestion, doses exceeding 20 mg/kg b.w. are considered as potentially lethal, i.e. one sip of a 2 % solution may be sufficient to cause death.

Due to the binding of magnesium, poisoning with hydrofluoric acid causes hypomagnesemia. Also hyperkalaemia may occur, and in particular the formation of calcium fluoride may result in life-threatening cardiac arrhythmia due to hypocalcaemia. Manifestations may include a drop in blood pressure, shock, clouded consciousness, convulsive seizures, metabolic acidosis, weakness of muscles, rhabdomyolysis, hepatic and pancreatic damage, osteolysis, and many others.

On inhalation, hydrofluoric acid acts as an irritant gas of moderate water solubility. The severity of manifestations will depend on the concentration and duration of exposure. Already concentrations as low as 5 ppm have been observed to cause irritation of the mucous membranes of the eyes and nose. Higher concentrations will cause dry cough, dyspnoea, bronchospasm, tracheobronchitis, pneumonitis, glottic oedema and chemical burns of the respiratory tract. After a latency period of up to ten hours, lung oedema, tracheobronchial haemorrhage and atelectasis may develop.

On the skin, painful inflammations and chemical burns will develop already at low concentrations of 0.1–0.3 %. However, also at higher concentrations of up to 20 %, pain and erythema may still develop after 24 hours or even later. Exposure to concentrations above 20–50 % will result in recognizable burns mostly within one to eight hours while concentrations above 50 % will lead to a rapid onset of pain associated with visible cell destruction. Manifestations may persist for several days. In the further course, erythema, blistering, oedema, whitish to grey-black discoloration of the skin, slow-healing ulcers and progressive colliquative necrosis in deep tissues, possibly associated with osteolysis, may develop.

In the eyes, chemical burns of any degree and colliquative necrosis may develop even several days later.

Poisoning involving hydrofluoric acid must always be taken seriously. Medical observation by a physician is required in any case. Special therapeutic measures have to be taken.

The most important measure consists in early local and systemic administration of calcium in the form of calcium gluconate. This will markedly reduce the risk of local damage and cardiac arrhythmia and thus also the risk of severe or lethal intoxication. Therefore, in the exceptional case of hydrofluoric acid *ingestion*, drinking of milk or, what is to be preferred, of calcium in the form of dissolved effervescent tablets on the scene of the accident is recommended. To control the existing high absorptive toxicity, vomiting should also be induced despite the local corrosive action. In the early stage of poisoning, the risk of perforation is to be considered as low as compared to that of a development of manifestations from absorption.

In the hospital, gastric lavage is administered, using a 1 % calcium gluconate solution which should take place during the first 90 minutes after ingestion. The scheme for treatment of chemical burns (see above) is applied and systemic therapy performed. This consists in. i.v. administration of calcium gluconate with simultaneous monitoring of the calcium serum level. After ingestion or inhalation of toxically relevant quantities or pronounced dermal contact, such measures should ideally start already in the ambulance because rapid initiation of an adequate therapy is decisive.

After *inhalation* of hydrofluoric acid, a CFC-free corticoid spray should be administered as a precaution. If pulmonary manifestations are observed, systemic administration of corticoids is required. In addition, calcium gluconate solution should be administered using a nebulizer.

After *dermal* exposure, the damage and risk of a systemic reaction must not be underestimated even in cases were no initial manifestations are observed. First measures include immediate flushing with plenty of water for 15–20 minutes or with 1 % calcium gluconate solution, if available. Single studies have also reported on the successful administration of polyethylene glycol (Lutrol, PEG 400). In any case, care should be taken that flushing is performed in a thorough and meticulous way and that the surrounding tissue is not contaminated with the fluid used for flushing. Soaked clothes must be removed. After exposure to low concentrations or a short duration of exposure and in the absence of pain, subsequent application of 2.5 % calcium gluconate gel will suffice. Higher concentrations or pain (cardinal sign) require s.c. injection of 10 % calcium gluconate solution into the skin area affected. In most cases, this will result in immediate pain relief and has to be repeated some hours later, if pain sets on again. In the acral regions, s.c. injection is difficult for anatomical reasons. If such areas are affected, intraarterial administration of calcium gluconate should be preferred. If required, a local anaesthetic has to be administered in addition and necrectomy performed.

After exposure of the *eye*, administration of 1 % calcium gluconate eye drops is recommended after immediate and sufficient flushing with water, physiological saline, or ideally, with 1 % calcium gluconate solution. In addition, a local anaesthetic may be required for pain control, and dexpanthenol can be administered to promote wound healing. Ophthalmologic follow-up examinations over several days are recommended.

In all cases of poisoning involving hydrofluoric acid, inpatient observation should be provided. If required, a Poison Control Centre can be consulted for advice and recommendations for further treatment.

Case report
Chemical burns from sodium hydroxide solution with a severe course

During the dyeing of zippers in a textile finishing company, both forearms of a worker were hit by the bath consisting of sodium hydroxide solution and other chemicals when he manipulated a pressurized container. Initially, the injured worker had intended to hide the accident from his employer. Therefore, it took four months until he was referred to inpatient treatment with extensive and severe chemical burns from sodium hydroxide solution.

Manifestations / course

Without consulting a doctor or informing his employer, the accident victim had treated his extended chemical burns in a "self-directed" way. Four months later, the injuries were seen by the family doctor, who referred the patient to a hospital specialized in accident surgery. Findings made there included extensive ulcers on both forearms (12 cm x 6 cm and 15 cm x 8 cm) associated with extensive soft-tissue defects in the extensor muscles. The wounds showed a greasy covering and in part, already granulation. In the depth of each wound, the radius lay bare in a small area.

Functional testing revealed a complete failure of the finger extensors and of the extensor pollicis longus tendon due to a complete loss of extensor digitorum muscles on both forearms. The radial and ulnar wrist extensors had remained intact so that moving the wrists was still possible. Flexor structures had also remained intact so that a complete closing of the fist was possible. Rotation movements of pronation and supination were restricted (30–40°) on both forearms. Finger joints were passively flexible. No sensory loss was found.

X-ray follow-up examination of both forearms revealed an extensive periostal reaction on both ulnas and radiuses associated with shell-shaped calciferation around the diaphysis. In addition, findings included ossification in the interosseous membrane and fibrous structures towards the wound ground. There was no obvious osteolysis.

Unfortunately, a therapy could not be initiated until four months after the accident because the patient had failed to see a doctor before that time. Subsequently, inpatient treatment was initiated immediately. The first measure to be performed was a comprehensive debridement followed by conditioning of the wound ground by means of a vacuum suction therapy. This was to be followed by split-skin grafting. Motor substitution surgery was considered to improve extension of the fingers. The result remains to be seen.

Notes

In the above case report, the severe sequelae of alkaline burns treated too late due to the patient's own fault are described in an impressive way. On the skin and mucous membranes, alkalies such as sodium hydroxide or potassium hydroxide solution and ammonia will cause local soapy colliquative softening and necrosis of the tissue. Due to ability of liquefying and dissolving protein, bases are characterized by a deeper action than acids. Rapid dilution of the toxic agent with water is the most important immediate measure to be performed. Subsequent treatment will depend on the localization and severity of the chemical burn suffered.

Evaluation of the case described

Based on the information given on the temporal relationship between dermal exposure and the appearance of manifestations, and in the absence of other causes, a causal relationship has been rated as probable in the case described.

Chemical burns from ammonia ingestion
A 45-year-old male had ingested about 10–20 mL of liquid ammonia from a soda water bottle and experienced symptoms immediately afterwards. Two hours later he sought medical advice in the emergency department of a hospital. He was given inpatient treatment for two days.

Manifestations / course
Immediately after ingestion, the patient had experienced a burning sensation in his throat and shortly afterwards, retrosternal pain. Findings made during medical examination two hours later included severe reddening of the oral mucosa. The tongue exhibited a whitish coating. Laryngoscopy revealed a severe reddening of the larynx, however, neither haemorrhage nor oedema was found. Oesophagogastroduodenoscopy revealed superficial lesions of the oesophageal mucosa without deep ulceration. The same applied to the region of the cardia, with haematin found in the fundus. The mucuous membranes of the corpus and antrum showed a mild gastritis without any lesions. Likewise, only a mild bulbitis was found to be present in the pylorus.

Treatment included the administration of proton pump inhibitors, piritramide and metoclopramide as an analgesics, and cephalosporine and metronidazole as an antibiotic therapy. Already on the following day the patient was symptom-free. He could be discharged and referred to outpatient care after two days.

Notes
Ammonia is readily soluble in water. Its 10 % solution is known as spirits of ammonia or liquid ammonia. Chemical burns of the oral cavity, oesophagus and stomach are to be expected after exposure to concentrations above 10 %.
As in the above case, accidental ingestion of liquid ammonia will most probably result in a mild first-degree chemical burn. Oesophago-Gastro-Duodenoscopy would not have been imperative, nevertheless, it provided a confirmation of the above assumption. No special therapy is required, measures will depend on the manifestations observed.

Evaluation of the case described
Based on the information given on the temporal relationship between ingestion and the occurrence of manifestations fortunately associated with a minor health impairment only, and in the absence of other causes, a causal relationship has been rated as probable in the case described.

3.4 Case reports

3.4.1 Poisoning associated with food supplements

3.4.1.1 Noni juice

Acute liver cell damage after consumption of noni juice

A 43-year-old female had consumed a daily quantity of 30 mL noni juice over a one-year period, in the belief to enhance her resistance to infections in this way. When she developed states of general indisposition, she discontinued the consumption of noni. However, in spite of the discontinuation, her general condition deteriorated, and she was finally admitted to hospital in July 2005.

A possible causal relationship between the consumption of noni juice and confirmed acute liver damage is being investigated.

Manifestations / course

Because of her general state of indisposition and gastrointestinal complaints, the patient discontinued the consumption of noni juice. Four weeks later, she initially consulted a non-medical (alternative) practitioner. However, her condition deteriorated considerably – she lost weight and developed jaudice – so that admission to hospital was required. In the hospital, acute hepatic damage was diagnosed (GOT: 70 U/L; GPT: 22 U/L; total bilirubin: 324 µmol/L; ammonia: 45 µmol/L). Findings also included signs of haemolytic anaemia. In the further course, the patient's hepatic parameters spontaneously improved, however, without reaching completely normal levels on outpatient follow-up examinations performed later. The most recent information was received by the BfR in December 2005. At that time, the patient was still under outpatient treatment. Examinations focussed on the tentative diagnosis of sarcoidosis. The BfR has not received any detailed information on diagnosis and therapy.

In the case of this patient, the attending physicians obviously disagreed on whether her acute liver cell damage had been caused by the consumption of noni juice. Rather, the simultaneous signs of haemolytic anaemia had suggested an antibody process, which resulted in the diagnosis of liver damage of unclear origin. Other physicians did not exclude an association with the consumption of noni juice.

Notes

Noni is the fruit of the tree, *Morinda citrifolia*. It grows on the islands of French Polynesia. Noni juice has been ascribed numerous healing effects by Polynesian natives. In these cultures, consumption of the juice is widespread.

For about ten years now, noni juice has been extensively marketed in the USA. In recent years, sales have increased also in Europe. Presently, noni juices are being sold as an "insider tip" on the market of food supplements. They are available at high prices (35–40 Euro per L) virtually only via the internet or by direct marketing. Noni juice is advertised to have positive effects on a number of different health disorders including serious illnesses such as cancer.

Noni juice contains essential amino acids and enzymes. Its favourable biological effects are said to be based predominantly on serotonin, terpenes and flavonoids. However, no scientific studies have been available so far that would demonstrate direct favourable effects of the bioactive substances contained in noni juice. Also the particularly important biological role of xeronine or proxeronine has been frequently mentioned but not scientifically substantiated so far.

The EU Scientific Committee on Food (SCF) has authorized the placing on the market of noni juice as a novel food ingredient in June 2003 commenting that this was a fruit juice as any other. The SCF stated that no evidence was available to substantiate special health benefits. However, according to the information available, e.g. on the internet, it is expressly the positive effect on health that has been advertised. The considerable difference between noni juice and other fruit juices consists in the retail price of ca. 40 Euro per L.

Other cases

So far, the BfR has been informed about two other cases associated with noni juice: In 2000, skin changes were reported which were assumed to be associated with the consumption of noni juice. A second case of health impairment possibly caused by noni juice was reported in 2004. It referred to acute hepatitis which developed after regular consumption of noni juice over a period of several weeks. Other causes of hepatitis could be excluded. The degree of severity of this health impairment was classified as moderate. Regrettably, further investigations performed with regard to a possible causal relationship have been unsuccessful.

Recently, three cases of acute hepatitis were reported from Austria, which were supposed to have been causally related to the consumption of noni juice. A 62-year-old female and a 45-year-old male had consumed noni juice over a period of several weeks for prophylactic reasons. Both were admitted to hospital with signs of acute hepatitis. After discontinuation of noni juice consumption, their hepatic parameters spontaneously returned to normal levels. Since other causes could be excluded, a possible causal relationship with noni juice was assumed to exist in these cases. A 29-year-old male suffered severe life-threatening liver damage after the consumption of noni juice. He could be rescued by liver transplantation.

Fig. 9: Indian mulberry

Evaluation of the case described

Based on the information given on the temporal relationship between the consumption of noni juice and the development of manifestations including confirmed hepatitis, a causal relationship cannot be excluded. As other cases have shown, this is not a singular case.

Given the authorization of noni juice as a food ingredient, it could be assumed so far that no health damage was to be expected to result from its consumption. When considering the cases reported in Germany together with those of acute liver damage in Austria that showed a temporal relationship with the regular consumption of noni juice over periods of several weeks, a possible causal relationship may at least be suspected.

In a summarizing view, it has to be stated that there have been repeated indications of a possible serious health risk involved in the consumption of noni juice calling for a reassessment of noni products.

3.4.1.2 Sodium selenite

Death resulting from suicidal ingestion of sodium selenite

A 57-year-old male suffering from chronic alcoholism had admitted to his wife to have in-

gested selenite. Therefore, she called an emergency physician. The patient stated to have ingested 1–2 g of sodium selenite in a suicidal attempt. He was immediately admitted to the intensive care unit of a hospital where his condition deteriorated rapidly. In spite of intensive therapy the patient died on the next morning from multiple organ failure.

Manifestations / course
Findings established by the emergency physician on his arrival included excessive sweating. The patient groaned because of convulsive abdominal pain and had vomited. Findings on admission to hospital documented a blood pressure of 60/35 mmHg and a heart rate of 120/min. A garlic-like foetor *ex ore* was established. Initially, the patient was conscious and responded to questions without delay.

Laboratory analyses revealed a blood alcohol level of 1.92 ‰ and a high CK of 679 U/L (reference value: 25–90 U/L). In addition, high levels of transaminases and gamma-GT were found. Subsequently, the patient rapidly became somnolent. Respiratory insufficiency required intubation, which could be performed without complications. Administration of high doses of corticoids was required due to the risk of toxic lung oedema. Due to suspected aspiration, an antibiotic therapy with cefuroxime was initiated. Hyperventilation was performed by means of a respirator. In spite of catecholamine administration and fluid substitution, a sufficient perfusion pressure could not be re-established. In spite of intensive therapy, the patient died on the next morning from multiple organ failure.

Notes
Selenium (Se) is a trace element essential for the human body. It is contained in a number of proteins and enzymes in the form of selenocysteine. Due to its important function as a cofactor of glutathione peroxidase, it represents an essential component of the antioxidative system. Animal studies have revealed its interaction with heavy metals such as arsenic, barium, lead, cadmium, mercury, silver, thallium and tin. Therefore, selenium has been assumed to have a detoxifying effect on the toxicity of these elements. Selenium deficiency will cause Keshan disease which is an endemic cardiomyopathy. Another condition known to occur is the Kashin-Beck disease, a syndrome characterized by dystrophic osteoarthrosis and spondyloarthrosis.

At selenium levels of less than 50 µg/L, administration of selenium preparations such as sodium selenite, selenomethionine or selenium yeast is indicated to improve antioxidative protection. In the Federal Republic of Germany, mean selenium plasma levels are 70–80 µg/L (reference range in adults: 50–120 µg/L plasma/serum).

In literature, little information exists regarding the mechanisms of acute toxic effects of selenium. In contrast, many studies are found that discuss possible protective effects of selenium.

Nevertheless, it is possible to derive from these some of the toxic effects and their mechanisms. The statements below refer to a personal communication by Prof. Dr. med. Gerhard Hennighausen, Centre for Pharmacology and Toxicology of Rostock University.

- Cytotoxic and antiproliferative effects of selenium at low concentrations (10 µg/L sodium selenite up to 1 mg/L);
- Disruption of glutathione functions due to oxidation of glutathione (GSH) to glutathione disulfide (GSSG), resulting in pro-oxidative effects; Substitution of SH groups in enzymes by selenium;
- Disruption of other functionally important SH groups or S-S bonds;
- Disruption of the function of metals forming selenides with selenium.

The most important cardinal sign of selenium poisoning is the very intensive smell of garlic in the expiratory air caused by the metabolic product, dimethyl selenide. However, no general manifestations can be described. Manifestations of poisoning observed in cases of selenosis include:
Foetor *ex ore* of garlic, gastrointestinal complaints such as nausea, abdominal pain and diarrhoea, tiredness, exhaustion, irritability, headache, peripheral neuropathy, rhinitis, hoarseness, dermal eczema, hair loss, softening of nails (onycholysis), and weight loss.

Up to the present, the BfR has received reports on three fatal cases of selenium poisoning. Common manifestations of these included the garlic-like smell, gastrointestinal complaints and increasing respiratory insufficiency followed by shock symptoms. In all these cases, cardiopulmonary resuscitation remained unsuccessful.

Evaluation of the case described
Based on the information received on the temporal relationship between the suicidal ingestion of 1–2 g sodium selenite and the development of manifestations, and in the absence of other causes, a causal relationship is probable.

3.4.2 Poisoning associated with foods

3.4.2.1 Roast veal, stuffed

Suspected food poisoning involving ca. 56 persons at a wedding celebration
At a wedding celebration hosting about 240 guests, 56 fell ill, 22 of which were admitted to hospitals. They could be discharged after one day. The suspected cause was a contamination of stuffed roast veal with staphylococcal toxins. However, the toxins could not be detected. Mushroom poisoning was excluded. The BfR has received reports from the Public Health Service and two medical reports from physicians.

Manifestations / course
Approximately 1.5 hours after the wedding lunch, about 40 of the 250 guests began to suffer from diarrhoea and vomiting. All persons affected reported to initially have vomited repeatedly which was followed by diarrhoea.

One of the medical reports referred to the 37-year-old bridegroom. He was admitted to hospital in a clearly reduced general condition with a pale appearance and sunken eyes. Clinical findings were normal except for a painful tenseness in the left hypogastric region. The ECG did not reveal any abnormal findings. Laboratory analysis revealed high creatinine levels (1.36–1.12 mg/dL), high CK (211–168 U/L) and a high leukocyte count (14 900–9 800/µL). The patient could be discharged on the second day after his admission to hospital.

The other medical report referred to an 8-year-old boy. He was admitted to hospital with abdominal pain, vertigo and weakness. He had eaten two bites only of the roast veal, but a double portion of potato gratin as well as the dessert offered. The boy had developed neither vomiting nor diarrhoea but suffered from persistent weakness and tiredness. His condition slowly improved under infusion therapy. Having tolerated several meals of a light diet, he could be discharged in an improved condition on the following evening. According to information given by his mother, the boy was still confined to bed suffering from weakness, appearing pale and sleeping for extended periods of time on the following days. The boy's father, who had eaten his and his son's portion of the roast veal, had developed diarrhoea.

A 70-year old female patient reported that at the wedding celebration, she had eaten a

complete lunch at noon consisting of soup, salad, main course including roast veal and potato gratin, and finally the dessert. She had eaten up all of her portions because the meal had been so delicious. At about 16:30 h, the first symptoms set in, together with a strong feeling of indisposition and an urge to breathe fresh air. After a short walk, she began to suffer from severe vomiting and an uncontrollable discharge of watery stools. She was given medical care on the scene and subsequently brought to the next hospital. She was discharged and referred to outpatient treatment on the following day. Two weeks after the incident, she was still suffering from inappetence, tiredness and weakness. She had been in a healthy condition prior to the festivity.

Notes
Based on the final report by the food control authority, there was a founded suspicion that the collective outbreak had been caused by a toxin. It was assumed that probably, the incident had to be attributed to a staphylococcal infection resulting in enterotoxin formation.

Altogether, 20 food samples were examined. Neither pathogenic organisms nor toxins could be detected in any of the 20 samples. Mushroom poisoning was excluded by the examination of food samples. Only the roast veal was classified as unfit for consumption because of its odour (test result: sensory deviation of olfactory properties). The sensory findings were substantiated by positive findings of putrefaction using Nessler's reagent, by detection of lead acetate in one sample and a low pH value. The roast veal had been cooked for ca. 16 hours at 75 °C, i.e. using the low-temperature cooking method. It is assumed that this process may not ensure a reliable killing of the pathogenic organisms present. Even a thoroughly cooked food may still contain toxins pathogenic for humans even if the responsible organisms can no longer be cultivated by microbiological methods.

The clinical picture of the illnesses suggested a toxic process both with regard to the temporal relationship and the manifestations observed. Cooking of stuffed meat parts using the low-temperature cooking method appears to involve a considerable and incalculable risk concerning the microbiological quality of the stuffing.

Evaluation of the case described
Based on the information given on the temporal relationship between the exposure and the onset of manifestations, and in the absence of other causes, a causal relationship has been rated as probable in the above cases of generally minor health impairment.

3.4.3 Poisoning associated with plants

3.4.3.1 Monkshood (*Aconitum napellus*)

Cases of poisoning with monkshood mistaken for parsley
The cases occurred at a barbecue party of a family and their friends. A salad offered at the party contained leaves of garden monkshood that had been mistaken for parsley. As a consequence, four out of six persons who had eaten the salad developed health complaints. All of them were subjected to monitoring at the intensive care unit.

Manifestations / course
Patient No. 1:
Two hours after ingestion of the meal, a 19-year-old female complained of numbness in her mouth and a tingling sensation in her entire body. Presumably, she had eaten the largest amount of salad. On arrival of the emergency physician, the patient's heart rate was 86/min and her systolic blood pressure, 100 mmHg. The ECG revealed a sinus rhythm associated with intermittent bigeminy.

On admission to hospital, findings included a reduced general condition, and the patient appeared bradyphrenic. Her skin colour was pale, her heart rhythm was tachycardic and arrhythmic. Her trembling all over was similar to muscular tremor, and she complained of numbness of all her extremities and her trunk. Laboratory parameters were within the normal ranges. The ECG performed on admission to hospital revealed a sinus rhythm and a continuous bigeminy with otherwise normal potential curves. After i.v. administration of magnesium, a normal sinus rhythm was found in the follow-up examination some hours later. The patient still complained of tremor and exhibited uncontrolled muscular twitching so that she was unable to walk or move adequately. All manifestations persisted for almost 24 hours.

After consultation of a Poison Control Centre, gastric lavage was performed and charcoal administered. This was followed by i.v. administration of magnesium with 2 g as a bolus, and 2 g/h via a perfusor. After two days, the patient could be discharged completely symptom-free and referred to her family doctor's care.

Patient No. 2:
Another female aged 20 years also complained of numbness in the region of her mouth and on both arms, and of meteorism, approximately two hours after the meal.

Clinical examinations revealed irregular heartbeats. The patient appeared lethargic and bradyphrenic. She persistently suffered from numbness and a tingling sensation on both hands. Routine laboratory analyses resulted in normal findings. The ECG revealed a sinus rhythm and first-degree AV block (PQ interval 0.34 sec). Intermittent bigeminus occurred. After i.v. administration of magnesium, cardiac arrhythmia persisted for three hours until it was followed by a normofrequent sinus rhythm. The therapeutic approach was similar to that in the case of the first patient. The patient was symptom-free on the following day and could be discharged.

Other patients
A 46-year-old male presented with numbness of extremities and meteorism on admission to hospital. The ECG revealed bigeminy. During the night, polymorphic ventricular extrasystoles were observed. Magnesium administration resulted in an improvement.

A 39-year-old female also presented with numbness in her oral region on admission to hospital. She exhibited initial agitation and tingling paraesthesia, but was completely symptom-free on the following day.

Two adolescents aged 12 and 15 years did not develop any manifestations and had no cardiac arrhythmia later. According to the information given, they had eaten only small amounts of the salad.

All six persons were subjected to monitoring at the intensive care unit.

The garden monkshood could be reliably identified by a biologist.

Notes
The monkshood (*Aconitum napellus*, garden monkshood, aconite, blue rocket, friar's cap) is the most poisonous plant indigenous to Europe. Equivalent toxicological properties are found in the yellow wolfsbane (*Aconitum vulparia*) and the European or Manchurian monkshood (*Aconitum variegatum*).

The monkshood grows mainly in mountainous regions of the temperate zones of Europe, but

also in Asia and North America. It is a popular ornamental plant grown in many gardens. Ingredients contained in all parts of the plant include toxic diterpene alkaloids, with the main active substance being aconitine. Therefore, all parts of the plant are toxic, and particularly so, the tuber and the seeds. Cases of poisoning have frequently occurred due to the tuber being mistaken for celeriac or horseradish roots. The leaves are known to have often caused poisoning after being used as a salad green either due to ignorance or as in the above case, because they were mistaken for parsley. The aconitine concentration found in the leaves is 2 %. The highest alkaloid concentration (up to 3 %) is found in the tubers in winter. For an adult, the lethal dose of pure aconitine is about 2–6 mg. Consequently, ingestion of a few grams of fresh vegetal matter only will suffice to absorb such a dose. The poison is rapidly absorbed through the mucous membranes or the gastrointestinal tract, but also through the intact skin. The toxic effect is based on an increased permeability of irritable membranes for sodium ions, a prolongation of sodium influx during the action potential and a delay of repolarization of sensory and motor nerve terminals. Due to the stimulation of peripheral sensory nerve terminals, a number of reflexes is induced. Higher concentrations will result in a paralytic effect on the sensory nerve terminals, and in addition, an initial stimulation and subsequent inhibition of the CNS. Effects on the heart consist in reflectory bradycardia and a stimulation of secondary and tertiary pacemaker centres resulting in arrhythmia. Death will result from cardiac arrest caused by ventricular fibrillation or central respiratory paralysis.

In folk medicine, the monkshood tuber has been administered in small quantities both internally and externally for pain control in conditions such as neuralgia, myalgia, muscular and articular rheumatism, inflammation of serosae and migraine. Due to the low number of its therapeutic uses, the substance is almost exclusively found in homeopathic preparations today. Al-

Fig. 10: Leaves of Aconitum napellus

Fig. 11: Leaves of parsley

ready small quantities may cause paraesthesia – most often a feeling of coldness and a tingling sensation or even total numbness (“icy water in the veins”, anaesthesia dolorosa), vomiting, vertigo, myospasms, generalized convulsions, paralysis, hypothermia, bradycardia and cardiac arrhythmia which may end in ventricular fibrillation and central respiratory paralysis. In cases of acute poisoning, manifestations will develop within a few minutes and will include racking vomiting, diarrhoea, slow and irregular respiration, arrhythmia and bradycardia, irregular and weak pulse, hypotension and visual disturbance. Death will occur due to respiratory paralysis and cardiac failure in a state of con-

sciousness. An adult may die from ingestion of only 2–4 g of the tuber. Dangerous manifestations may occur already after a short latency period of 10–20 minutes. Ingestion of any amount, even if only suspected, has to be rated as life-threatening. Due to a possible rapid onset of dramatic manifestations, transport to a hospital accompanied by an emergency physician should be arranged for, as done in the cases described above. Recommended therapeutic measures include immediate gastric evacuation by administration of gastric lavage. Subsequently, administration of charcoal and Glauber's salt and monitoring of the patient at an intensive care unit should be provided for. In cases of most severe arrhythmia (torsade de pointes), administration of magnesium at high doses is recommended, initially as a bolus and followed by continuous infusion. This approach was also taken in the above cases.

Evaluation of the cases described:
On the basis of the information given on the temporal relationship and in the absence of other causes for the manifestations developed by the patients, a causal relationship between the ingestion and the manifestations observed has been rated as probable in the cases described above.

3.4.3.2 Siberian peashrub (*Caragana arborescens*)

Accidental ingestion of pods of the Siberian peashrub in a schoolyard requiring inpatient treatment
Eleven 8-year-old pupils had nibbled the pods of a shrub growing in their schoolyard. Initially, laburnum had been suspected to be the plant involved. Therefore, all children were admitted to hospital and put under medical observation. Still on the same day, an official of the urban green space planning office responsible for greenery in schoolyards searched for laburnum, which has been banned from schoolyards. However, he found only the peashrub.

Manifestations / course
At the time of their transport to the hospital, no manifestations had been observed yet in the children. In the course of the afternoon, three out of the eleven children affected developed quite severe manifestations and exhibited a reduced general condition. According to the information by the attending physician in the hospital, these were the children who had eaten the largest amount of pods, although the exact quantity ingested could not be established. The predominating manifestations included gastrointestinal complaints such as abdominal pain, nausea, vomiting and diarrhoea. They were associated with a tendency towards collapse and headache. Therefore, all children involved had to stay at the hospital overnight. They could be discharged in a symptom-free condition on the following day.

Notes
The Siberian peashrub, or pea tree (*Caragana arborescens*) is a member of the Fabaceae family. It is native to eastern Siberia and the temperate zones of China. The shrub or tree is up to 5 m high and quite often found in parks and gardens. Its leaves are alternate and pinnately compound with leaflets in pairs – in contrast to the laburnum, which has long-stalked trifoliate leaves each consisting of three elliptic leaflets. In contrast to laburnum pods, those of the peashrub are spiky, they have multiple seeds and burst open in their ripe state. Exact data on toxicity are still missing. Therefore, special importance is to be attributed to the collection of knowledge gained in cases of poisoning involving this species.

Although the Siberian peashrub is regarded as toxic, it is very doubtful whether the plant con-

Fig. 12: Peashrub

tains cytisine (quinolozidine alkaloid) similar to laburnum. Particularly the seeds contain lectins, i.e. molecules consisting of protein and carbohydrate components. One of these lectins has the ability to bind to N-acetyl galactosamine, and another, to L-canavanine (ca. 6 %), a fact that may have been responsible for the toxic effects observed. In a publication dated 1966, the following statement is found: "The fat-containing seeds make a good feed for poultry and have also been recommended as an emergency food for humans." Given the manifestations described in the above case reports, such statement can no longer be regarded as valid.

Evaluation of the cases described:
Based on the information given on the temporal relationship between the ingestion and the onset of manifestations, and in the absence of other causes, a causal relationship has been rated as probable in the above cases of generally minor health impairment.

3.4.4 Poisoning associated with antifreeze, ethylene glycol

3.4.4.1 Suicidal ingestion

Severe case of ethylene glycol poisoning after ingestion of a print head cleaner
A 49-year-old female had ingested 100 mL of a print head cleaner, probably in an attempt to commit suicide. The identification of the toxic component was difficult because there was neither any additional information given on the package nor had the Poison Control Centres any data available on the product concerned. Meanwhile, the manufacturer of the product had gone insolvent. Police investigations found the former manager of the firm who confirmed glycol to be the toxic component. Ethylene glycol was detected by chemical analysis. After eight days of inpatient treatment, the patient was discharged and referred to the care of her family doctor.

Manifestations / course
The patient was found unconscious in the flat by her husband and then admitted to hospital by emergency ambulance. Presenting in a state of deep coma and Kussmaul's breathing, she was intubated and respirated.

Laboratory examinations revealed a considerable anion gap acidosis. Blood gas analysis revealed a pH of 6.9 mmol/L (reference range: 7.35–7.45), standard bicarbonate 3.0 mmol/L (reference range: 22–26), pCO_2 20.8 mmHg (reference range 35–45), pO_2 41.4 mmHg (reference range: 70–100), base excess -26 mmol/L (reference range: -3.0–3.0). Toxicological analysis revealed ethylene glycol blood levels of 0.25 mg/mL and 0.09 mg/mL, respectively, on the day of admission and on the following day. The time of ingestion could not be established. Since laboratory findings on admission of the patient to hospital suggested a considerably advanced

ethylene glycol metabolism, the ingestion was assumed to have occurred about 10–15 hours earlier.

Therapeutic measures included the administration of sodium hydrogen carbonate and ethanol as an antidote. Initially, administration of fomepizole was considered. It was omitted, however, because on arrival of this antidote, which had to be ordered, ethanol infusion was already being administered. In addition, haemodialysis was performed. On the following day, a creatinine increase to 5.2 mg % and oliguria were observed as signs of intrarenal failure. After numerous complications requiring an artificial pneumothorax, among other measures, the patient could be discharged with retention values having returned to normal levels after eight days of inpatient treatment.

Evaluation of the case described
Based on the information given on the temporal relationship between the ingestion and the development of manifestations and given the detection of toxic ethylene levels in the blood, and in the absence of other causes, a causal relationship has been rated as confirmed in the above case.

3.4.4.2 Accidental ingestion

Ethylene glycol poisoning in children
Two siblings aged 6 (with trisomy 21) and 3 years had ingested an unknown quantity of an antifreeze. They had been sitting on the back seat of their parents' estate car when they discovered the bottle with the product stored behind them in the car, then took the bottle and opened it. At that time, the children had not been guarded by family members. Only afterwards, the parents noticed the empty bottle and spilled antifreeze. Ingestion could not be reliably excluded. Therefore, the children were presented at a paediatric hospital and after consultation of a Poison Control Centre, admitted immediately to a university hospital for onward treatment with fomepizole as an antidote. After the third day of inpatient treatment, the children could be discharged in a good general condition and referred to outpatient care.

Manifestations / course
In the 6-year-old child exhibiting the typical signs of trisomy 21, no abnormal findings were made on admission except for perioral eczema and rhinitis. The blood gas analysis revealed a pH of 7.37 (reference range: 7.35–7.45), a standard bicarbonate of 19.4 mmol/L (reference range: 22–26 mmol/L) and a base excess of –5.9 mmol/L (reference range –3.0–3.0 mmol/L).

Still on the day of admission, a therapy involving fomepizole as an antidote was initiated. Already on the second day of treatment, the acid-base balance returned to normal. Thus, the patient could be discharged and referred to outpatient treatment after administration of four doses of fomepizole.

The 3-year-old child was in a good general condition, no abnormal findings were made on admission. The blood gas analysis revealed a standard bicarbonate of 20.8 mmol/L and a base excess of -4.2 mmol/L. Also in this patient, a therapy with fomepizole was initiated still on the day of admission. The antidote was administered twice a day in a dosage based on the child's body weight. As in the older sibling, the initial mild and compensated acidosis returned to normal on the second day of treatment. Also the younger child could be discharged on the third day of treatment and referred to outpatient treatment after administration of four doses of antidote.

Evaluation of the case described
Based on the information given on the temporal relationship between the ingestion and the development of manifestations including the confirmation of mild acidosis, a causal relationship has been rated as probable in both cases described above.

Fig. 13: antifreeze

Notes
Ethylene glycol is a colourless and odourless fluid with a pleasant and sweet taste. It is used as an antifreeze, e.g. for car radiators, and for a number of other purposes. After oral ingestion, it is absorbed readily and completely. A 25 % share of the ethylene glycol absorbed will remain unchanged and is excreted by the renal route, while a 75 % share will undergo oxidation by the hepatic alcohol dehydrogenase (ADH) to form glycolaldehyde and subsequently, glycolic acid (mean biological half-life: 4.5 h). A minor share of the glycolic acid will undergo further oxidation to oxalic acid, which is eliminated by the renal route. Above all, formation of the toxic metabolites, glyoxalic acid and glycoaldehyde will result in damage to the CNS, liver, kidneys, lungs and heart. The formation of organic acids will cause severe acidosis. Deposition of oxalates in the walls of small vessels (brain and kidneys) may occur. Mainly crystallization in the renal tubules will cause renal failure due to blockage (oxalosis).

The toxic dose has been stated to be 0.1 ml/kg b.w., the toxic plasma level, 0.1 mg/mL.

Manifestations are characterized by four stages:
Stage one:
Central nervous, gastrointestinal and metabolic manifestations within a period of between a few minutes and 12 hours after ingestion involving drunkenness (in the absence of alcohol foetor), somnolence which may develop into coma, agitation, nystagmus, convulsions, nausea, vomiting, abdominal complaints, metabolic acidosis.
Stage two:
Cardiorespiratory manifestations within a period of 12–14 hours after ingestion involving dyspnoea, tachypnoea, mild hypertension and arrhythmia, pulmonary oedema, circulatory failure.
Stage three:
Renal manifestations within a period of 24–72 hours after ingestion involving oliguria, creatinine increase, proteinuria, haematuria, oxaluria, renal failure due to acute tubular necrosis and cerebral oedema.
Stage four:
Central nervous manifestations within several days after ingestion involving bilateral peripheral facial palsy, increase of cerebrospinal protein levels, anisocoria and visual disturbance, hyperreflexia, ataxia, dysphagia and vomiting.

Since the toxicant is absorbed quickly, its primary removal is meaningful as a therapy only within the first hour after ingestion. The gastric contents should be evacuated using a gastric tube. Charcoal administration is ineffective due to its low binding capacity and therefore not indicated. As an antidote, ethanol is administered. Its principle of action is a blocking of the ethylene glycol metabolism on the basis of a higher affinity for ethanol of the alcohol dehydrogenase. Thus, the latter is no longer available for the metabolism of ethylene glycol, which will prevent the formation of toxic metabolites and result in the elimination of the non-toxic unchanged ethylene glycol. For a couple of years, experience has also been gained from adminis-

tration of the highly effective alcohol dehydrogenase inhibitor, 4-methylpyrazole (fomepizole) as another antidote and alternative to ethanol.

The frequent presence of extremely severe acidosis may require administration of high doses of 1M sodium hydrogen carbonate as a symptomatic measure. In cases of uncontrollable acidosis or plasma levels of 0.5 mg/mL and above, early haemodialysis is recommended as an effective measure for secondary detoxification, in addition to the antidote therapy. Decisive importance has been attributed to an early initiation of therapy, which may have a considerable influence on the course of the poisoning.

3.4.5 Cases reported by environmental health clinics

3.4.5.1 Naphthol

Health complaints suspected to be caused by textiles

A 24-year-old female presented to an environmental health clinic with health complaints she had experienced to intensify at her workplace in a textile retail establishment. She was admitted to hospital for toxicological examinations. Toxicological analysis revealed naphthol and pyrethroid levels in her urine.

Manifestations / course

The patient suffered from vertigo, nausea, a sensation of a lump in her throat, headache and dry cough. Such complaints had intensified at her workplace in the textile retail business. Her condition had largely improved during periods of absence from her workplace, e.g. during periods of leave. The patient's duties at the workplace included unpacking, distribution and selling of textiles from overseas, among others such from India and Third-World countries. She stated that the textiles had a "musty" smell, and that her hands had always discoloured to grey during her working day. Particularly when opening packages from overseas, she had suffered from cough, excess tear secretion, vertigo and nausea, pruritus and reddening of the skin in different areas.

Toxicological analysis revealed a beta-naphthol level in her urine of 18.00 µg/L. In addition, elevated levels were detected of the pyrethroid metabolites, phenoxybenzoic acid (1.0 µg/L; reference range: 0.0–0.7), cis-Br2CA (0.6 µg/L; reference range: 0.0–0.9), and trans-C12CA (0.9 µg/L (reference range: 0.0–0.9).

Clinical-chemical analysis performed prior to her annual leave (possible exposure) revealed pathological levels of LDH 220 U/L (reference range: 135–214), haematocrit 44.1 % (reference range: 37–43), neutrophils 77 % (reference range: 40–70), lymphocytes 18 % (reference range: 25–40), and eosinophils 1 % (reference range: 2–4). After four weeks of leave, no pathological levels were found.

Appropriate measures of occupational safety or if possible, a change of the workplace were recommended, although the confirmed low naphthol and pyrethroid exposure did not sufficiently explain the manifestations observed.

Notes

Beta-naphthol is used for the production of aromatic substances, colorants and tanning agents. In the Regulations on Dangerous Substances and Materials, it has been classified as "harmful by inhalation and if swallowed" and as "dangerous for the environment". Pyrethroids are used in cotton plantations for plant protection and ectoparasite control on wool-producing animals.

Under the German Textile Labelling Act, labels in textiles are required to provide information on the textile fibres, but not on the auxiliary substances used. As far as clothes are concerned, these have been assigned to the group of "Other Commodities" under the Act on Manufacture, Treatment, Handling and Distribution of Foods, Tobacco Products, Cosmetics and Other Commodities (Foods and Other Commodities Act). According to § 30 of this Act, production or treatment of commodities by methods possibly harmful to health has been banned. This also applies to finishing and impregnating agents. Compliance with the legal provisions is a responsibility of the manufacturer. Control of the compliance with the legal provisions is in the responsibility of the German federal Länder. However, similar to other goods referred to as "other commodities", neither authorization nor notification are legally required for clothes. This is why the authorities have no comprehensive knowledge on these products. No sufficient information has been available on the use of dyeing and finishing agents, particularly in imported textiles.

Evaluation of the case described
On the basis of the information given on the temporal relationship between the exposure and the development of manifestations of a minor health impairment, and in the absence of other causes, a causal relationship has been rated as possible in the case described above.

3.4.5.2 Surface coatings, adhesives

Exposure to solvents from adhesives over decades
A 64-year-old male presented at the environmental health clinic with health complaints he attributed to his working environment as an interior decorator. Over decades, he had been using a variety of surface coatings and adhesives such as neoprene adhesives, epoxy resin and synthetic resin. Toxicological

Fig. 14: Adhesives, paints

analyses performed did not find any definite indications of the presence of solvents from adhesives. Nevertheless, the case was reported to the competent Berufsgenossenschaft as an occupational illness because of the clear association with the working environment and exposure to solvents over decades.

Manifestations / course
The patient complained of sweating, nausea, vomiting, headache and dyspnoea during his work with adhesives and surface coatings. In addition, he complained of tingling paraesthesia in his feet. He had been suffering from these symptoms since the autumn of 2003. Already in the previous year, a diagnosis of beginning polyneuropathy had been established on the basis of clinical and electrophysiological examinations performed at a university hospital. There, the patient had been recommended to consult an environmental health clinic for examination to clarify a possible toxic origin of his complaints. Paraesthesia was experienced on both hands, particularly in the patient's ring and little fingers. In addition, he suffered from cramps in his toes and calf muscles and a burning sensation in his feet. The patient also reported to react to petrol, tar and nail polish remover with similar but less pronounced symptoms. In addition, he had been suffering

from bronchial asthma for 35 years, and a tentative diagnosis of diabetes mellitus had been made.

Based on clinical and neurological examinations (the latter had been performed for orientation purposes) a normal organ status was found.

The toxicological analysis for solvents revealed an acetone concentration of 5 mg/L. This was still within the normal physiological range. Other volatile components found included butanol, benzenedicarboxylic acid ester and a phenol derivative in a very low concentration. However, the detection of these substances alone did not permit any further interpretation. Altogether, no reliable indications of a presence of solvents from adhesives were found. Also, it had to be taken into account that the patient did no longer work and almost no recent exposure had taken place.

However, it was established that the patient had experienced his symptoms in a plausible temporal relationship with the use of a number of adhesives in his working environment as an interior decorator. To exclude an underlying psychiatric disease, a structured clinical interview was performed which did not reveal any abnormal findings. Due to the clear relationship with the patient's workplace and his exposure to solvents over decades, the case was reported to the Berufsgenossenschaft as an occupational illness in 2005. In addition, allergological examinations were recommended to elucidate the origin of the patient's health complaints.

Notes

Surface coatings may contain a wide range of solvents. Adhesives may contain different parent substances such as synthetic rubber and synthetic resins, as well as a number of solvents including aromatics and aliphatics.

Exposure to neurotoxic organic solvents may result in toxic polyneuropathy or encephalopathy. According to the current state of scientific knowledge, such substances include n-hexane and n-heptane belonging to the aliphatic hydrocarbons. Among the ketones, butanone-2 and 2-hexanone, and among the alcohols, methanol, ethanol and 2-methoxyethanol may cause toxic neuropathy. Among the aromatic hydrocarbons, benzene, toluene, xylene and styrene have been proved to be neurotoxic solvents. The chlorinated aliphatic hydrocarbons include dichloromethane, 1,1,1-trichloroethane, trichloroethene and tetrachloroethene.

Organic solvents are predominantly inhaled through the lungs. However, owing to their high volatility they are also absorbed through the skin. They will spread over the entire body, particularly in the nervous system. Subsequently, they are exhaled again through the lungs in an unchanged state, and in part, metabolized and eliminated through the kidneys. Elimination half-life periods vary between a few hours and two days. There are various targets for the metabolites of neurotoxic solvents (e.g. 2,5-hexanedione as a neurotoxic metabolite of n-hexane and methylbutyl ketone) in the nerve cell which in part, have not yet been identified. Axonal transport disruption will initially result in functional disturbances in the form of paraesthesia and sensory loss. In the further course, morphological disturbances associated with axonal damage may develop. This process may be influenced by other neurotoxic factors such as diabetes, alcohol or medicinal products.

In a number of patients, polyneuropathy caused by solvents will improve after termination of the exposure during their use. However, in many cases, the disease will persist or even deteriorate after discontinuation of the harmful work. Hence, persistence or deterioration of the dis-

ease after termination of the harmful work does not exclude solvents as the cause of such health impairment. In a revised version of its information leaflet, BK-No. 1317, of 11 February 2005 (Polyneuropathy or encephalopathy due to organic solvents or mixtures of these), Annex to the Regulations on Occupational Diseases (BKV), the Hauptverband der Berufsgenossenschaften (German Federation of institutions for statutory accident insurance and prevention) has taken account of this fact in its scientific argument.

Evaluation of the case described
Based on the information given on the temporal relationship between the exposure and the development of manifestations of a moderate health impairment, and in the absence of other causes, a causal relationship has been rated as possible in the case described above.

3.4.6 Poisoning by inhalation

3.4.6.1 Wood preservatives

Considerable health impairment after exposure to wood stain
A 38-year-old female had been applying a wood preservative stain for several days. Subsequently, she developed generalized oedema, hepatosplenomegaly and additional signs of right ventricular failure and temporary pancytopenia and obstructive ventilatory disorder. After termination of the exposure, regression of disease took place without essential therapeutic assistance.

Manifestations / course
In July 2005, the patient applied a wood stain to the beams of her house, working for ca. 2.5 hours. The wood stain applied contained more than 50 % naphtha and mineral spirits, less than 5 % dichlofluanid, less than 0.05 % butyl diglycol and less than 0.05 % 2-methyl-2,4-pentanediol. During the work, she wore neither a mask nor gloves for protection. It has to be assumed that a considerable inhalational exposure took place since the roof overhang formed a semi-closed room where fumes collected over the patient's head, and the wood stain was applied to an extended surface. The patient noticed a solvent-like smell and an unusual taste on her tongue, and she developed a dry cough and headache. After the first day of work, the initial exposure resulted in facial swelling without change of the colour of her skin. On the following day, the patient carried on working for about 4.5 hours. She noticed pronounced headache, nausea and aching muscles. The facial swelling increased. Irrespective of her health complaints, she carried on working for another eight hours. In the evening after work, she noticed oedemas on her lower legs for the first time. On the following day, her general condition was markedly reduced. In addition to her lower legs and her face, also her hands were swollen, and she suffered from increasing nausea. Four days after she had started her painting work, she presented to her family doctor with marked anasarca (her jeans fitted no longer due to a body weight increase of 7 kg within a few days). On the following day, the patient developed orthopnoea for the first time. She vomited repeatedly, and the oedemas increased. She complained of a recurrent oppressive feeling in her retrosternal and epigastric region when in a lying position. She was admitted to hospital.

A friend who had helped her with the painting work on two days for 4.5 and 8 hours, respectively, did not develop any symptoms.

Computer tomography performed at the hospital revealed pulmonary effusions, hepatosplenomegaly, broad congestion of hepatic veins, portal hypertension, gall-bladder wall thickening, discrete perihepatic ascites

and cardiomegaly. A tentative diagnosis of myocarditis was made. No more information on the differential diagnosis was received. Abnormal findings revealed by laboratory analysis included tricytopenia with an Hb of 9.7 g/dL, thrombocytes of 129 G/L and leukocytes of 3.2 G/L. GPT was elevated: 67 U/L. Body plethysmography was performed and revealed bronchial obstruction associated with an increase in expiratory resistance. At the time of this examination, the general condition of the patient had improved without essential therapeutic assistance.

Since the patient had no medical history of lung disease and no other inhalation toxicants could be identified in her history, a ventilation disorder induced by a toxic or allergic cause had to be assumed. This was to be examined by appropriate testing. The results are still pending.

Notes

The product used in the above case is a wood stain containing naphtha as a solvent, i.e. a mixture of aliphatic (and aromatic) hydrocarbons, mineral spirits and glycols. As an anti-blue stain agent, the product contained < 5 % dichlofluanid. According to the information received, a considerable inhalational (and dermal) exposure to the components took place.

Mineral spirits and **naphtha** cause irritations of the skin and mucosa. Inhalation of higher concentrations may result in lung damage and central nervous disturbances. Extended exposure is known to cause hepatic and renal damage as well as moderate medullary hypoplasia.

Likewise, exposure to **glycols** has been observed to cause irritation of the skin and mucosa and inflammatory infiltrates of the bronchi, and in single cases, changes in the blood count and hepatic damage. Also sensitizing properties have been recorded.

Dichlofluanid was introduced into the market in 1964 as an active substance in plant protection products and wood preservatives, mainly acting against fungal infestation. Under the Regulations on Dangerous Substances and Materials, it has been classified as “harmful” (Xn). A striking property is a higher inhalation toxicity (LC_{50} rat, inhalation 1.2 mg/L air 4 h and in studies in 1966, 0.3 mg/L, respectively) when compared with oral and dermal toxicity (LD_{50} rat oral and LD_{50} rat, dermal > 5000 mg/kg b.w.). Dichlofluanid is irritating to skin and eyes and has sensitizing properties. Observations made so far have resulted in the assumption that also in humans, adverse effects may occur in the form of irritation of the eyes and mucous membranes, increase in general susceptibility to infections, dejection, weakness, decrease in vitality, nausea, headache, respiratory disorders, deficiency of red blood cells, liver and kidney disorders, and sensitizing effects.

Evaluation of the case described

Based on the information given on the temporal relationship between the exposure and the development of manifestations of a moderate health impairment, and in the absence of other causes, a causal relationship has been rated as possible in the case described above.

3.4.6.2 Carbon monoxide

Occupational accident associated with an internal combustion engine

In the context of their work as grinders, two workers aged 49 and 42 years had been working with machinery powered by an internal combustion engine. They had been working for about 1–1.5 hours in a closed room from which the exhaust gases could not escape. Because of health complaints, inpatient

treatment was required which included hyperbaric oxygenation.

Manifestations / course
Both patients suffered from headache and nausea. Their carbon monoxide Hb levels (COHb) were 31.5 % and 24.6 %, respectively. Except for tachycardia (100–109/min), there were no abnormal findings. Both patients were treated with hyperbaric oxygenation. As a result, their symptoms disappeared. During subsequent observation over 12 hours, the patients' conditions remained stable. Also follow-up examinations performed six days later did not reveal any neurological or other deficiencies.

Carbon monoxide poisoning from heating with wood bricks
A 35-year-old female suffered a carbon monoxide poisoning in the context of flue gas inhalation after she had heated her stove with wood bricks. The district chimney sweep and the responsible police station were informed about the life-threatening incident by the attending emergency centre.

Manifestations / course
The patient had been found early in the morning by her daughter with a disturbance of consciousness, encopresis and enuresis. On admission to the hospital, organ findings were normal but the blood gas analysis revealed an elevated COHb level of 23.7 %. Therefore, the patient was administered hyperbaric oxygenation. On the following day, arterial blood gas analysis revealed a COHb level of 0.1 %, associated with a pO_2 of 97.5 mmHg and balanced electrolytes. The patient could be discharged in a symptom-free condition and referred to outpatient care.

Notes
Carbon monoxide (CO) is produced by incomplete combustion of coal and carbon compounds. It is a colourless, odourless and tasteless gas and burns with a blue flame forming carbon dioxide. It is slightly lighter than air and therefore, rises with warm air. Due to its diffusing power it readily penetrates walls and ceilings. Combustion gases from internal combustion engines may contain 4–11 % CO.

Carbon monoxide is haemotoxic. It binds to haemoglobin to form carbon monoxide Hb (CO-Hb) by displacing the oxygen. Possible organ damage due to oxygen deficiency may result in permanent brain damage or even acute death from suffocation. The affinity of carbon monoxide to haemoglobin is 300 times higher than that of oxygen. This is the reason for the hazard posed by CO. In acute poisoning situations, individuals differ considerably as to their sensitivity to carbon monoxide. Because of their higher respiration rate and more intense metabolism, adolescents and small and delicate persons are more at risk, as a rule, than adult and sturdy persons.

Already at COHb levels as low as 4 %, changes can be detected in psychomotor tests. Headache, weakness and vertigo will occur, as a rule, if ca. 30 % of the haemoglobin has become bound to carbon monoxide. Levels above 40–50 % will result in collapse and unconsciousness. If the respiration is deprived of 75 % of the haemoglobin, the inevitable result will be death from suffocation. As compared to younger individuals, lethal COHb concentrations become lower with increasing age. Carbon monoxide poisoning is particularly often followed by late damage and secondary diseases that may occur mainly as sequelae of hypoxia experienced over a more or less extended period. Frequent sequelae include brain damage of different types because the central nervous system is highly sensitive in its response to hypoxia. Other sequelae recorded include myocardial necrosis

and pneumonia. Also changes in the peripheral nerves or even paralysis have been reported. Similar to the central manifestations, they are in part caused by primary vascular damage.

The German MAK (maximale Arbeitsplatzkonzentration – maximum admissible concentration at the workplace) has been set at 30 mL/m³ (33 mg/m³), and the BAT (Biologischer Arbeitsstofftoleranzwert – biological threshold limit value), at 5 % COHb in whole blood at the end of the exposure period.

Evaluation of the cases described:
On the basis of the information given on the temporal relationship between carbon monoxide inhalation and the occurrence of manifestations including the detection of clearly elevated blood levels, a causal relationship has been confirmed.

3.4.6.3 Nitrous gases

Reaction of nitric acid with metal, delayed type lung oedema
A 69-year old female had stored a bottle of nitric acid in her bathroom. When the bottle broke, its contents spread over the floor and came into contact with a metal shelf. The reaction between metal and nitric acid resulted in a formation of nitrogen oxides, which were inhaled by the patient while she was wiping up the liquid. She only could inform her relatives over the telephone. The patient was sitting in her living room with severe dyspnoea when found by her daughter. Later, she had to be administered artificial respiration. After 12 days of intensive therapy she could be transferred to her regional hospital.

Manifestations / course
Findings made by the emergency physician included cyanosis, a rattling respiration and an oxygen saturation level of 78 %. In addition, hypertension up to 220 mmHg was found. Initial treatment included administration of glycerol trinitrate and furosemide. Oxygen supply resulted in an improvement of the patient's condition. After the toxic origin of the lung oedema had been confirmed by the daughter, inhalation and systemic administration of corticosteroids were initiated already in the ambulance.

On the next morning, i.e. 18 hours after the accident had happened, the patient required artificial respiration due to lung oedema of delayed type. In the further course, placing of a central venous catheter into her right jugular vein was followed by pneumothorax requiring intercostal drainage. Under catecholamine treatment, the patient's circulation was stable.

Due to her severe pulmonary problems, the patient was transferred to a specialized hospital with drained pneumothorax and in a reduced general condition. The respiratory situation rapidly improved under administration of high doses of corticosteroids. In the further course, the patient developed peak blood pressure values of more than 300 mmHg followed by rapid falls in blood pressure. This was followed by signs of hemiparesis on the left side of her body. Computer tomography confirmed ischaemia from deficient perfusion in the context of a circulatory shock.

When her respiratory function had improved, the patient could be extubated after eight days. On the following day, a sudden fall in oxygen saturation was observed. The cause was a total atelectasis of the left lung due to a mucous plug, which was removed by bronchoscopy after fiberoptic intubation. The intercostal drainage could also be removed on the 9th day of treatment with the right lung being unfolded.

Abnormal findings made in the course of the illness by clinical-chemical analysis included

leukocytosis (27 000/µL; reference range: 4 300–10 000/µL), CRP up to 11.0 mg/dL (reference value: below 5 mg/L), an increase in CK up to a maximum of 980 U/L (reference range: 10–70 U/L), and creatinine levels of up to 3.7 mg/dL (reference value below 1.36 mg/dL). After a total of 12 days of intensive therapy, the patient could be transferred to her regional hospital.

Notes
Nitrous gases will always form when concentrated nitric acid comes into contact with metals or organic substances such as coal, wood, paper etc. They are volatile mixtures of several nitrogen oxides, mainly nitrogen dioxide and nitrogen monoxide.

Due to their extremely high lipid solubility, they may penetrate into the bronchi and alveoli where damage is caused by formation of nitric and nitrous acids. Concentrations of 110–270 ppm will cause toxic lung oedema. At such concentrations, a latency period of one to four hours has been described, and at lower concentrations, of eight to 24 hours. However, also foudroyant courses characterized by almost uncontrollable lung oedema and lethal outcomes within 24 hours have become known. Also symptom-free patients should be medically observed over six weeks after exposure because manifestations of obliterating bronchiolitis may develop several days or even weeks.

From the therapeutic aspect, oxygen supply is the most important measure. A central role has also been attributed to the administration of corticosteroids.

Evaluation of the case described
Based on the information given on the temporal relationship between exposure by inhalation and the occurrence of manifestations including severe lung oedema, and in the absence of other causes, a causal relationship has been rated as probable in the case described above.

3.4.6.4 Industrial cleanser

Occupational accident involving an industrial cleansing product with health impairment persisting for years
In the context of his work as a window and building cleaner at a cleaning company, a 46-year-old worker had used up 6–8 L of an industrial cleansing product within 6 hours. Situated under a domed glass roof, he had applied the undiluted cleaning product above his head by means of an industrial sprayer without taking appropriate protective measures, wearing unsuitable gloves and no protective mask. Temperatures prevailing in the dome were about 25–30°C, and there was no ventilation. This resulted in a considerable exposure via the inhalational, dermal and oral routes. Components of the industrial cleansing product included butoxyethanol, ethanolamine and isopropanol, plus other substances. Since the occupational accident, the patient has been suffering from impairment of his mental and physical ability persisting even after four years.

Obviously, occupational safety rules were not sufficiently complied with: Only later during the work and on his request, the worker was provided with a protective suit and gloves. No protective mask was worn. Altogether, the exposure is assumed to have been higher than that of the limit concentrations for ambient air at the workplace.

Manifestations / course
The patient stated that due to dyspnoea, he had to work with his mouth open. This had resulted in dripping of the cleaning fluid onto his face and into his mouth. He had also suffered from vertigo. After termination of the work, he suffered from severe headache, which persisted for years with varying pro-

nouncedness. In addition, he stated to have been feeling tired and exhausted. He stated to have been suffering from a pronounced feeling of illness. He had felt obliged to continue his work and therefore went to work for another four weeks, which took him great effort. Then, he suffered a breakdown associated with very high temperatures above 40°C, shivers and convulsive abdominal pain. In the meantime, his headache had increased and at times been unbearable. He was admitted to hospital where a diagnosis of acute hepatitis was established. Transaminases had increased considerably. Pathogenic agents were excluded by appropriate detection methods. The tentative diagnosis of a chemically induced hepatopathy due to the cleansing product was made. Subsequently, the patient developed a transitory psychotic syndrome associated with mnestic and concentration disorders, and an increase of tinnitus he had been suffering from before. Against his unbearable headache, the patient had to be administered morphium at times. One year after the incident, he suffered a posterior myocardial infarction. Since that time, he suffered from an increased tinnitus on both sides.

Compared with former times when he had been symptom-free, the patient now experienced a most severe impairment of all his receptive and creative abilities. About 1.5 years after the incident, a psychological expert opinion reported severe concentration disorders, pronounced sleep disorders and aggressive as well as autoaggressive behaviour substantially caused by tinnitus. This was followed by most distressing depressive reactions.

A medical expert opinion made after four years reported the following conditions treated in the patient:
Chronic cephalgia associated with a condition after health damage due to solvents, in addition, coronary heart disease associated with a condition after posterior myocardial infarction, and tinnitus. The consequence stated was a persisting restriction of the patient's ability to perform moderately strenuous work. Four years after the incident, the patient is employed as a porter. He has stated to feel completely exhausted after his working day.

Notes
The product applied in the above case was a basic floor cleanser containing corrosive components such as potassium hydroxide (ca. 3 %). Solvents contained included, among others, ca. 20 % ethylene glycol monobutyl ether, ca. 10 % monoethanolamine and ca. 6 % isopropanol. The product has been labelled as "corrosive". Safety precautions include the wearing of appropriate protective clothing and protective gloves (made e.g. of butyl or fluorine rubber, unsuitable materials include natural rubber, natural latex and PVC) and safety goggles/face protection. During the work, sufficient ventilation must be provided. In exceptional situations such as accidental release of substances or air concentrations exceeding the limit concentrations of the substances in workplace air (ethylene glycol monobutyl ether: 20 mL/m^3, monoethanolamine: 2 mL/m^3), wearing of a respiratory protection mask is required. At elevated temperatures, vapours may be released in quantities high enough to form an explosive mixture with air.

The main routes of exposure to these solvents are the respiratory tract and the skin. The substances are assumed to be readily absorbed. Due to the extremely high blood/air distribution coefficient, the absorption rate will depend in particular on the alveolar ventilation. Hence, physical strain, as in the above case, will result in a strong increase of the quantity absorbed. A damp and warm indoor climate as described will also enhance dermal absorption. The absorption rate through the gastrointestinal tract

appears to be limited. For all three routes of exposure, no data on humans are available, but only data from animal studies. In addition to irritating effects on the skin and mucosa and on the respiratory tract culminating in toxic lung oedema, depending on the concentration, manifestations described after exposure to high concentrations also include disturbances of the central nervous system associated with neurovegetative disorders such as restlessness, hyperreflexia and vomiting. Manifestations observed in volunteers after eight hours of inhalational exposure to 100 and 19 ppm ethylene glycol monobutyl ether included acute irritating effects on the mucous membranes and effects on the CNS associated with nausea and headache. In a single human case, a hepatotoxic effect was observed after high inhalational exposure to monoethanolamine. Based on the results of animal studies, hepatotoxic effects have also been rated as probable.

Evaluation of the case described
Based on the information given on the temporal relationship between the exposure and the development of manifestations of a moderate health impairment, and in the absence of other causes, a causal relationship has been rated as confirmed in the case described above.

3.4.7 Surfactants

Death after ingestion of surfactants: A particular risk for patients suffering from dementia
In a state of mental confusion, a 79-year-old male ingested ca. 200 mL of a detergent containing surfactants at his home. Despite cardiopulmonary resuscitation (CPR), the patient died at the hospital within 60 minutes, from lung oedema.

Manifestations / course
The mentally disabled patient had been staying at home alone during daytime. He had been attended once a day by a home care service and in the mornings and evenings, by his relatives. He was found in the evening on his couch by his son, who noticed a “strange manner of speaking”. The open bottle of the detergent and a cup with remains of the detergent were found on the floor. The quantity ingested amounted to about 200 mL. On his immediate admission to hospital, the patient was awake but did not show any adequate reaction. He suffered from pronounced dyspnoea and bubbling crepitation over the lung fields. Respiratory and circulatory arrest set in within ten minutes. The patient died in spite of CPR including suction, intubation and adrenaline (epinephrine) administration. No post-mortem was performed.

Notes
Measures taken so far: On account of a considerable number of accidents involving surfactant-containing products as well as other household products and disinfectants, the predecessor institution of the BfR, the Federal Institute for Health Protection of Consumers and Veterinary Medicine (BgVV) conducted an expert hearing involving representatives of nursing and consumers’ associations, of the industry and the Poison Control Centres already in November 2001. It was the aim of the hearing to point out the background of such accidents and to develop strategies for their prevention.

In its Annual Report (Cases of Poisoning Reported by Physicians in 2001), the BgVV therefore placed special emphasis on the problem of accidental ingestion by elderly and disoriented persons. In parallel, a corresponding press release was issued (BgVV- Pressedienst 11/2002). In the context of the press release, 12 000 information leaflets in the German, Turkish, Russian, Serbian/Croatian and Polish languages were distributed to hospitals and chronic care institutions.

Since 1995, the number of cases of severe health impairment after ingestion of products containing surfactants in the age groups over 65 years has increased to 15 including as many as 13 deaths. In 2003 and 2004, however, no severe cases were reported to the BfR, possibly as a consequence of appropriate instructions given to nursing and cleaning staff.

Evaluation of the case described

On the basis of the information given on the temporal relationship between the accidental ingestion and the occurrence of manifestations, and in the absence of other causes, a causal relationship has to be assumed in the case described.

4 Annex

4.1 Spectrum of cases reported in the period 1 January – 31 December 2005

Table 12: 5 045 reports vs. degree of severity of health disturbances, classified by children and adults, with the adult cases differentiated by exposure in the private sphere and the working environment (except for cases with a causal relationship rated as "absent")

Incriminated products/uses	Reports, total numbers					Health impairment moderate/severe				
First level Second level Third level	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*
I. Medicinal products	117	25	91	15	76	5	1	4	4	
II. Veterinary medicinal products	7	1	6	1	5					
III. Chemical products	2668	111	2556	26	2530	144	28	116	12	104
Wastes, solid	19		19		19					
Waste gases	211		211	2	209	5		5	1	4
Sewage	14		14		14	1		1		1
Paints and related materials	204	1	203	2	201	6		6		6
Paint removers / strippers	15		15		15					
Alkyd resin paints										
Emulsion paints	2		2		2					
Artist's painting materials										
Glossy paints	48		48		48	4		4		4
Parquetry sealers	1		1		1					
Pigments										
Primers	9		9		9	1		1		1
Radioisotopes, radionuclides	1		1		1					
Paint thinners	75	1	74		74	1		1		1
Fire lighting products	25	22	2	2		8	8			
Building materials, auxiliary products	21		21		21	1		1		1
Building materials	157		157		157	14		14		14
Fuels, solid; auxiliary products	1		1		1					
Fuels, liquid	83	46	37		37	19	19			
Petrol	22		22		22					
Ethanol for technical use	4		4		4					
Lamp oil	46	46				19	19			
Fuels, gaseous	2		2		2					
Office materials, chemical	49	1	48	2	46	22		22	1	21
Decoration materials	2		2	1	1	1		1	1	
Dental materials	40		40	3	37	1		1		1

Incriminated products/uses	Reports, total numbers					Health impairment moderate/severe				
First level Second level Third level	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*
Disinfectants/sterilizers	349		349		349	8		8		8
Deodorants for technical use	7	6	1		1					
Diagnostic agents/reagents										
Printing, auxiliary products	1		1		1					
De-icing products	1		1		1					
Fire extinguishing media	9		9		9					
Flame retardants										
Galvanic cells	104	1	103		103					
Dry cells	7		7		7					
Button batteries	1	1								
Accumulators	96		96		96					
Galvanizing agents	3		3		3					
Galvanizing agents, auxiliary products	1		1		1					
Gases for technical use	1		1		1					
Antifreezes	6	3	3	1	2	1		1	1	
Glass-working, auxiliary products										
Rubber, production materials										
Semiconductors, production materials										
Household auxiliary products, chemical-technical										
Hydraulic fluids	34		34		34					
Refrigerants	4		4		4					
Ceramics, auxiliary products	3		3		3					
Ceramic materials										
Glues	93	1	92	1	91	2	1	1		1
Coolants	18		18		18					
Plastics, starting materials	21		21		21	1		1		1
Plastics, formulating materials	2		2		2					
Leather processing products										
Luminophors										
Solvents for technical use	83	2	81	3	78	4		4		4
Soldering and welding products (except welding fumes)	11		11		11					
Measuring equipment, chemical-technical										

Incriminated products/uses	Reports, total numbers					Health impairment moderate/severe				
First level Second level Third level	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*
Heating meters										
Mercury thermometers										
Thermometer fluids										
Metallurgy, auxiliary products	16		16		16	2		2		2
Dairy, auxiliary products										
Paper-making, auxiliary products	1		1		1					
Photography, auxiliary products	14		14		14					
Cleansing products	834	25	809	8	801	39		39	6	33
Drain cleanser	14	1	13	1	12	2		2	1	1
All-purpose cleansers	55	2	53		53	1		1		1
Oven and grill cleansers	19		19	1	18	1		1	1	
Cleansers for electronic products										
Descaling products	33	1	32		32	3		3		3
Front wall and stone cleansers	4		4		4					
Stain removers	3	2	1		1					
Floor polishes	3		3	1	2	1		1		1
Dishwashing products, manual	8	2	6		6					
Dishwashing products, machine	17	2	15	1	14	1		1	1	
Dishwasher cleansers	9		9		9					
Glass cleansers	6		6		6	1		1		1
Industrial cleansers	80		80	1	79	6		6		6
Rinsing additives for dishwashers	8	2	6		6					
Plastic cleansers	4		4		4					
Glossy oaint cleansers										
Milking machine cleansers	31		31		31	2		2		2
Metal cleansers	21		21		21	1		1		1
Furniture polishes	4	3	1		1					
Soot removers										
Lavatory cleansers	37	4	33		33					
Shoe and leather cleansers										

Incriminated products/uses	Reports, total numbers					Health impairment moderate/severe				
First level Second level Third level	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*
Carpet/upholstery cleansers	1		1	1		1		1	1	
Detergents	15	1	14	1	13	2		2	1	1
Detergents, auxiliary products	5	3	2		2	1		1		1
Joke articles	1		1		1					
Lubricants	24	1	23		23					
Welding fumes	38		38		38	2		2		2
Dust-laying oils										
Toys										
Textile, auxiliary products	2	1	1	1		1		1	1	
Propellants/sprays	2		2		2					
Water treatment products	5		5		5					
Pet shop products	2		2		2					
IV. Cosmetics / personal hygiene products	65	1	64	16	48	3		3	2	1
Hair care products	30		30	12	18	1		1	1	
Permanent wave products	5		5		5					
Depilatory products	11		11	11						
Hair dyes / colorants	9		9		9					
Hair conditioners	4		4		4					
Hair tonics	1		1	1		1		1	1	
Shampoos										
Skin care products	28	1	27	3	24	2		2	1	1
Bath oils/ salts										
Tanning products										
Creams / ointments	7		7	3	4	1		1	1	
Deodorants										
Face tonics										
Make-up products										
Oils										
Perfumes / after shaves	5	1	4		4	1		1		1
Powder					9					
Soaps	15		15		15					
Sun blockers										
Dental products	5		5	1	4					
Nail care products	2		2		2					
V. Pesticides	121	5	116	12	104	11		11	8	3
Acaricides										

Incriminated products/uses	Reports, total numbers					Health impairment moderate/severe				
First level Second level Third level	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*	*Total*	*Chil-dren*	*A-dults*	*Home*	*Work*
Fungicides	15		15	1	14					
Herbicides	15	1	14	1	13	2		2	1	1
Wood preservatives	4		4	1	3	2		2	1	1
Insecticides	42	2	40	7	33	7		7	5	2
Carbamates	1	1								
Chlorinated hydrocarbons	4		4	2	2	3		3	2	1
Phosphoric esters	16	1	15	2	13	2		2	1	1
Pyrethroids	12		12	3	9	2		2	2	
Molluscicides	1		1		1					
Repellents	2	2								
Rodenticides	2		2	1	1	1		1	1	
Anticoagulants										
Phosphates										
Seed dressings	3		3		3					
VI. VI. Agrochemicals (other than pesticides)	25		25	3	22	4		4	2	2
Fertilizers	18		18	2	16	1		1	1	
Plant care products	2		2	1	1	2		2	1	1
Growth regulators										
VII. Narcotic drugs	1		1		1					
VIII. Plants	27	18	9	1	8					
IX. Fungi	19	1	17	17		11		11	11	
X. Animals	4		4	1	3					
XI. Foods and beverages	124	5	99	67	32	8	1	7	5	2
Alcoholic beverages	12		12	2	10	1		1	1	
Food additives										
Tobacco and tobacco products	2	2								
Food supplements	20		20	20		2		2	2	
XII. Warfare and anti-riot agents	8		8		8					
Pyrotechnic products	1		1		1					
Tear gas	2		2		2					
XIII. Miscellaneous	83	2	81	2	79	5		5		5
Textiles	18		18	1	17	1		1		1
Clothing	14		14		14	1		1		1
Furnishing fabrics	1		1		1					
XIV. Primary substances	1830	4	1826	16	1810	114	1	113	7	106
XV. Industrial accidents	8		8		8	1		1		1

4.2 Notification form

Bundesinstitut für Risikobewertung
Dokumentations- und Bewertungsstelle
für Vergiftungen
Postfach 33 00 13

14191 Berlin

Stempel, Telefon-Nummer und Unterschrift der/des Ärztin/Arztes

Mitteilung bei Vergiftungen
nach § 16e Abs. 2 des Chemikaliengesetzes
(Telefon: 01888-412-3460, Fax: 01888-412-3929. E-Mail: giftdok@bfr.bund.de

1. Angaben zur/zum Patientin/en:

Alter: Jahre ☐ Monate (bei Kindern unter 3 Jahren) ☐ ☐ männlich ☐ weiblich Schwangerschaft (**freiwillig** auszufüllen) ☐ ja ☐ nein

2. Vergiftung ☐ **Verdacht** ☐

Unbedingt Handelsname der Zubereitung/des Biozid-Produktes oder Stoffname, aufgenommene Menge und Hersteller (Vertreiber); ggf. vermutete Ursache

a.

b.

c.

3. Exposition ☐ akut ☐ chronisch ☐ oral ☐ inhalativ ☐ Haut ☐ Auge sonstiges, welche ☐

Art der Vergiftung:	☐ akzidentell (Unfall)	☐ gewerblich	☐ Verwechslung	
	☐ suizidale Handlung	☐ Abusus	☐ Umwelt	☐ Sonstiges
Ort:	☐ Arbeitsplatz	☐ im Haus	☐ Schule	
	☐ Kindergarten	☐ im Freien	☐ Sonstiges	
Labor-Nachweis:	☐ ja	☐ nein		
Behandlung:	☐ keine	☐ ambulant	☐ stationär	
Verlauf:	☐ nicht bekannt	☐ vollständige Heilung	☐ Defektheilung	☐ Tod
	☐ Spätschäden (nicht auszuschließen)			

(**freiwillig** auszufüllen)

4. Symptome, Verlauf – stichwortartig – (ggf. anonymisierte Befunde, Epikrise beilegen)

4.3 List of Poison Control Centres

Berlin	BBGes – Giftnotruf Berlin Institut für Toxikologie Klinische Toxikologie und Giftnotruf Berlin	Oranienburger Str. 285	D-13437 Berlin	Phone: +49-30-19240 Fax: +49-30 68 67 21 E-Mail@giftnotruf.de www.giftnotruf.de
Berlin (ending 31 December 2006)	Charité – Universitätsmedizin Berlin Campus Virchow Klinikum Klinik für Nephrologie und internistische Intensivmedizin, Giftinformation	Augustenburger Platz 1	D-13353 Berlin	Phone: +49-30-4 50 65 35 55 Fax: +49-30-4 50 55 39 15 giftinfo@charite.de www.charite.de/rv/nephro
Bonn	Informationszentrale gegen Vergiftungen Zentrum für Kinderheilkunde Universitätsklinikum Bonn	Adenauerallee 119	D-53113 Bonn	Phone: +49-2 28-19240 Fax: +49-2 28-2 87 33 14 GIZBN@ukb.uni-bonn.de www.meb.uni-bonn.de/giftzentrale
Erfurt	Gemeinsames Giftinformationszentrum der Länder Mecklenburg-Vorpommern, Sachsen, Sachsen-Anhalt und Thüringen	Nordhäuser Str. 74	D-99089 Erfurt	Phone: +49-3 61-73 07 30 Fax: +49-3 61-7 30 73 17 Info@ggiz-erfurt.de www.ggiz-erfurt.de
Freiburg	Zentrum für Kinderheilkunde und Jugendmedizin Vergiftungs-Informations-Zentrale	Mathildenstr. 1	D-79106 Freiburg	Phone: +49-7 61-19240 Fax: +49-7 61-2 70 44 57 giftinfo@kikli.ukl.uni-freiburg.de www.giftberatung.de
Göttingen	Giftinformationszentrum-Nord der Länder Bremen, Hamburg, Niedersachsen und Schleswig-Holstein (GIZ-Nord) Universität Göttingen – Bereich Humanmedizin	Robert Koch-Str. 40	D-37075 Göttingen	Phone: +49-5 51-19240 Fax: +49-5 51-3 83 18 81 giznord@giz-nord.de www.Giz-Nord.de
Homburg	Informations- und Beratungszentrum für Vergiftungsfälle Klinik für Kinder- und Jugendmedizin		D-66421 Homburg/Saar	Phone: +49-68 41-19240 +49-6841-1628314 Fax: +49-6841-1628438 kigift@uniklinikum-saarland.de www.uniklinikum-saarland.de/de/einrichtungen/andere/giftzentrale

Mainz	Klinische Toxikologie und Beratungsstelle bei Vergiftungen der Länder Rheinland-Pfalz und Hessen Universitätsklinikum	Langenbeckstr. 1	D-55131 Mainz	Phone: +49-6131-19240 +49-6131-232466 Fax: +49-6131-232469 +49-6131-176605 giftinfo@giftinfo.uni-mainz.de www.giftinfo.uni-mainz.de
Munich	Giftnotruf München Toxikologische Abteilung der II. Medizinischen Klinik und Poliklinik, rechts der Isar der Technischen Universität München	Ismaninger Str. 22	D-81675 München	Phone: +49-89-19240 tox@lrz.tu-muenchen.de www.toxinfo.org
Nuremberg	Giftnotrufzentrale Nürnberg Medizinische Klinik 2, Klinikum Nürnberg Lehrstuhl Innere Medizin-Gerontologie, Universität Erlangen-Nürnberg	Prof.-Ernst-Nathan-Str. 1	D-90419 Nürnberg	Phone: +49-911-398 2665 Phone: +49-911-398 2192 muehlberg@klinikum-nuernberg.de www.giftinformation.de Giftnotruf: +49-911-398245 1 oder +49-911-398266 5

4.4 Press releases on toxicological problems issued by the BfR in 2005

Exercise care to prevent intoxications on the home and hobby sphere
Physicians' notifications of accidents involving chemicals provide important information on risk protection
07/2005, 21 February 2005

Heavy metals in ceramic glaze can pose a risk to health
BfR recommends lower maximum levels for the migration of lead and cadmium from ceramic objects to foods
08/2005, 11 March 2005

Risk of mix-up with Bear's garlic
BfR warns pickers about fatal consequences of mistaking free-growing poisonous plants for bear's garlic
10/2005, 15 April 2005

Bouncing playdough contains high levels of boric acid
Boric acid level in toys must be minimised
11/2005, 19 April 2005

Poppy seed for baking is not a soporific for infants
BfR issues a warning about serious damage to health
12/2005, 29 April 2005

Can toners damage health?
BfR examines possible links in a pilot study
13/2005, 06 May 2005

BfR warns against intoxication incidents involving liquid grill lighters
Small children are particularly at risk
15/2005, 24 May 2005

Poisonous plants and inedible mushrooms
Updated version of popular BfR information leaflets now available
16/2005, 03 June 2005

Less poison in planes
New airplane disinsection method is easier on passengers and crew
23/2005, 05 July 2005

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bfr@bfr.bund.de