Human Toxicol. (1987), **6**, 19–29

The Epidemiology and Prevention of Paraquat Poisoning

Lesley J. Onyon and Glyn N. Volans

National Poisons Information Service, The Poisons Unit, Guy's Hospital, London SE1 9RT

1 In the UK there was an increase in the annual number of deaths associated with paraquat poisoning between 1966 and 1975. Since that time there has been little change in numbers.
2 High mortality is associated commonly with suicidal intent. Serious accidental poisoning from paraquat has never been frequent in the UK and there have been no deaths reported in children since 1977.
3 The National Poisons Information Service has monitored in detail all reports of paraquat poisoning since 1980. Of the 1074 cases recorded there were 209 deaths. In recent years serious poisoning has been more commonly associated with ingestion of concentrated products by males. Local exposure to paraquat has not resulted in systemic poisoning.
4 International data for paraquat poisoning is incomplete and difficult to compare. There is a scarcity of morbidity data at both international and national levels. Information obtained from Poison Control Centres indicates that paraquat poisoning occurs in many countries but detailed comparisons are hindered by lack of standardised methods of recording.
5 Various measures to prevent paraquat poisoning have been introduced. Their effectiveness has not been studied in detail. Some support is provided by the low incidence of serious accidental paraquat poisoning in the UK, but because of the suicidal nature of paraquat poisoning it is unlikely that current preventative measures will influence the number of deaths occurring each year.
6 Preventative measures against paraquat poisoning should be tailored to national needs, based on and assessed by epidemiological studies.

Introduction

The preceding papers have stressed both the importance of paraquat to agriculture, and its safety when correctly used. Paraquat is, nevertheless, toxic to man and this toxicity has been the subject of a great deal of attention in the scientific and 'lay' press. The first cases of paraquat poisoning occurred in 1964, in Ireland and New Zealand (Bullivant, 1966) and by 1970 some 600 fatalities had been reported in the world literature (IPCS, 1984). In spite of the interest, it remains a difficult task to describe the mortality and morbidity associated with paraquat poisoning in different countries in strict epidemiological terms. Such a description requires appropriate and comparable mortality and morbidity statistics. A recent review found that 'because of the different requirements or practices for notification or reporting of cases of poisoning in the many countries in which paraquat is used, the magnitude of the problem is difficult, if not impossible, to determine' (IPCS, 1984). We concur with this view after reviewing the information available from Poison Control Centres, routine sources of mortality and morbidity data and from scientific reports. The information available from these sources is presented and the measures aimed at preventing paraquat poisoning reviewed.

Sources of information

1. Hospital based surveys

The Home Accident Surveillance Scheme (HASS) records standardised data from a sample of twenty Accident and Emergency (A&E) departments in England and Wales on all types of home accidents (Consumer Safety Unit). A request was made for the number of admissions due to home accidents with weedkillers occurring during the period October 1982 to October 1984. In addition the results of an epidemiological study of acute poisoning cases attending twenty one A&E departments in England and Wales were examined (Murray, Francis & Thompson 1986).

2. Poison Control Centre reports

These reports include the results of a five-year surveillance of paraquat poisoning undertaken by the National Poisons Information Service (NPIS) and the

manufacturer (ICI plc) since 1980. Details of the methods used have been previously published (Hart & Bramley, 1983; Whitehead, Volans & Hart, 1984). In addition the results of a survey amongst European Poison Control Centres of the incidence of paraquat poisoning, undertaken by the secretary of the European Association of Poison Control Centres (EAPCC) (Wickstrøm, 1984) have been examined together with other information from Poison Control Centres given directly to the authors.

3. Mortality statistics

Statistics for England and Wales published in the *Pharmaceutical Journal* for 1966–1973 and by the Office of Population Censuses and Surveys (OPCS) for 1974–1984 have been used. Data was not published in 1981 in England and Wales due to industrial action.

For Scotland, statistics were obtained from the Annual Report of the Registrar General from 1967 to 1984.

4. Other sources

Information from the Agrochemical Poisoning Appraisal Panel (APAP) which is administered by the Health and Safety Executive to investigate reports of occupationally related pesticide poisonings together with information published in the scientific press has been analysed.

Morbidity from paraquat poisoning

1. Hospital based surveys

From October 1982 to October 1984, over 287,000 home accidents were reported to HASS. Of the 39 which were due to weedkillers only seven could be identified as containing paraquat. Four of these involved children under 2 years of age, but only one was admitted into hospital. Limitations in the HASS reporting system, for example fatalities not being included and trade names not always being recorded, may mean that this number of cases is an underestimate.

A preliminary report showed that over a period of one year 22 195 cases of acute poisoning attended A&E departments in England and Wales. Only 14 cases of exposure to paraquat were recorded (Murray *et al.*, 1986). The number of admissions to the hospitals in the study represent approximately 12.5% of all admissions in England and Wales. The total number of exposures due to paraquat may therefore be estimated at 112 but final figures are expected to be higher (J. Francis, personal communication).

A retrospective study of cases of self-poisoning presenting at the United Norwich Hospitals during the five-year period 1978–1982 found twelve admissions due to paraquat (Adams, 1986).

An important source of morbidity data, the Hospital-In-Patient enquiry (HIPE), fails to document admissions due to paraquat. This is because the International Classification of Diseases (ICD) used for coding the cause of admission has no specific code for paraquat. Any information would be contained in a general category, such as admission due to toxic effects of 'other substances chiefly non-medicinal as to source'.

Outside the UK we found no published national morbidity information concerning paraquat. A national study conducted by the Environmental Protection Agency in the United States concerning hospitalised pesticide poisonings failed to document any cases due to paraquat, even though 2954 admissions due to pesticides were reported in 1974 (G.R.A. and I., 1981).

We therefore concur with a recent report that there are no published national morbidity statistics for pesticides (Vale & Buckley 1986) and that this is particularly true for paraquat. For this reason Poison Control Centres have been identified as potential sources of 'morbidity data' (Brzezinski 1976; Volans & Wiseman, 1986).

2. Poison Control Centre (PCC) reports

(a) United Kingdom – National Poisons Information Service (NPIS). Over the period 1980 to 1985, more than 1000 cases of exposure to paraquat were reported to the NPIS (Table 1). 70% (760) of all cases involved ingestion. Other reported routes of exposure were inhalation (9.8%), skin contact (9.3%), eye contact (2.3%) and injection (0.7%). Of these routes only ingestion and injection led to symptoms of systemic poisoning, though one case of skin contact resulted in a positive urine test.

Of all the cases of ingestion 13% of patients were under five-years-old, 2% were aged between 5 and 12 years, and 85% were older than 12 years. Outcome of the incident was confirmed in 81% of cases (67% survival) by the attending physician, generally about four to five months after the incident. It was not possible to obtain complete follow-up because of difficulties in tracing patients who were not admitted into hospital.

The proportion of survivors having symptoms was estimated for 1984, when 74% of adults had symptoms whereas in the under 5 age group only one child (8.3%) had.

The Agrochemical Poisoning Appraisal Panel recorded three occupationally related paraquat poisonings in 1982.

Thus paraquat poisoning does not seem to represent a problem: to children, by skin and eye contact, inhalation, and through occupational contact.

Table 1 Paraquat cases notified to the NPIS over the years 1980–1985

Year	Total number of cases	Number of cases of ingestion	Age and outcome of ingestion cases: < 5 yrs S	D	NK	5 – 12 yrs S	D	NK	> 12 yrs S	D	NK	Number of deaths by ingestion	Total number of deaths
1980	152	121	11	–	3	–	–	1	54	37	15	37	37
1981	169	127	4	–	2	1	–	–	57	45	18	45	47
1982	223	154	16	–	5	–	–	1	78	38	16	38	41
1983	198	143	15	–	8	–	–	5	64	33	18	33	33
1984	189	132	12	–	9	3	–	1	58	32	17	32	32
1985*	143	83	9	–	8	1	–	1	26	19	19	19	19
Total	1074	760	67	–	35	5	–	9	337	204	103	204	209

S: Survival D: Death NK: Not known
* Provisional figures

Table 2 Cases of paraquat poisoning reported internationally

Country	Cases (fatalities in brackets) 1980	1981	1982	1983	1984	Source
UK	152 (37)	169 (47)	223 (41)	198 (33)	189 (32)	NPIS
England & Wales	(24)	NK	(31)	(36)	(31)	OPCS
Scotland	(6)	(12)	(9)	(5)	(9)	Registrar General
Denmark			(3 over 3 years)			Copenhagen PCC
France	90	106	83	NK	NK	Wickstrøm*
		27 (20)	–	–	–	Frelan 1983
Germany (West)			(8 from 1978–1983)			Wickstrøm*
Greece	25	49	65	85	80	Athens PCC
Eire	NK (13)	NK (4)	59 (9)	54 (14)	53 (7)	Dublin PCC
Netherlands	29	39	19	NK	NK	Wickstrøm*
Czechoslovakia			(2 to 3 per annum)			Wickstrøm*
Norway			(3 over 12 years)			Wickstrøm*
Poland	6 (3)	7 (0)	12 (10)	NK	NK	Wickstrøm*
Spain	NK	NK	7	NK	NK	Wickstrøm*
Sweden	1 (0)	2 (0)	0	0	0	Stockholm PCC
Switzerland	10 (1)	8 (2)	8 (1)	6 (3)	3 (2)	Zurich PCC
Israel	NK	(4)	(2)	(0)	(0)	Haifa PCC
Australia	NK	NK	11	8	10	Canberra PCC
Fiji	–	–	49 (30)	59 (33)	–	Groundar 1984
Japan					(1300)	Naito 1986
USA	NK	NK	NK	NK	153 (1)	AAPCC 1984

* Personal communication

(b) Other countries Cases of paraquat poisoning recorded over the period 1980–1984 are shown in Table 2. This shows that there are differences in the extent of paraquat poisoning with relatively little occurring in Sweden, Norway, Czechoslovakia and West Germany. However the comparability of these figures is not known because of the different methods of recording and following-up cases and nature of the services provided by the different countries. For example poison control centres in the United States accept enquiries from members of the public, unlike centres in the United Kingdom.

Mortality from paraquat poisoning

1. National mortality statistics

Figure 1 shows the number of fatalities recorded per year in England and Wales, Scotland and Eire for the period 1965–1984. In England and Wales, the first fatalities due to paraquat poisoning were recorded in 1966. Over the ten-year period to 1975, fatalities increased from 1 to 43 cases per annum with a rapid increase occurring between 1971 and 1975. Numbers since 1975—although fluctuating—have not changed significantly, 31 were recorded in 1984.

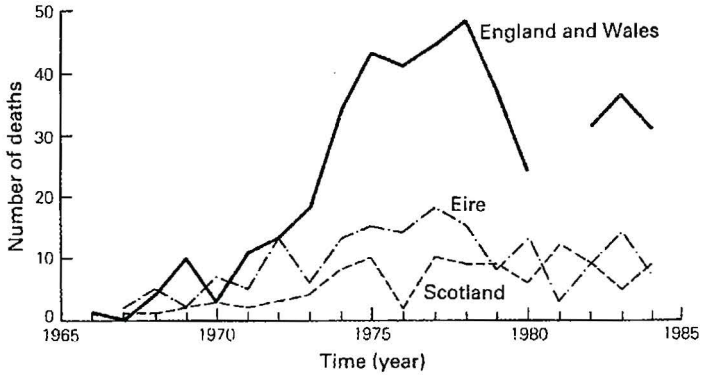

Figure 1 Deaths due to paraquat 1966–1984 for England and Wales, Scotland and Eire
Sources: England and Wales, Pharmaceutical Journal (1966–1973) OPCS DH4 Series (1974–1984).
Scotland, Annual Report of the Registrar General (1966– 1984).
Eire, Fitzgerald *et al.* 1978 (1967–1976). Dublin Poisons Unit (1977–1984).

For Scotland, the trend is similar to that for England and Wales. One case was recorded in 1967 rising to 10 cases in 1975, since when the numbers have remained relatively stable—9 were recorded in 1984.

For Eire, an increase from 1 to 13 fatalities per annum occurred between 1967 and 1972 but since then numbers have been fluctuating at 12 ± 4 per year.

A review of the status of paraquat poisoning in Eire between 1967 and 1976 (Fitzgerald, Barniville, Flanagan *et al.*, 1978) revealed that the mortality in terms of population was approximately seven and a half times that in Great Britain. Figure 2 shows the population-corrected incidence of paraquat poisoning in England and Wales, Scotland and Eire. It can be seen that the incidence of paraquat fatalities in Eire remains high, approximately four times that of England and Wales and twice that of Scotland.

Poisoning with solid and liquid substances in England and Wales remained fairly constant over the period 1973–1980 (Osselton, Blackmore. King *et al.*, 1984) but in recent years (1981–1983) a decline has occurred. Similar trends are seen for Scotland. Over these periods the proportion of deaths due to paraquat continued to increase. Thus, in England and Wales, paraquat has accounted for an average of 1.4 ± 0.2% of all poisonings with solid and liquid substances since 1975. Over the same period in Scotland it has accounted for an average of 2.3 ± 0.8% with a maximum of 3.78% being recorded in 1984.

There is thus no evidence to suggest that paraquat poisoning is becoming a less important cause of mortality in the UK.

Information on the intent of the poisoning is recorded at the inquest into the death and can either be 'suicidal', 'accidental' or 'not determined' whether suicidal or accidental. Figure 3 shows the total numbers of fatalities classified into these categories over the period 1966–1984 for England, Wales and Scotland. It can be seen that although the numbers recorded for suicidal intent follow very closely those of the total, numbers due to accidents remain below five per annum, with the exceptions of 1975 and 1976 (10 and 6 respectively). The number of not determined cases reached a maximum in 1977 (12 per annum) coincident with a decline in accidental deaths. However, the influence of this category on the number of accidental deaths remains difficult to interpret, and it is more likely that the number of suicidal deaths would be influenced by the 'not determined' category because of religious and other constraints in bringing in a verdict of suicide. It is clear from these figures that the rise in paraquat fatalities which occurred from the late 1960's to the early 1970's was due to an increase in the use of paraquat for suicide.

Of the 428 deaths recorded as due to paraquat, over the period 1966–1984 in England and Wales, 75% were male and 25% female. 77% of the suicidal deaths and 65% of accidental deaths over this period occurred in males. A similar sex distribution is found for the 105 recorded fatalities in Scotland; 74% of the total were male; 78% of suicides and 59% of accidental deaths were male.

Details regarding age of patient are not available from published mortality statistics which give broad age ranges. However, over the period 1968–1977 six paraquat fatalities occurred in British children under the age of ten; these were accidental in nature. For comparison over the same period 49 children (under

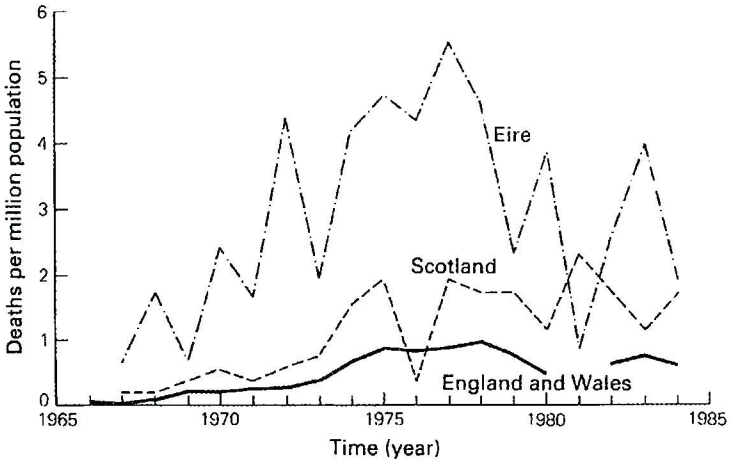

Figure 2 Deaths due to paraquat 1966–1984 per million population for England and Wales and Scotland
Sources: England and Wales, Pharmaceutical Journal (1966–1973). OPCS DH4 Series (1974–1984). Population Trends 42 1985.
Scotland, Annual Report of the Registrar General (1966–1984). Population Trends 42 1985.
Eire, Fitzgerald *et al.* 1978 (1967–1976). Report on Vital Statistics CSO Dublin 1982.

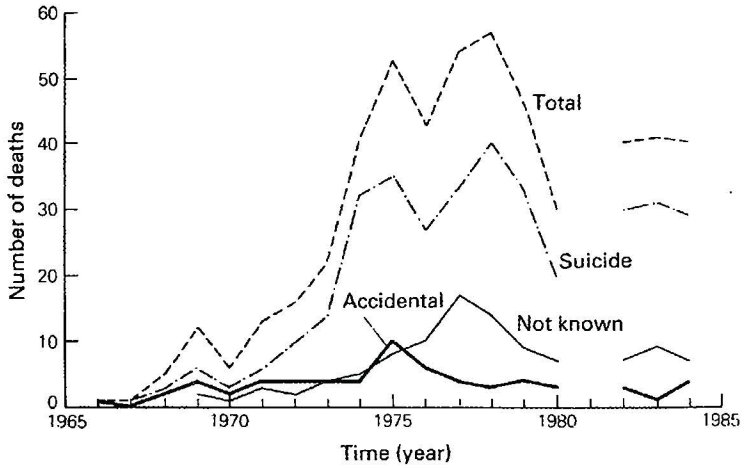

Figure 3 Deaths due to paraquat 1966–1983: Accidents vs suicide, for England, Wales and Scotland
Sources: England and Wales, Pharmaceutical Journal (1966–1973). OPCS DH4 Series (1974–1984).
Scotland, Annual Report of the Registrar General (1966– 1984).

ten) died as a result of accidental ingestion of tricyclic antidepressants (Frazer, 1980). Since 1977 there have been no reported deaths due to paraquat in this age group.

The Registrar General for Scotland was able to provide details of occupation in 79 of the 81 fatalities occurring from 1975 to 1984. Only in 13 cases (16%) was there any direct link between occupation and access to paraquat (e.g. farmer, market gardener, groundsman). In Eire it was found that a wide range of occupational types were involved in paraquat poisonings but that intentional poisoning was commonest among agrochemical workers (Fitzgerald *et al.* 1978).

2. Other countries

No national published mortality statistics listing paraquat were found. Data on mortality is published by the World Health Organisation, however only a broad categorisation of poisoning based on ICD codes is given, paraquat therefore is not listed. The same limitations were found with vital statistics available from individual countries.

Additional information regarding fatalities due to paraquat may be obtained from Poison Control Centres.

3. Poison Control Centre reports

a. United Kingdom—National Poisons Information Service. Of the 1074 cases of paraquat exposure reported to the NPIS, 209 cases proved fatal. There was a predominance of males (72%) and of deliberate intent (85%) involved in the fatalities (Table 3). The mean age for males was 44.6 (S.D. = 16.5) and 54.1 for females (S.D. = 14.2).

71% of fatalities involved concentrated liquid formulations (Table 3) and 22% granular formulations. There was a marked predominance of male fatalities involving the liquid formulations (78% male, 22% female) although there was no such difference with the granular products (54% male, 46% female).

Table 3 Type of product, intent and sex of patient involved in 209 fatalities reported to the NPIS (1980–1985)

	Accidental	*Deliberate*	*NK*
Liquid concentrate			
Male	4	100	12
Female	—	30	2
Total = 148			
Granular			
Male	3	21	1
Female	2	18	1
Total = 46			
NK			
Male	—	8	2
Female	1	1	3
Total = 15			
Totals: 209	10	178	21

Note: Liquid concentrate: Gramoxone, Dextrone, Gramonal, Cleensweep
Granular: Weedol, Pathclear

Of the 209 fatalities, 204 were due to ingestion of the product, two were due to injection, one intravenously and one intramuscularly and in three cases the route of exposure was not known.

Information was available as to the geographical distribution of 173 of the fatal cases. In absolute numbers most fatalities occurred in Greater London, West Midlands and Belfast. However, this could be due to the large populations and/or the presence of centres taking an interest in the treatment of paraquat poisoning. Figure 4 shows that, when corrected for population differences, there are disproportionately large numbers of fatalities (seven or more per million) occurring in Belfast, Devon, Cornwall, Norfolk, West Sussex and West Glamorgan—a pattern which probably reflects the scale of the agricultural industry in these areas.

Information regarding occupation was only available for 67 of the 209 fatalities. Of these 39% had occupations which gave ready access to concentrated paraquat products, the remaining occupations were varied, with no obvious relationship to agriculture.

There are differences between the number of fatalities reported to the NPIS and those recorded from death certificates in England, Wales and Scotland. Both sources of data contain an unknown degree of bias. The NPIS relies principally on voluntary reporting of cases, although since 1980 by following up cases reported to the manufacturer or in newspapers the surveillance has been more complete. Official mortality statistics rely on the correct diagnosis of the cause of death which has been shown to be inaccurate in many instances of poisoning (Vale, Buckley & Meredith, 1984). If the number of deaths reported to the NPIS are compared to those reported by the OPCS and Registrar General of Scotland, the differences are small, e.g. 41 compared with 40 in 1982, 33 compared with 41 in 1983 and 32 compared with 30 in 1984. The degree of overlap remains unknown, requiring comparison of death certificates with NPIS records, and has not been possible within the scope of this study. In Eire, mortality statistics over the period 1967–1976 were obtained from a study combining official mortality and PCC statistics (Fitzgerald *et al.*, 1978), whilst after 1976, statistics were obtained solely from the PCC. There does not seem to be any great jump in the mortality trend shown in Figure 1 so perhaps differences between PCC and official mortality statistics are in fact small.

b. Other countries Mortality data from a survey into the incidence of paraquat poisoning amongst members of the EAPCC is shown in Table 4 together with information from other PCC's and literature. There are wide differences from country to country in the annual numbers of deaths due to paraquat per year, ranging from 1300 in Japan to 1 in Denmark and zero in Sweden. There are also wide variations in the number of fatalities per million population, e.g. 0.004 per million (USA) and 47.0 per million (Fiji). The mortality ratios range from 74% in one French study (Frelon *et al.*, 1983), 58% (Fiji) and 52% (Poland) to 0.6% (USA) suggesting that the proportion of suicides and accidental exposures are different in

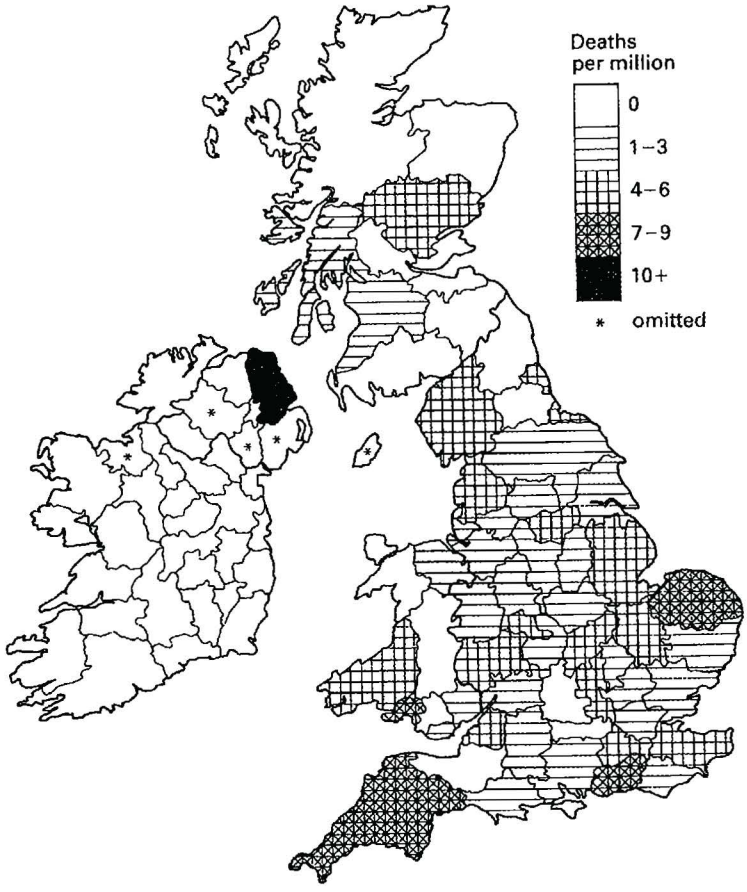

Figure 4 Deaths due to paraquat 1980–1985 (UK): Deaths per million population by county as reported to NPIS. (n = 173.)

different countries. Although the figures are not strictly compatible due to uncertainties with the populations covered, they do indicate that some countries have bigger problems with paraquat poisoning than others.

In France, paraquat poisoning has been monitored in detail by Poison Control Centres (Conso, 1979; Frelon, Merigot, Garnier *et al.*, 1983; Ethymiou, 1983). Paraquat was first marketed there in 1965, but fatalities were not recorded until 1973, rather later than in the UK. Accidental deaths declined in proportion to suicides over the period 1973 to 1977. All deaths resulted from ingestion. Ethymiou (1983) reported that although accidental and occupational poisoning represented 74% of all cases, suicidal poisoning was associated with the highest mortality, as were the more concentrated products. Over a three-year period only one child died. More cases were reported in rural areas. Hence the situation in France is very similar to the UK.

A series of reviews concerning poisoning cases in Malaysia (Amarasingham & Lee, 1969; Amarasingham & Hee, 1976 and Amarasingham & See unpublished data) found that paraquat has replaced arsenite as the most commonly consumed poison. From 1977 to 1981 paraquat was responsible for 31% of all poisoning cases, with 79% mortality. The incidence of males and females was similar and poisoning was predominantly due to its suicidal use by the poorer ethnic groups who presumably had greater access to the product. Accidental cases of poisoning occurred after the concentrated formulations were decanted locally into poorly labelled containers.

Table 4 Fatalities due to paraquat reported internationally (over the years 1980–1984)

Country	*Population (millions)*	*Deaths per annum (average)*	*Deaths per million population per annum*	*Cases per million population per annum*	*Mortality % (average)*	*Source*
UK	56.6	37.6	0.66	3.3	20	NPIS
England & Wales	49.6	30.3	0.61	–	–	OPCS
Scotland	5.2	8.2	1.6	–	–	Registrar General
Denmark	5.1	1.0	0.20	–	–	Copenhagen PCC
France	54.2	–	–	1.7	–	Wickstrøm*
		20.0	(0.36)	(0.5)	74	Frelan 1983
Germany (West)	61.6	1.3	0.02	–	–	Wickstrøm*
Greece	9.8	–	–	6.2	–	Athens
Eire	3.5	9.4	2.7	15.8	18	Dublin PCC
Netherlands	14.3	–	–	2.0	–	Wickstrøm*
Czechoslovakia	15.4	2.5	0.16	–	–	Wickstrøm*
Norway	4.1	0.25	0.06	–	–	Wickstrøm*
Poland	36.7	4.3	0.12	0.23	52	Wickstrøm*
Spain	37.9	–	–	0.9	–	Wickstrøm*
Sweden	8.3	–	–	0.07	–	Stockholm PCC
Switzerland	6.5	1.8	0.28	1.3	21	Wickstrøm*
Israel	4.1	1.5	0.37	–	–	Haifa PCC
Australia	15.4	–	–	0.6	–	Canberra PCC
Fiji	0.67	31.5	47.0	80.6	58	Groundar 1984
Japan	118.4	1300	11.0	–	–	Naito 1986
USA	232.0	1.0	0.004	0.7	0.6	AAPCC

* = Wickstrøm, E. personal communication

Population sources: Eurostat 'Basic Statistics of the Community' 1984
UK CSO 'Regional Trends' 1985

Prevention

A range of measures have been introduced or proposed for the prevention of paraquat poisoning.

1. Communication

Information concerning the toxicity of paraquat, correct usage and the dangers of inappropriate storage should be given to agricultural and domestic users. Product labelling is one way of communicating this information. The earliest product labels for paraquat gave no indication of its toxicity, but when the problem of poisoning became apparent appropriate changes were made and present day labels leave the user in no doubt about the need to handle the product with care. Labelling should be in an appropriate language with symbols carefully chosen to be meaningful to the user. For example in some parts of the world the snake is more meaningful as a hazard warning of poison than the skull and crossbones.

No matter how good the label, it cannot be assumed that the user will read it carefully. It is therefore important to use additional forms of communication; posters and booklets such as those produced by GIFAP (GIFAP 1983), appropriate audio visual aids and educational campaigns by the press and television regarding safe handling and storage. Media coverage of paraquat poisoning can have a detrimental effect; reports of individual cases, often sensationalised, may influence others to use paraquat as a means of suicide (Barraclough, Shephard & Jennings, 1977). Therefore restricting or controlling such publicity might help reduce the number of suicides using paraquat (Hayes, 1980).

2. Packaging

Restricting pack size is an obvious way to limit the dose likely to be ingested. Additionally the type of package can affect the accessibility of the product. The proposed Child Resistant Packaging Regulations will not require child resistant closures for paraquat containing products currently on sale in the UK since they will not apply to solids or products exclusively for use in agriculture [Child Resistant Packaging Regulations 1986 (Draft)]. It is unlikely that they would affect the incidence of serious poisoning in children since children do not ingest toxic amounts of

the domestic products or gain access to the commercial preparations in their original containers. Accidental poisoning with these products in adults and children normally occurs as a result of inappropriate decanting and labelling. Packaging changes are unlikely to deter the suicidal patient.

3. Formulation changes

Changes in the concentration of paraquat within a product will also limit the dose ingested. Thus the marketing of a 2.5% w/w granular formulation represents a reduction in hazard from the earlier 5% w/w formulation. It has been shown that the granular formulation is less of a hazard than the liquid concentration (Table 3) and it has been proposed in this respect that a diluted liquid concentrate, 10% w/v, should replace the 20% w/v product currently marketed.

Other formulation changes have involved the use of 'additives'. An unpleasant smelling 'stenching' agent was added to liquid formulations in 1975 and in 1981 a blue colour was added to liquid and solid paraquat products to serve as a warning.

In 1977 a centrally-acting emetic agent, codenamed PP796, was added to liquid formulations at a concentration of 0.05% w/v and to solid formulations at a concentration of 0.02% w/w. This concentration of emetic was calculated to cause vomiting if the minimum lethal dose was swallowed and in animal experiments such a concentration increased the lethal dose of paraquat by a factor of three to five (Rose, 1976). Recently (1985) the concentration of emetic in solid formulations has been doubled. Two authors have commented on the effectiveness of the emetic in reducing mortality in man. In France it was concluded that the emetic (identified in 14 cases, 11 of whom died) did not modify prognosis. In contrast preliminary findings of a study in the UK have found that there may be some reduction in mortality with emetic addition (A. P. Whitehead, personal communication). Emetic addition was not associated with any adverse effects (Denduyts-Whitehead, Hart & Volans, 1985). Even so the efficacy of the emetic at reducing mortality in man remains to be substantiated.

Another suggestion for prevention has been put forward as a result of the development of a novel formulation which forms a semi-solid mixture when small amounts of water are added, thus making it difficult to ingest large quantities (Naito & Yamashita, 1986).

4. Legislation

Legislation has restricted the availability of the commercial concentrate in many countries and in some countries a total ban has been applied (West Germany and Sweden). In the UK the Poisons Act of 1972 restricts the sale of concentrated formulations to 'persons engaged in the trade or business of agriculture, horticulture or forestry'. The sale of these concentrated products is further restricted by limiting the number of licenced dealers. In Eire, similar legislation was passed in 1968 and 1975. The effects of this legislation on the incidence of paraquat poisoning were studied by Fitzgerald *et al.* (1978) who found that there was a drop in the number of accidental poisonings, due to a decrease in the practice of decanting commercial products into household containers. There was no change in the number of suicides after this legislation was passed. Legislation may have the effect of increasing other forms of suicidal poisoning. It was following a ban of arsenite as a weedkiller in Malaysia in 1976 that paraquat poisoning became such a problem (Amarasingham & See, unpublished data). Those countries where paraquat is banned can be seen from Table 2 to have a very low incidence of paraquat poisoning. However such a severe course of action may not be appropriate for all countries and must take into account the agricultural importance of paraquat in that country.

Asked whether measures taken against paraquat poisoning had been effective, members of the European Association of Poison Control Centres (EAPCC) concluded that the addition of an emetic or staining agent had not had the desired effect and that strict regulations on the sale of the liquid concentrate did not seem to be wholly effective (Wickstrøm, personal communication).

Discussion

Comparisons of the incidence and severity of paraquat poisoning between different countries are severely limited by the lack of standardised methods of official data collection and recording. Nevertheless it is apparent that paraquat remains an important cause of mortality worldwide and there is little evidence that paraquat poisoning is decreasing in frequency.

There are differences in the incidence of paraquat poisoning amongst the countries studied. There are also regional differences within the UK, Northern Ireland and Scotland have higher incidences than England and Wales, and paraquat poisoning in Eire has always had a higher incidence than in the UK. In some countries paraquat has not so far presented a serious problem in spite of its widespread usage—for example, USA and Australia. In contrast Japan and a number of other countries, notably Fiji, are currently facing epidemics of paraquat poisoning far more severe than those seen in Europe.

Mortality from paraquat poisoning is closely related to suicidal intent; thus in the USA (mortality 0.6%) 88% of cases were accidental whilst in Fiji (mortality 58%), 66% had suicidal intent. Additionally the predominance of males amongst fatalities correlates well

with the known epidemiology of suicides (Weissman, 1974).

The increase in suicidal use of paraquat in the UK over the period 1966–1975, accounts for the rise in fatalities (Figure 3). Why there should have been this increase in so many countries is unknown. In the UK there was no proportionate increase in sales of the commercial product over the period when the rapid increase in fatalities occurred (T. B. Hart, personal communication). However the availability of the liquid concentrate remains an important factor. The wide range of occupations recorded amongst fatalities may mean that legislation restricting the availability of paraquat is not sufficient. Substitution and public awareness are additional factors which may influence the use of a particular product for suicide (Low *et al.*, 1981). Substitution has been shown to have had an effect in Malaysia where paraquat replaced arsenite poisoning but the influence of substitution in the UK is not known. Public awareness, increased by media reporting may be an important factor but remains difficult to investigate.

In many countries serious accidental and occupational poisoning and poisoning in children is rare. The nature of paraquat poisoning is largely suicidal and it is unlikely that current preventative measures will influence the number of deaths occurring each year. Few preventative measures have been monitored in such a way as to demonstrate their effectiveness.

On the basis of our experience in the UK, we believe that Poison Control Centres (PCCs) have an important role in monitoring the incidence and severity of poisoning and providing epidemiological data. PCCs are well placed then to develop schemes to evaluate preventative measures. Care must be taken when making direct comparisons between different PCCs because of the different populations covered and differences in the methods used to assess cases. There is currently much interest in the suggestion that PCCs should agree to standardise some aspects of data collection. We would tentatively suggest that since paraquat poisoning in Europe is widespread and involves relatively small numbers of a discrete type of poisoning it would form a useful model for international collaboration between PCCs.

The authors acknowledge the help of colleagues in the National Poisons Information Service and other Poison Control Centres and, in particular, the information provided by Dr E. Wickstrøm and Professor A. N. J. van Heijst on behalf of the European Association of Poison Control Centres. We should also like to thank John Gelder for his help with the illustrations.

References

ADAMS, R. H. M. (1986). An accident and emergency department's view of self-poisoning. A retrospective study from the United Norwich Hospitals: 1978–1982. *Human Toxicol.*, **5**, 5–10.

AMARASINGHAM, R. D. & LEE, H. (1969). A review of poisoning cases examined by the Department of Chemistry, Malaysia, from 1963 to 1967. *Med. J. Malaysia*, **XXIII** (3), 220–227.

AMARASINGHAM, R. D. & HEE, T. T. (1976). A review of poisoning cases examined by the Department of Chemistry, Malaysia, from 1968 to 1972. *Med. J. Malaysia*, **XXX** (3), 185–193.

AMARASINGHAM, R. D. & SEE, L. A. (unpublished report). A review of human poisoning cases examined by the Toxicology Division of the Department of Chemistry, Petaling Jaya, Malaysia, from 1977–1981.

BARRACLOUGH, B., SHEPHERD, D. & JENNINGS, C. (1977). Do newspaper reports of Coroners' inquests incite people to commit suicide? *Br. J. Psych.*, **131**, 528–532.

BRZEZINSKI, Z. J. (1977). Poisoning in the European Region. *Acta Pharmacol. Toxicol.*, **41** (Suppl 2), 470–484.

BULLIVANT, C. M. (1966). Accidental poisoning by paraquat. Report of 2 cases in man. *Br. Med. J.*, **1**, 1272–1273.

CHILD-RESISTANT PACKAGING (Safety Regulations) 1986 (Draft). Department of Trade and Industry, United Kingdom.

CONSO, F. (1979). Paraquat poisoning: experience of Poison Control Centers in France. *Vet. Hum. Toxicol.*, **21** Suppl, 112–113.

Consumer Safety Unit. The Home Accident Surveillance System (H.A.S.S.). Published annually. Department of Trade and Industry. United Kingdom.

DENDUYT-WHITEHEAD, A. P., HART, T. B. & VOLANS, G. N. (1985). Effects of the addition of an emetic to paraquat formulations on acute poisoning in man. *J. Toxicol. Clin. Tox.*, **23**, 422–423.

EFTHYMIOU, M-L. (1983). Resultats d'une enquette nationale sur trois ans concernant les intoxications par paraquat. *Xeme. Symposium national de medicine Agricole*. 29 April 1983.

FITZGERALD, G. R., BARNIVILLE, G., FLANAGAN, M. *et al.* (1978). The changing pattern of paraquat poisoning: An epidemiologic study. *J. Irish Med. Assoc.*, **71** (4), 103–108.

FRAZER, N. C. (1980). Accidental poisoning deaths in British children 1958–1977. *Br. Med. J.*, **280**, 1595–1598.

FRELON, J. H., MERIGOT, P., GARNIER, R., BISMUTH, C. & EFTHYMIOU, M-L. (1983). Facteurs pronostiques de l'intoxication augue par le paraquat etude retrospective sur les cas enregistiés au Centre Anti-Poisons de Paris en 1981. *Toxicol. Eur. Res.*, **5** (4), 163–169.

GIFAP (Groupement International des Associations Nationales de Fabricants de Produits agrochimiques) (1983). Guidelines for the safe and effective use of pesticides. Published by GIFAP, Brussels.

GOVERNMENT REPORTS, ANNOUNCEMENTS AND INDEX (1981). National Study of Hospitalised Pesticide Poisonings. U.S.A.

GROUNDAR, R. P. (1984). Paraquat poisoning in Fiji (Abstract). *J. Forensic. Sci. Soc.*, **24** (4), 376.

HART, T. B. & BRAMLEY, A. (1983). Paraquat Poisoning in the United Kingdom (Abstract). *Human Toxicol.*, **2**, 417.

HAYES, W. J. (1980). Factors limiting injury from pesticides. *J. Environ. Sci. Health*, **B15** (6), 1005–1021.

IPCS (International Programme on Chemical Safety) (1984). Environmental Health Criteria 39. Paraquat and Diquat. p. 74.

LOW, A. A., FARMER, R. D., JONES, D. R. & RHODE, J. R. (1981). Suicide in England and Wales: an analysis of 100 years 1876–1975. *Psychol. Med.*, **11**, 359–368.

MURRAY, V. S. G., FRANCIS, J. & THOMPSON, N. (1986). The incidence of paraquat poisoning in an epidemiological study (Abstract). *Human Toxicol.* (this issue).

NAITO, H. & YAMASHITA, M. (1986). Present status of paraquat poisoning in Japan and a new safe formulation of paraquat (Abstract). *Human Toxicol.* (this issue).

Office of Population Censuses and Surveys (1975). DH4 Series. Mortality – accidents and violence. Table 10.

OSSELTON, M. D., BLACKMORE, R. C., KING, L. A. & MOFFAT, A. C. (1984). Poisoning-associated deaths for England and Wales between 1973 and 1980. *Human Toxicol.*, **3**, 201–221.

ROSE, M. S. (1976). The concentration of PP796 required to produce emesis in experimental animals and an estimation of the emetic dose in man. ICI plc, Central Toxicology Laboratory Report No. CTL/R/390/R and 391.

VALE, J. A. & BUCKLEY, B. M. (1986). Intoxication by pesticides. In *Proceedings of the Conference on the Prevention of Accidental Poisoning in Childhood*, Brussels, 21–22 Nov. 1985, ed. W. Rogmans. European Consumer Safety Association: Amsterdam.

VALE, J. A., BUCKLEY, B. M. & MEREDITH, T. J. (1984). Deaths from paracetamol and dextropropoxyphene (distalgesic) poisoning in England and Wales in 1979. *Human Toxicol.*, **3**, 1355–1435.

VOLANS, G. N. & WISEMAN, H. M. (1986). Epidemiology of accidental poisoning of children in the European Region. In *Proceedings of the Conference on the Prevention of Accidental Poisoning in Childhood*, Brussels, 21–22 Nov. 1985, ed. W. Rogmans. European Consumer Safety Association: Amsterdam.

WEISSMAN, M. A. (1974). The epidemiology of suicide attempts 1960–1971. *Archs Gen. Psychiatry*, **30**, 737–746.

WICKSTRØM, E. (1984). The frequency and problems of paraquat poisoning in Europe, as experienced by the Poison Control Centres. (Abstract) Presented at the Xth International Congress of the European Association of Poison Control Centres, Stockholm, 17–20 June 1986.

WHITEHEAD, A. P., VOLANS, G. N. & HART, T. B. (1984). Toxicovigilance for pesticides. Paraquat poisoning in the United Kingdom. *J. Toxicol. Medicale*, **1**, 5–53.