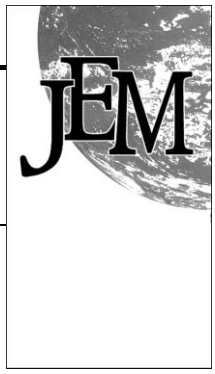

Toxic equivalency factors for PAH and their applicability in shellfish pollution monitoring studies†

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Fish and shellfish are exposed to a wide range of polycyclic aromatic hydrocarbons (PAH) following oil spills at sea, and can become contaminated as a result. Finfish have a more effective mixed-function oxidase enzyme system than shellfish, and are therefore able to metabolise and excrete PAH more effectively than the invertebrates. Thus, contamination by high-molecular weight PAH, including those with carcinogenic potential and so of concern with regard to human consumers, is therefore usually observed in shellfish, and particularly in bivalve molluscs. Oil spills are not the sole source of PAH, however, as parent compounds are also generated by a wide range of combustion processes. In this paper, consideration is given to monitoring data gathered following recent oil spills (both of crude oil and diesel fuel), alongside data from other studies. These include studies conducted around a former gasworks site and downstream of an aluminium smelter in the UK, and from mussel monitoring studies undertaken in the UK and the USA (including the *Exxon Valdez* oil spill and the National Status and Trends programme), and in other countries in Europe. For comparative purposes the PAH concentrations are summed and also expressed as benzo[*a*]pyrene equivalents, their relative concentrations being weighted in relation to the carcinogenic potential of individual PAH compounds using toxic equivalency factors (TEF). Our aim was to assess the utility of this approach in fishery resource monitoring and control following oil spills. Certainly this approach seems useful from the data assessed in this study, and the relative ranking of the various studies seems to reflect the relative degree of concern for human consumers due to the differing contamination sources. As a simple tool for control purposes it is equally applicable to PAH derived from oil spills, and from industrial and combustion sources.

Introduction

Polycyclic aromatic hydrocarbons (PAH) derive mainly from anthropogenic sources and are widely distributed in the environment, particularly around industrial and urban centres.[1–3] PAH are formed as products of incomplete combustion of fossil fuels and other organic matter, and major sources include emissions from wood and coal burning, motor vehicles, power stations and refuse incinerators. PAH are constituents of crude oil itself, being formed during the decay of plants over millions of years, and are also present in most refined products. A consequence of this is that there are literally thousands of individual PAH compounds, especially when the substituted PAH, which are dominant in crude oils and associated distillates, are included.[4] This presents a particular challenge to the analytical chemist. Analysis by high pressure liquid chromatography allows the determination of mainly parent compounds, while gas chromatography-mass spectroscopy permits the analysis of many more parent and branched compounds. Resolution of the many alkylated compounds is, however, still limited and consequently PAH data is often reported as a combination of individual parent compounds and groups of substituted PAH of between 2 and 5 rings. This results in a list of between 35 and 45 PAH compounds/groups being presented following an individual analysis.[5,6]

Although the smaller, 2- to 4-ring PAH compounds are of little concern as far as carcinogenicity is concerned, many of the larger PAH compounds are of concern with regard to human health as they have the potential to produce potent carcinogens.[7] This relates, to some extent, to PAH structure. Such a complex mixture of compounds means that the term "total PAH" is non-specific and the components summed to yield this figure must always be specified. Even when specified, however, it may not allow direct comparisons to be made due to the fact that the individual compounds making up this total may be quite different. Thus there is a need for some alternative parameter to enable an assessment of possible effect.

PAH are also readily accumulated by fish and shellfish,[8] and particularly by bivalve molluscs. As an example, following the *Braer* oil spill, the concentration of PAH (2- to 6-ring, parent and alkylated) in queen scallop gonad was found to be in excess of 26,000 μg kg^{-1} wet weight;[9] and following the *Sea Empress* oil spill the concentration of a similar range of PAH was around 100,000 μg kg^{-1} wet weight in one sample of mussels (Kelly, unpublished data). When oil becomes entrained in sediments the contamination can be particularly long-lasting,[10–12] essentially because oil can be remobilised by benthic organisms or, in sufficiently shallow waters (depths of less than *ca.* 25 m), storm events. Examples of this can be seen in the spills from the *Amoco Cadiz*, *Exxon Valdez*, *Braer* and *Erika*. Restrictions on harvesting of oysters remained in place for seven years following both the *Amoco Cadiz* spill in Brittany in 1978,[13] and for Norway lobsters and mussels off Shetland following the *Braer* oil spill in 1993. Fishery restrictions such as these are often put in place following oil spills, at least until levels of contamination can be established by analysis of

tissues.[14] More general contamination resulting from urban and industrial sources is usually not assessed, and in most countries there are no standards or guideline values for acceptable levels of PAH in fish and shellfish for human consumption. Examples include the USA following the *Exxon Valdez* spill[15] and the UK following the *Sea Empress* spill.[16] In this paper we consider the issue of PAH contamination following oil spills and the applicability of toxic equivalency factors to the assessment of contamination. This approach allows the hazard posed to consumers by consumption of contaminated shellfish following oil spills to be both determined during an incident, and compared with that due to other urban and industrial sources.

Methods

Data were collated from a variety of sources for inclusion in this study, including the *Sea Empress*, *Braer* and *Exxon Valdez* oil spills. All data were collected under appropriate analytical quality control protocols and so should be directly comparable, within the limits of interlaboratory comparability as shown by laboratory proficiency studies.[17] There is some variation in the number of PAH analysed in the studies being compared, as is usually the case in environmental studies of these contaminants.[6] Whilst some standardisation has been attempted for small suites of combustion-derived PAH (for example, the 6 PAH of Borneff; or the 16 PAH in the USEPA priority pollutant list), the vast range of parent and alkylated PAH present in crude oils has meant that in almost no two studies has exactly the same range of PAH been determined. A basic list of PAH to be determined for this purpose has been suggested,[6] but its adoption is limited by the limited availability of pure standard materials for many compounds and the relatively limited number of PAH compounds certified in reference materials produced to date. For this reason we have expressed PAH concentrations both as the sum of the PAH determined in each study and as toxic equivalencies. In the latter case the concentrations of individual PAH are expressed as equivalents of benzo[*a*]pyrene based upon their relative carcinogenic potency, and these values are summed to yield benzo[*a*]pyrene equivalents (BaPEs). A number of sets of toxicity/potency equivalency factors can be found in the literature, although they are all based on a unit value for benzo[*a*]pyrene as the available toxicology data for this compound are much better than those data available for any of the other PAHs.[18] Short-term studies are available for many of the other parent PAHs which allow approximation of their relative potencies. The toxicity/potency equivalency factors differ in the way the factors have been calculated, generally because they are based on the studies most relevant to the matrices that equivalencies are to be calculated for, and this leads to variations in the factors applied for specific compounds. For example, the Kuwait Institute for Scientific Research conducted an exposure assessment for local consumers following the Gulf oil spill of 1991.[19] They investigated the levels of 10 PAH in fish and developed a measurement using mutagenic and carcinogenic potencies of benzo[*a*]pyrene as well as the equivalency values derived by ICF-Clements Associates,[20] and subsequently applied (with the addition of values for fluoranthene and pyrene) by Bolger *et al.*[21] following the *Exxon Valdez* oil spill. Collins *et al.*[22] discussed and calculated potency equivalency factors for use in studies of air contamination, and these have subsequently been applied to American and British atmospheric data. They also considered using the toxic equivalency values developed by Nisbet and LaGoy,[23] which extended the ICF-Clements Associates list to include PAH which had not been definitely classified as carcinogens. The USEPA[24] has published relative potencies for known carcinogenic PAH, and these have been used as a basis for other developments of equivalency factor lists, for example by the Office of Environmental Health Hazard Assessment of the Californian EPA.[25] These factors are compiled in Table 1.

For the purposes of this paper we have used the same factors as those used by Bolger *et al.*[21] in their hazard and risk assessment undertaken for the US Food and Drug Administration of the consumption of contaminated seafood by native Alaskans following the *Exxon Valdez* oil spill in 1989.[18,21] Their assessment was based upon a maximum 1×10^{-6} additional cancer risk from eating fish and shellfish contaminated as a result of the oil spill, with the contamination expected to persist for a period of 10 years. In other US studies reported in this paper these assumptions have been varied as they are conducted by individual state's authorities in the absence of national or international guidelines.[26] In addition, estimates are often made separately for consumers with high and low intakes of these seafood items. These two estimates may be described as delineating "safe" and "unsafe" levels; where "safe" concentrations are below the value calculated for those with the higher dietary intake, and "unsafe" above the value calculated for those with the lower intake. No allowance has been made for other PAH determined in the datasets considered in this paper, but for which no TEF values were cited by Bolger *et al.*[21]

In the current study no allowance has been made for alkylated PAH other than within the values for the sum of all PAH determined. Parent compound data only have been used in the calculation of BaPE values – these have been calculated in the same manner for all studies presented in this paper.

Table 1 Compiled toxic equivalency factors for PAH

Compound	USFDA[a]	KSIR carcinogenic[19]	KSIR mutagenic[19]	OEHHA[25]	USEPA[24]	Nisbet and LaGoy (1992)[23]
Dibenz[*a,h*]anthracene	1.05[b]				1.00	5.00
Benzo[*a*]pyrene	1.00	1.00	1.00	1.00	1.00	1.00
Indeno[1,2,3-*cd*]pyrene	0.25			0.1	0.1	0.1
Pyrene	0.13	0.081	0.2			0.001
Benzo[*b*]fluoranthene	0.11			0.1	0.1	0.1
Benzo[*k*]fluoranthene	0.07			0.1	0.01	0.1
Benzo[*ghi*]perylene	0.03					0.01
Fluoranthene	0.02					0.001
Benz[*a*]anthracene	0.014	0.145	0.62	0.1	0.1	0.1
Chrysene	0.013	0.0044	0.37	0.01	0.001	0.01
Anthracene		0.32	0.06			0.01
Acenaphthene						0.001
Acenaphthylene						0.001
Fluorene						0.001
2-Methylnaphthalene						0.001
Naphthalene						0.001
Phenanthrene						0.001

[a]As used in Bolger *et al.* (1996)[21] [b]Correcting error see Field *et al.* (1999) Table A3-1.[35]

Where data were presented in the original datasets on a dry weight basis they have been converted to a wet weight basis. This was done either using factors for individual samples presented with the data or, where these were not given, by applying a dry/wet factor of 14% derived from both our own studies and reported data from Alaska.

Assessment of PAH data

Comparing and assessing PAH data generated following oil spills is problematic.[6] There are a very large number of PAH compounds which can be monitored, and their behaviour, fate and effects vary widely. PAH are of concern for three main reasons: firstly, low molecular weight PAH can be directly toxic to marine animals; secondly, because metabolites of some of the high molecular weight PAH are potent animal and human carcinogens (benzo[*a*]pyrene is the prime example); thirdly, low molecular weight PAH can cause taint in the fish and shellfish, resulting in consumer rejection of the product and an associated loss in consumer confidence which has an obvious impact on the fish and shellfish industries. Carcinogenic activity is closely related to structure, however, and other PAH with a molecular weight of 252 Da, and so isomeric with benzo[*a*]pyrene, (benzo[*e*]pyrene, perylene, and four isomers of benzofluoranthene) are either much less potent or inactive. There are therefore two problems with comparing and assessing data, as different suites of PAH can be (and are) analysed in fish and shellfish tissues following different incidents, and simple summations of the concentrations of those compounds do not reflect the risk to consumers.

Similar problems have been faced for dioxins and furans, and in this case the use of toxic equivalents (TEQs) has become commonplace. The TEQ is expressed as an equivalent concentration of the most toxic dioxin compound, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) by applying toxicity factors to the concentrations of the other compounds. These factors reflect the relative potency of each of the other dioxin and furan compounds to that of TCDD itself. This approach is now being considered more seriously for PAH (see Table 1), although the situation for PAH is rather more complicated. Dioxins and related compounds bind directly to a receptor without biotransformation and this binding is considered to be responsible for their toxicity. PAHs, in contrast, cannot express their carcinogenic potential until they have been metabolised to forms which can bind to DNA.

Results

Nineteen datasets were examined (Table 2). The sum of the individual measured PAH concentrations (ΣPAH) presented in Table 2 is in each case the sum of the concentrations of PAH and groups determined in that study. As stated above there is considerable divergence in the suites of PAH determined (particularly for alkylated compounds) and it is therefore difficult to make exact comparisons across studies. In the

Table 2 Summary of PAH data considered in this study (ΣPAH and BaPE concentrations in μg kg^{-1} wet weight)

Location	Date	Purpose	*n*	No of PAH[b]	ΣPAH	BaPE[c]	Reference
Scotland	1999–2001	Loch Leven below aluminium smelter	18	37	95 to 7,530	5.3 to 800	36
England	1999	Former gasworks site at Shoreham	15	18	57 to 6,450	1.9 to 379	37
Wales	1996	Crude oil spill (*Sea Empress*)	230	18	34 to 103,000	0 to 326	Kelly (unpublished data)
USA	1986–1996	National status and trends programme (mussel watch)	1689	43	1.0 to 3,760	0 to 222	www.orca.nos.noaa.gov/projects/nsandt/
England and Wales	1997	Spatial survey of commercial shellfish	85	18	36 to 8,930	1.1 to 141	Jones and Kelly (unpublished data)
Alaska, USA	1990–1992	Monitoring study	2297	39	0 to 41,840	0 to 129	Data from Exxon Valdez State/Federal Trustee Council Hydrocarbon Database
Arcachon Bay, France	1995	Research programme[a]	7	14	39 to 338	6.3 to 57	38
Alaska, USA	1989	Crude oil spill (*Exxon Valdez*)	42	35	0 to 18,460	0 to 53	28
England	2000	Crude oil spill (*Tetney monobuoy*)	4	23	214 to 322	14 to 17	Kelly (unpublished data)
England	2000	Gasoil spill (*Erimo*)	4	23	235 to 631	12 to 15	Kelly (unpublished data)
England	2000	Gasoil spill (*Lagik*)	6	23	143 to 355	5.3 to 18	Kelly (unpublished data)
Scotland	2000	Spatial survey of natural and rope-grown mussels	14	37	35 to 719	0.6 to 20	McIntosh (unpublished data)
Germany	1995	Smoked fish[a]	5	13	27 to 197	0.7 to 9.4	27
Alaska, USA	1993–1997	Monitoring study	249	39	4.5 to 1,370	0.1 to 9.3	Data from Regional Citizens Advisory Council
Scotland	1995–2000	Monitoring study in Shetland	14	37	15 to 706	0.1 to 8.2	McIntosh (unpublished data)
Scotland	1999–2001	Loch Etive (reference site)	5	37	26 to 150	0.7 to 5.5	36
Mediterranean Sea	1995–1996	Research programme[a]	26	14	3.6 to 55	0.2 to 4.6	39,40
Baltic Sea	1996	Research programme[a]	9	14	13 to 51	1.2 to 4.2	39
England	2000	Comparative data for traditionally hot-smoked fish	8	18	97 to 2,990	0.1 to 4.4	Kelly (unpublished data)

[a]Alkylated PAH not determined, so ΣPAH values artificially low compared to other similar studies. [b]The number of PAH equates to both individual compounds and groups of compounds. As an example, the 37 PAH determined for Loch Leven include individual compounds, such as benzo[*c*]phenanthrene, benz[*a*]anthracene, chrysene/triphenylene and benz[*b*]anthracene, together with the C_1- and C_2-alkyl derivatives of the catacondensed 4-ring PAH. This collection represents 6 of the 37 PAH determined in this case. [c]Calculated on the basis of the concentration and the toxic equivalency factor for the 10 PAH listed in Table 1, column 1, under USFDA.

studies cited here the number of individual parent PAH and groups of alkylated PAH determined varies from 13 to 43, in a total of 4,748 samples of mussels and 13 samples of smoked fish of various species. In our own studies undertaken during 2000 a sum of 23 individual PAH and groups were determined: acenaphthylene, acenaphthene, fluorene, naphthalene and its C_1- to C_3-alkyl derivatives; phenanthrene, anthracene and their C_1-alkyl derivatives; fluoranthene, pyrene, and their C_1-alkyl derivatives; benz[*a*]anthracene, chrysene/triphenylene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*e*]pyrene, benzo[*a*]pyrene, perylene, indeno[1,2,3-*cd*]pyrene, benzo[*ghi*]perylene and dibenz[*a*,*h*]anthracene. In earlier work, where 18 compounds and groups were determined, acenaphthylene, acenaphthene, fluorene, and the C_1-fluoranthenes/pyrenes were omitted, and the benzofluoranthenes were determined as a single group.

ΣPAH concentrations vary across the studies from undetectable to 100,000 μg kg^{-1} wet weight, and BaPE concentrations from 0 to 800 μg kg^{-1} wet weight. The highest ΣPAH concentration was seen close to the site at which the *Sea Empress* grounded, whilst the highest BaPE value occurred below an aluminium smelter in Scotland which used Soderberg electrodes. Even around heavily contaminated sites the lowest values tend to be close to zero, as the studies are generally designed so as to include a proportion of background samples taken beyond the immediate area of contamination.

Considering the compiled data listed in Table 2, the lowest BaPE values (maximum less than 10 μg kg^{-1} wet weight; 7 of the 19 datasets) derive from monitoring studies in Alaska post-1992 (3 years after the oil spill from the *Exxon Valdez*), and from research studies undertaken in Scotland and the Baltic and Mediterranean Seas. In these studies many of the sites are remote from known sources of PAH. More surprisingly perhaps, this group also includes data for traditionally hot-smoked fish from both England and Germany; hot-smoking is a process which is generally assumed to add significantly to parent PAH concentrations in the finished product.[27] BaPE values in an intermediate range (maximum 10 to 100 μg kg^{-1} wet weight; 6 of the 19 datasets) were observed in Arcachon Bay in France, following three small oil spills in England in 2000, and in Alaska in 1989 immediately following the *Exxon Valdez* oil spill. The shellfish samples analysed following the three small spills in England were all assessed as uncontaminated by these incidents. The BaPE values determined in these cases (5.3 to 18 μg kg^{-1} wet weight) therefore represent the general background contamination resulting from diffuse inputs of PAH in the three areas sampled (the Wash, and the Humber and Wyre estuaries). A similar range of BaPE values (0.6 to 20 μg kg^{-1} wet weight) was seen in natural and rope-grown mussels surveyed during 2000 from around the Scottish coast. The initial Alaskan data (from samples collected in 1989) were those assessed by Bolger *et al.*,[21] and this dataset also included PAH determinations of two samples of smoked salmon obtained locally from Old Harbour on Kodiak Island and Tatitlek in Prince William Sound.[28] These were analysed in duplicate, yielding mean ΣPAH concentrations of 8,360 and 23,600 μg kg^{-1} wet weight, and BaPE values of 43 and 150 μg kg^{-1} wet weight respectively. The majority of the PAH contamination in both samples consisted of 2- and 3-ring alkylated PAH, suggesting a significant oil contamination, presumably prior to smoking. Unsmoked salmon from the same locations showed much lower PAH concentrations though, and mainly of parent (unsubstituted) PAH. Analyses of traditionally smoked fish produced in England (cod, mackerel, trout, salmon, and herring) have yielded much lower BaPE values (BaPE values 0.1 to 4.4 μg kg^{-1} wet weight), as have similar products from Germany (halibut, red-fish, salmon, and sprat; 0.7 to 9.4 μg kg^{-1} wet weight).

The highest BaPE values (maximum $>$ 100 μg kg^{-1} wet weight; 6 of the 19 datasets) were found in spatial surveys undertaken in the UK and USA. These included shellfish from sites close to urban and industrial areas, in Wales following the *Sea Empress* spill, around a former gasworks site in southern England and downstream of an aluminium smelter in Scotland. Fig. 1 shows the maxima for ΣPAH and BaPE for all of the 19 studies considered in this paper. Studies in which combustion sources dominate cluster close to the Y-axis (gasworks, smelter), whilst those deriving from crude oil spills (*Exxon Valdez*, *Sea Empress*) are angled at a shallow angle to the X-axis. Spills involving lighter refined products (diesel fuel, kerosene, aviation fuel) would presumably lie closer still to the X-axis as they are distillate cuts of a limited boiling range and

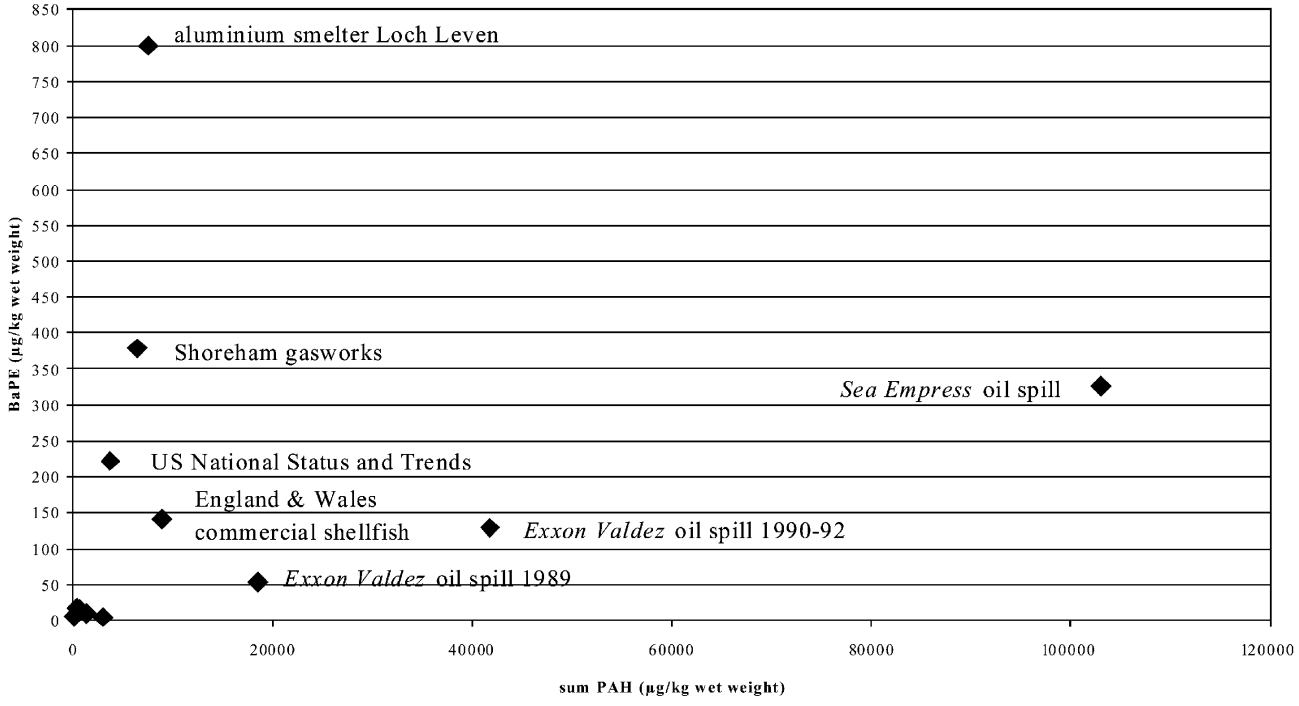

Fig. 1 The relationship between maximum values of ΣPAH and BaPE in the 19 datasets examined in this study (μg kg^{-1} wet weight).

do not contain significant quantities of the higher-molecular weight PAH which contribute most to the BaPE values. The highest values in the US National Status and Trends mussel watch studies also seem, from the position on the chart, to be predominantly combustion-derived. This type of plot could also help to ascribe the dominant sources in areas where both types of inputs can be seen, such as in Milford Haven in the UK.[29]

Discussion

When an oil spillage occurs at sea, there is often a tacit assumption that the oil is impacting a clean area which is otherwise unaffected by PAH contamination. In practice this is rarely, if ever, the case, as PAH are ubiquitous contaminants related primarily to the proximity of urban centres. In the US National Status and Trends mussel watch programme, ΣPAH concentrations determined in mussels and oysters varied by a factor of more than 1,000-fold, from 1.0 to 3,760 μg kg^{-1} wet weight,[30] with clusters of high concentrations occurring around the New York area, and in Puget Sound in Washington State, for example. The PAH profile, and hence the BaPE concentration relative to ΣPAH, will, however, vary with the source of the PAH. Crude oil spills give rise to contamination primarily by low-molecular weight PAH (2- and 3-ring compounds) which are highly alkylated, although certain bivalve molluscs tend to show a proportional increase in the concentrations of the 4- to 6-ring compounds. PAH of 2- to 4-rings generally exhibit little or no carcinogenic potential,[7] but are of concern due to their acute toxicity or tainting properties.[9,31] Spills of heavy fuel oil (HFO), as for instance that from the *Erika* in France in 1999, are becoming more common relative to those involving crude oil. As these higher-boiling distillation fractions and blends generally contain a greater proportion of PAH with more than 4 rings, which include those which can be carcinogenically activated, they pose a significant contamination risk.

Although no standards or guideline values have been set for PAH in current UK legislation, the UK Food Standards Agency, advised by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) , have adopted an interim pragmatic guideline limit value of 15 μg kg^{-1} wet weight for any one of three named PAH considered to be of greatest concern (benz[*a*]anthracene, benzo[*a*]pyrene and dibenz[*a,h*]anthracene). The full text of the COT statement is available on their website (http://www.foodstandards.gov.uk/committees/cot/). The guideline limits are to be applied only in emergencies such as oil spills, and where adequate background data do not exist to demonstrate that the levels observed in foodstuffs do not result from the emergency under consideration. Concentrations above the limit value would be likely to initiate fishery closures intended to protect human consumers.

There have been a number of instances in the USA in which the BaPE approach has been used to estimate risk and to develop fishery management criteria. The establishment of "acceptable" levels for PAH ingestion (*i.e.*, those calculated as presenting little or no additional cancer risk to consumers) can only be developed for a specific area, as they will be dependent on the patterns of consumption of contaminated fishery products. Such values cannot therefore simply be applied globally, but it is useful to observe the order of magnitude into which these values fall and their similarity across incidents and locations.

Following a heavy fuel oil spill from the bulk carrier *New Carissa* in Coos Bay, Oregon, in 1999, the development of criteria for the reopening of shellfisheries yielded "safe" (<10 μg kg^{-1}) and "unsafe" ($>$ 45 μg kg^{-1}) BaPE concentrations for PAH determined in shellfish tissues.[26] In this incident the "safe" and "unsafe" values were derived based on upper (32.5 $gday^{-1}$) and average (7.5 $gday^{-1}$) estimates of shellfish consumption. Interestingly, the two shellfish samples yielding the highest BaPE concentrations around Coos Bay showed PAH profiles which indicated a source other than the *New Carissa* oil. Other studies have yielded criterion values of a similar order to those established by Gilroy. For fuel oil spills from the *Kure* in Humboldt Bay, California, in 1997, pass/fail values calculated as BaPE were 5 and 34 μg kg^{-1}; and from the *Julie N* in Portland, Maine, in 1996, 16 and 50 μg kg^{-1} respectively (all on a wet weight basis, Ruth Yender, NOAA, personal communication). Using these broad classifications to rank the data in Table 2 indicates that contamination from diffuse sources generally falls within the lower part of this band (BaPE values up to about 20 μg kg^{-1} wet weight), whilst large contamination events (oil spills or PAH leaching from a former gasworks site) yield much higher values.

Conclusions

Given the differing suites of individual PAH compounds analysed in the studies reported here it would be difficult to compare the results of the different studies merely by summing the PAH determined. For decisions relating to closure and re-opening of fisheries it is the risk posed to human consumers that is important. For this purpose the use of TEFs to generate data expressed as benzo[*a*]pyrene equivalents has proved to be a useful framework for evaluating potential effects of oil spills on food supplies.[18] Our own comparison of a number of datasets across a wider range of contamination sources and geographic areas support this conclusion. Additional datasets should now be gathered, and the approach further evaluated. It should be borne in mind, however, that this approach does not address the wider aspects of environmental impacts on wildlife, resource aspects of fisheries or mariculture, or the potential for enhanced toxicity of PAH due to the action of ultraviolet light.[32–34] Analysis of a wide range of PAH in tissues and other matrices, and detailed interpretation of these data, are still required in order that ecotoxicological hazards can be addressed.

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