

FOOD CONTAMINANTS: SCIENTIFIC AND PUBLIC HEALTH IMPLICATIONS

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Summary

This paper attempts to put into perspective the issue of contaminants in foods, and includes both microbiological and chemical contaminants, whether naturally present (intrinsic toxicants such as mycotoxins) or occurring as a result of human activity or environmental conditions. The public has a different perspective on food safety priorities than do food professionals. To the extent that governments reflect public opinion, research priorities and regulations often address those issues about which the public is concerned, even though they are of a lesser food safety impact. Prioritising food safety issues is critical. In developing exposure data, an important factor involves sampling, an area that historically has received little attention by the research community. The problems involved in developing exposure data are of such magnitude that risk analyses often become a matter of estimating relative risk based on questionable exposure data (incidence and level data) and even more questionable toxicological data. This may force the regulator to turn from science to the more practical economic and political factors in setting regulatory levels. It is not surprising, therefore, that there is wide disparity in regulatory levels among countries. There is a great need, therefore, for implementing carefully designed quality control programs, and for continuing research efforts to improve the precision of analytical methods, the toxicological data base on toxicants in foods, and methods of risk assessment. Such research will be invaluable in setting limits for food toxicants, in developing regulatory control programs, and in providing information that can be used to protect public health and educate the consumer.

I. INTRODUCTION

Food contamination is a problem of global dimensions. It is acknowledged to be a problem by most countries in their regulatory approach toward foods. Unfortunately, there is much misunderstanding and confusion surrounding the issue of contaminants in foods. To the food scientist, contamination is the presence in food of a specific organism or chemical which may or may not have a negative effect on humans if ingested. To the consumer, food contamination is often interpreted as some undefinable, but undesirable, component of food; the result is a loss of confidence in the safety of the particular food involved and an overestimation of the benefits of 'natural' foods compared to other foods.

This paper will focus primarily on the chemical contamination of foods rather than microbiological contamination. This is not meant to trivialise the microbiological (ie bacterial and viral) contamination of foods. The Food and Drug Administration (FDA) for many years has held to the view that, in the ranking of food safety issues, the most serious, important, and pressing problems involve microbial contamination, with nutritional imbalance the second most troublesome problem (Schmidt 1975; Table 1).

Table 1. Food safety priorities established by FDA

1. Microbial contamination
2. Nutritional imbalance
3. Environmental contamination
4. Natural toxicants
5. Pesticide residues
6. Food additives

At the time that the FDA set these priorities (1975), the naturally occurring toxicants, both intrinsic and extrinsic, were grouped and rated as being of slightly less concern than the 'environmental', ie manmade, contaminants. Pesticide residues, in the opinion of food safety experts, were of low priority, while food additives were of least concern, primarily because these chemicals were deemed controllable, an extremely important variable when dealing with risk management.

In the USA, according to a recent estimate (Archer and Kvenberg 1985), at least 24 million and possibly as many as 80 million cases of foodborne disease occur annually, with as many as 9000 fatalities (Cohen 1987). The incidence of bacterial-related cases of food poisoning appears to be rising. Although it is difficult to identify the reasons for this, the following factors are involved (WHO 1992): (a) increased population; (b) changes in the way foods are processed, ie the increasing demand for processed vs fresh foods, 'fast' foods, frozen foods, and specially packaged foods; (c) the increasing concentration of the industry to achieve economies of scale, eg in the USA recently, a breakdown in the controls in a modern milk processing plant led to widespread salmonellosis; (d) new, virulent strains of bacteria; and (e) better reporting of food-related illnesses and improved record keeping.

Setting aside, then, the subject of microbiological contamination of foods, let us focus on the issues surrounding chemical contamination of foods, discussing, in sequence, the types of chemical contaminants encountered, the analytical problems that must be addressed in developing the data needed for making exposure estimates, and finally the setting of regulatory levels and the enforcement of such levels.

II. TOXIC CHEMICALS IN FOODS

'Toxic' chemicals in foods can be classified into four types (Table 2); each type raises food safety issues.

The environmental contaminants produced by microorganisms include mycotoxins, such as aflatoxins, which are produced by molds growing on foods; phycotoxins (ie toxins produced by algae that are then ingested by marine organisms), such as paralytic shellfish poisons, domoic acid, and 'ciguatoxins'; and a large number of environmental pollutants, such as lead, polychlorinated biphenyls (PCBs), dioxins, etc. Many of these contaminants were once used legally but their use was limited as further evidence of their toxic effects became known. These environmental contaminants are difficult to deal with for many reasons, including, in some cases, their extreme acute toxicity (eg dioxins), the fact that they often are known to be mammalian carcinogens, and the fact that they are often present at sub-ppm and sub-ppb concentration ranges.

Table 2. Chemicals in foods causing food safety concerns

Chemical type	Examples	Degree of concern ¹	
		Regulatory	Public
Environmental contaminants			
Microbial origin	Mycotoxins ← <i>fungi</i> Phycotoxins ← <i>algae</i>	+++ ++++	+ ++
Industrial origin	PCBs Pb Dioxins Radionuclides	+ + + +	+++ +++ +++ ++
Residues	Pesticides Animal drugs	++ ++	++++ +++
Intrinsic toxicants	Phytoalexins Allelochemicals Pyrrolizidine alkaloids Glycoalkaloids	+ + + +	- - - -
Food additives	Direct Indirect	- -	+++ +++

1. As estimated by the authors.

Residues of pesticides and animal drugs are more easily dealt with because they are introduced into the food chain by direct human activity and for this reason, are regulated by controlling the approval for use. In the USA, the Environmental Protection Agency (EPA) evaluates the risk posed by the use of a particular pesticide, approves or disapproves its use, and sets a regulatory level for control of such use. FDA is charged with ensuring that the regulatory level is not exceeded in the food product. In a 1990 survey conducted by the Food Marketing Institute (FMI 1990), pesticide residues were ranked by consumers as their highest food safety concern. However, according to a World Health Organisation (WHO) report on health and the environment, 'there is no indication of any harm to human health arising from residues of agricultural chemicals in food, when limits established by Codex are followed' (WHO 1992), and although 230,000 cases of poisoning and deaths are reported each year worldwide, these are largely the result of accidental exposure to or misuse of agricultural chemicals.

Animal drugs are similarly controlled, in that the manufacturer must obtain approval for use from the FDA and a regulatory level is set. In both cases, the manufacturer has the responsibility for developing all pertinent information required for making an assessment of safety before obtaining approval for use.

The intrinsic toxicants include those compounds which occur naturally in food plants in

large numbers, largely unidentified and unquantified. They include such well-known toxins as the pyrrolizidine alkaloids in comfrey tea, the glycoalkaloids in potatoes and tomatoes, and the cyanogenic glycosides in legumes, to name only a few. Included in the intrinsic toxicants are the phytoalexins produced by plants to ward off infection by a pathogen or in response to cutting or bruising, and the allelochemicals (pesticidal compounds) produced by plants to ward off insects. There is growing interest in this category of naturally occurring toxicants as a result of recent advances in biotechnology leading to new, genetically engineered plant species (eg the Robo tomato). Although many of these compounds are acutely toxic, exhibit strong mutagenic and/or teratogenic activity, and even carcinogenic properties, exposure is generally extremely low and is balanced by a growing number of newly identified compounds which seem to have protective (ie anticarcinogenic) properties. Unravelling the combined effects of the natural components of plant foods will occupy food scientists for many years.

III. PUBLIC HEALTH IMPLICATIONS

Consumer surveys indicate that the public's food safety concerns relative to chemicals in foods are quite different from those expressed by food safety professionals (Pariza 1990); there is, in fact, an inverse relationship. Food safety concerns expressed by the consumer result in tremendous pressures on governments to take regulatory steps to reduce or even eliminate such substances from foods. The food scientist plays a major role in mitigating these concerns, and in advising regulatory officials to ensure that research emphases (and funds) are not misplaced, and that 'over-regulation' does not occur due to overly conservative risk assessments. This is a difficult task given the current status of our knowledge base for estimating exposure and the unresolved problem of estimating human consequences based upon animal-derived toxicological data.

In setting a level to be used for regulatory purposes, a logical sequence of events should be followed. The first phase in this process can be characterized as the 'discovery' phase, in which a food safety problem is identified; the toxic entity is isolated, purified, and chemically characterised; and analytical methods are developed to identify and quantitate the toxin in foods. These methods are subsequently evaluated in method performance studies (collaborative studies). The second phase then becomes one of assessment of exposure and toxicological consequences, ie measurement of incidence and levels, and an indepth evaluation of the toxicological properties. These studies are the foundation upon which the risk assessment is built. The setting of a regulatory level can then be justified or defended. Once a regulatory limit is set, one must then ask the following questions: (1) Is the level chosen enforceable using currently available methodology? (2) What sort of program must be put in place to ensure compliance? Consideration of the former leads to the corollary question: What confidence do we have in the analytical methodology upon which the incidence and occurrence data are based?

IV. SCIENTIFIC RESERVATIONS

In spite of advancements in analytical science and the many examples in the scientific literature of analyses being accomplished in the sub-ppb range, in food analyses these contamination levels are often at the very limits of the capability of available sampling/analytical methodology. This is because in the analysis of any particular lot of food, the total analytical variability is the sum of (a) the sampling variability, (b) the subsampling variability, and (c) the variability encountered in the analysis of the test sample (Horwitz 1988). Of these three, the sampling variability is the major contributor for heterogeneously contaminated lots. Of course,

when homogeneous contamination is encountered, as in a processed food, the sampling variability becomes much smaller.

In the few cases where research on sampling has been done, for inhomogeneously contaminated lots, the lower the contamination level, the greater the sampling error. Frequently the sampling error is so large that improvements in analytical methodology can have only a minimal effect on the total interlaboratory relative standard deviation (RSD_R). Each analyte/matrix combination is different with respect to the magnitude of the sampling error. There are, of course, ways to minimize this error, eg by taking larger samples. Subsampling errors also require careful attention because this error component can be large depending on the success of the grinding/mixing operations.

What about the variability of the analytical method itself? The only way to measure such variability between laboratories is through a method performance study. Through evaluation of the data from such studies, it has been shown that as the contamination level is lowered, the interlaboratory coefficient of variation (RSD_R) becomes larger (Horwitz 1972). This generalisation has been found to be independent of method of analysis, matrix, or analyte. In fact, it has been found that the coefficient of variation doubles as the concentration is lowered two orders of magnitude (Horwitz 1972), and this principle is described by the following equation:

$$RSD_R (\%) = 2(1.05 \log C) = 2C^{-0.1505}$$

where concentration (C) is expressed as a decimal fraction. A plot of this equation yields the so-called 'Horwitz Horn', showing the predicted precision as a function of analyte concentration (Horwitz et al. 1980). The validity of this equation has been confirmed by examining the data obtained in method performance studies on a wide variety of analytes (Horwitz and Albert 1992). By making a simple calculation, one can easily show that at a concentration of 1 ppb (10^{-9}), one would expect a RSD_R of 45% (see Table 3).

Table 3. Predicted precision for analytical methods as a function of concentration

Concentration	RSD_R (%)
1 ppm (10^{-6})	16
0.1 ppm	23
10 ppb (10^{-8})	32
1 ppb	45
0.1 ppb (10^{-10})	64

One can evaluate the capabilities of an analytical method by comparing the RSD_R determined experimentally in an interlaboratory method performance study with that calculated using the Horwitz equation; the ratio of the two values is termed the HORRAT (Horwitz Ratio; Horwitz and Albert 1984).

$$HORRAT = RSD_R (\text{found}) / RSD_R (\text{predicted})$$

* For well-behaved analytical methods this ratio is close to unity. On the other hand, HORRAT values >2 , that is, values twice those predicted by the Horwitz equation, indicate that the method is not acceptable for regulatory purposes with respect to precision.

IV. MYCOTOXINS

An excellent illustration of the problems faced by the food scientist in advising regulatory officials in dealing with food contaminants is that of mycotoxin (especially aflatoxin) contamination of foods. Over 50 countries currently have set legal limits for controlling the presence of aflatoxins in foods; an additional 15 countries have set regulatory levels for eight other mycotoxins (Table 4) (van Egmond 1989). These limits vary tremendously, depending on whether the country involved is a developed or underdeveloped one, and whether the country is a net exporter or importer of the particular commodity involved.

Table 4. Regulatory limits for mycotoxins in foods

Mycotoxin	Commodity	Limit (ppb)	
		US	Other countries
Aflatoxin	Foods	20	0-50 (typical 5)
	Feeds	20	(typical 15)
	Cottonseed meal	300 (cattle)	
	Corn, peanuts	300 (cattle)	
		200 (swine >100 lb)	
		100 (breeding cattle)	
Aflatoxin M1	Milk	0.5	0.01-0.5
Chetomin			0
Deoxynivalenol	Feeds	4000	5-2000
	Wheat	2000	
	Foods	1000	
Ochratoxin	Foods		5-50
Patulin	Fruit juices		20-50
Phomopsin	Lupin products		5
Stachybotryotoxin	Feeds		0
T-2 toxin	Grains		100
Zearalenone	Foods, corn		30-1000

These regulatory limits seem to be a practical compromise between the desire to have a carcinogen-free commodity (absolute safety) and the economic consequences of the setting of regulatory limits. There is, of course, some concern also that the limits might appear to be set as a subterfuge for trade barriers. Certainly in the case of aflatoxin, there was little scientific basis, or the available scientific information was not used, in setting regulatory levels in most countries (Stoloff et al. 1991). Most recently the Codex Committee on Food Additives, in an attempt to harmonise regulatory levels internationally with those of the European Community, proposed a limit of 10 ppb for aflatoxin in processed foods.

There are, of course, two major reasons from the scientific point of view for this disparity in regulatory levels. One is the great difficulty encountered in estimating exposure; the other is the equally difficult problem of assessing human health effects based upon animal toxicological data. Since exposure levels must be based upon the measurement of incidence and occurrence of a particular toxicant, the first issue to address, in the case of mycotoxins, was the question of sampling. The extensive work done on sampling (Campbell et al. 1986), the development of sampling plans, and the measurement of sampling variability in the analysis of

foods for mycotoxins should be a warning to those who would belittle the difficulty in dealing with nonhomogeneous contamination problems, and consequently the magnitude of the research effort required.

As was mentioned previously, the total analytical variability in any analysis is clearly the sum of the sampling, subsampling, and analytical variability. In the case of aflatoxin contamination of corn, the relative magnitudes of these components of the total variability have been estimated (Table 5; Whitaker 1991). Total variability increases as the contamination level falls, so that below 5 ppb the variability becomes >50%, the point at which statistical control over the analysis begins to be lost. In addition, the sampling and subsampling variabilities become the major factors at lower concentration levels.

Table 5. Relative magnitude of error components in testing cracked corn for aflatoxin

Level (ppb)	Coefficient of variation (%)			
	Sample (5 kg)	Subsample	Analysis (50 g)	All sources
1	88.9	50.0	26.4	105.4
2	62.9	35.4	26.4	76.8
3	51.3	28.9	26.4	64.6
5	39.8	22.4	26.4	52.7
10	28.1	15.8	26.4	41.7
15	23.0	12.9	26.4	37.3
20	19.9	11.2	26.4	34.9
30	16.2	9.1	26.4	32.3
50	12.6	7.1	26.4	30.1

In a recent study (Whitaker et al. 1992) 40 lots of runner peanuts were analysed using 5 lb (2.27 kg) samples, 100 g subsamples, and a high performance liquid chromatographic (HPLC) method. The sampling component amounted to 90.7% of the total variability, whereas the subsampling and analytical variability amounted to only 7.2% and 0.1% of the total variability, respectively. The mean aflatoxin level in this study was 100 ppb.

What confidence can be placed in the analytical method itself, ie what is the magnitude of the analytical coefficient of variation? Horwitz and coworkers have recently critically analysed the results of method performance (ie interlaboratory collaborative) studies published through 1991 involving mycotoxins. Nearly 1000 data sets, ie the results obtained on a single test sample, were examined. A summary of the calculated HORRAT values using these data is shown in Table 6.

Table 6. Cumulative distribution of HORRAT values for mycotoxins

Mycotoxin	No. of assays	HORRAT	
		Ave.	% <2
Aflatoxin B ₁	209	1.36	86
B ₂	110	1.11	95
G ₁	70	1.74	71
G ₂	53	1.63	75
B+G	178	1.38	85
M	67	1.26	87
Deoxynivalenol	14	2.76	29
Fumonisin	10	2.34	10
Ochratoxin	24	1.86	67
Patulin	13	1.74	77
Sterigmatocystin	8	1.98	75
Zearalenone(ol)	32	2.33	53
Drugs	996	0.73	98
Pesticides	953	0.95	93

good!
homogeneous
-higher levels

Analysis of these data reveals that most of the methods for aflatoxin show precision in the acceptable range, with a HORRAT <2, ie with RSD_R values experimentally less than two times the values predicted by the Horwitz equation. However, the precision observed with methods for mycotoxins other than aflatoxin is much poorer, reflecting undoubtedly the lesser degree of effort placed on developing methodology for these compounds. This does not mean that the methodology is totally unsatisfactory, only that for certain analyte/substrate combinations, at a particular analyte concentration, the precision of the results obtained was not within the bounds considered satisfactory from a historical basis. For example, the raw data for ochratoxin are shown in Table 7, and those for fumonisin in Table 8. The precision of the methods studied is good for ochratoxin A, but not good for the only method included in the data bank for fumonisin. Of course, many newer methods for determination of fumonisins are now available, but these have not yet been evaluated in interlaboratory method performance studies.

Finally, analysis of the data developed on method performance studies led to the following conclusions: (1) There has been very little improvement in the between-laboratory precision (RSD_R) over the past 20 years in spite of the considerable advances in analytical technology. This probably reflects the major difficulties encountered in preparing the test samples used in such studies and in controlling the quality of the standards used. (2) The precision observed for the thin layer chromatographic (TLC) methods was about the same as that observed for the HPLC methods and better in general than that observed for immunoassay-based methods. (3) The available analytical methods are capable of analysing foods for aflatoxin in the ppb range with a minimum limit of measurement of about 5 ppb B₁. (4) The data show that at 30 ppb about 95% of the results will be positive, whereas at 5 ppb only about 50% will be positive. Of course, the variability can be reduced by using multiple, independent test samples and averaging the results. (5) The data from the method performance studies on M₁ in milk reveal that no study has yet been conducted at the level proposed by Codex for M₁ in

liquid milk (0.05 ppb), and that the methods for dry milk that have been evaluated in an interlaboratory study are not capable of meeting that goal (0.05 ppb) either.

Table 7. HORRAT values for ochratoxin A data sets

Matrix	Method	Level (ppb)	RSD _R (%)	HORRAT
Barley	HPLC	14.3	25.8	0.85
		35.7	31.7	1.20
	TLC	50.1	61.3	2.44
		86.3	33.3	1.44
		86.7	38.8	1.68
Green coffee	TLC	231.4	54.6	2.74
		28.5	92.7	3.31
		38.3	28.6	1.09
		100.9	36.8	1.63
		156.4	38.7	1.83
Corn	HPLC	157.5	11.8	0.56
		16.1	29.0	0.97
		38.1	31.0	1.19

Table 8. HORRAT values for fumonisin B1 data sets

Matrix	Method	Level (ppb)	RSD _R (%)	HORRAT
Corn	HPLC	212.1	37.1	1.83
		301.8	40.2	2.10
		506.3	38.2	2.15
		1077.5	39.5	2.49
		1814.8	38.9	2.66

In recent years, much effort has been devoted to the development of screening assays for mycotoxins, primarily immunological-based assays (Pohland et al. 1991). These assays are designed to screen out the presumed large numbers of negative samples, ie those with aflatoxin levels below a certain limit, from positive samples; the positive samples would then be reanalysed using a quantitative procedure. These screening assays require a completely different statistical approach. They show great promise for their intended use when employed with the proper laboratory quality control procedures.

V. PESTICIDES

Another illustration of government agencies dealing with a regulatory issue is pesticide residues in foods. Once a pesticide residue limit is set, it is incumbent upon the appropriate

regulatory agency to monitor compliance with the limit. The availability of sampling plans and a knowledge of the capabilities of the analytical methods used are essential in the implementation of such control programs. An example of the type of program followed in the USA is FDA's pesticide program.

The overall goals of this monitoring program are to (a) ensure compliance with regulatory limits; (b) develop incidence and level data to allay consumer concerns about pesticides; (c) develop basic scientific information on the source, occurrence, and prevalence of residues; and (d) develop information on exposure to be used in risk assessment.

FDA uses three approaches to carry out its pesticide program: (a) regulatory monitoring, (b) incidence/level monitoring, and (c) the Total Diet Study. Under regulatory monitoring, FDA monitors both domestic and import foods, except for meat, poultry, and egg products, to enforce tolerances set by EPA. Domestic samples are collected as close as possible to the point of production in the food processing chain; if the sample is from an import lot, it is collected at the point of entrance into the USA. The laboratory sample is usually analysed using a multiresidue method (MRM) (PAM I 1968 and revisions). The MRMs available are capable of analysing about half of the pesticides with EPA tolerances. If a pesticide not detectable by an MRM is suspected, a single residue method is used (PAM 2 1968 and revisions). The lower limit of reliable measurement for these methods is in the 0.1-50 ppm range, well below tolerance levels. In 1991, under regulatory monitoring, FDA analysed 19082 samples (8466 domestic and 10616 imported from 102 countries) (FDA 1992). Of these samples, 18214 were surveillance samples, ie samples collected with no information suggesting the lot of food from which they were collected contained illegal pesticide residues. In addition, 868 compliance samples, ie samples collected because of suspicion of a pesticide problem, were collected and analysed. In the case of the domestic samples, no pesticide residues were found in 64% of the 8281 surveillance samples, <1% had over tolerance residues, and <1% had residues for which there was no tolerance for that particular pesticide/commodity combination. Of the compliance samples, 48% of the 185 domestic samples contained no residues, and not unexpectedly, 19% were found to have over tolerance residue levels or residues for which there was no tolerance. For import samples, 69% of 9933 samples had no detectable residues, <1% had over tolerance residues, and 2% had residues for which there were no tolerances. These results are typical; over the past 5 years the % of domestic samples with no residues (NR) found has ranged from 58 to 65, and for imports from 56 to 69. The % of over tolerance or no tolerance residues has been <1 for domestic samples and 2-5 for imports (Table 9).

Table 9. Pesticides in foods: 5-year summary

No. of Year samples	Domestic (%)			Import (%)		
	NR	>Tol.	No Tol.	NR	>Tol.	No Tol.
1987 14492	58	1	1	56	<1	5
1988 18114	60	<1	1	62	<1	5
1989 18113	65	<1	<1	67	<1	3
1990 19146	60	<1	<1	64	<1	4
1991 18214	64	<1	<1	69	<1	2

Incidence/level monitoring is complementary to regulatory monitoring. Under this approach, surveys are conducted to fill gaps in FDA knowledge about particular

pesticide/commodity combinations by analysing randomly selected samples to determine the presence and levels of certain pesticides. In 1991, for example, three special surveys were conducted. One involved a survey of shellfish and finfish (188 samples) for environmentally persistent pesticides; low levels of chlorinated pesticides were found, none violative. Another survey involved the analysis of 806 samples of pasteurised milk from 63 metropolitan areas for chlorinated pesticides; 49.4% (398) of the samples contained residues. The most frequently found were p,p'-DDE (212 findings) and dieldrin (172). These pesticides have not been registered for agricultural use in the USA for over 20 years; however, because of their persistence in the environment, they are still found at low levels in some foods, especially those of animal origin. In a survey of processed foods, 5565 analyses were carried out for selected pesticides. In this survey, pesticide residues were identified 600 times; in some samples more than one pesticide was found. None of the samples contained violative residues (ie over tolerance) and no pesticides were found for which there was no tolerance.

The third approach in FDA's pesticide program is the Total Diet Study, which is designed to yield information on dietary intakes of pesticide residues by eight age/sex groups. Under this approach, FDA collects foods from supermarkets or grocery stores four times per year, once from each of four geographical regions of the country. Each collection is composed of 234 food items selected on the basis of information developed in nationwide dietary surveys. Each of the four collections is a composite of like foods purchased in three cities in that region. The foods are prepared table ready and then analysed for pesticide residues. In 1991, 936 food items were analysed; 51 pesticide residues were identified and quantitated using modified methodology allowing residue measurement down to 1 ppb. The levels of pesticides found, along with food consumption data, are used to estimate the dietary intakes of the pesticide residues. Malathion continues to be the residue most frequently found because of its use on a wide variety of crops.

V. CONCLUSIONS

In summary, the development of the incidence and level data necessary to make exposure estimates is dependent on an ability to analyse a food product with confidence. The first problem faced by the analyst is to obtain a sample representative of the bulk food. This is not a trivial task, particularly with heterogeneously (nonuniformly) contaminated lots, and the experience gained with aflatoxins has shown that the sampling error is by far the major contributor to the total analytical error.

The second problem is to minimise the analytical error. The most effective means of accomplishing this is to: (a) select a method which has been shown to have suitable precision (HORRAT <2), and (b) institute a strong laboratory quality control program. Such a program should include training of the analyst using practice samples at the expected contamination levels, use of check samples on a continuing basis, and use of well-characterised quantitative standards. If these procedures are followed rigorously, the data developed in surveys of various types can be used with confidence to estimate human exposure. Two examples, mycotoxins and pesticide residues, are used to illustrate these points.

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