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Autor: Pitt, John I.

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Application of the food safety objective concept to the problem of aflatoxins in peanuts*

John I. Pitt, Food Science Australia, North Ryde, NSW, Australia

Introduction and background

Aflatoxins are a hazard in foods because they are potent carcinogens, capable of causing cancer in nearly all animal species studied. Aflatoxins are also acute, chronic and genotoxic poisons (1). In addition, recent evidence suggests strongly that aflatoxins can be immunosuppressive (2).

The primary sources of aflatoxins are the common fungi *Aspergillus flavus* and the closely related species *A. parasiticus*. *A. flavus* is very common in tropical and subtropical regions of the world, and is particularly associated with peanuts and other nuts, and with maize and other oilseeds. *A. parasiticus* is primarily associated with peanuts, and has a more restricted distribution (3).

Many countries have established very low maximum permitted levels of aflatoxins in foods, usually in the range of 1 to 25 µg/kg total aflatoxin (4). The Codex Committee on Food Additives and Contaminants of the Codex Alimentarius Commission has recommended to this Commission that the limit for foods in international trade be set at 15 µg/kg total aflatoxins.

Aflatoxins are readily detected by a very strong fluorescence in ultraviolet light. Analyses may be performed using thin layer chromatography, high performance liquid chromatography or immunoassays (5, 6).

Risk assessment

Aflatoxins are among the most potent mutagenic and carcinogenic substances known. Extensive experimental evidence has shown that aflatoxins are capable of inducing liver cancer in nearly all animal species, including birds, fish, dogs and other monogastric animals, and primates (1, 7). However, translating that information to humans has proved to be extremely difficult. Early epidemiological studies (8) indicated that the level of exposure to aflatoxin correlated with the incidence of liver cancer, but some later reports suggested that liver cancer induction was due to hepatitis

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B virus and independent of the occurrence of aflatoxin (9). Other work has supported the role of aflatoxins in human liver cancer. A study in Swaziland established that liver cancer rates correlated well with aflatoxin intake, which varied with region, but not with hepatitis B, which did not show geographical variation (10). A good correlation was reported between the incidence of liver cancer and the level of aflatoxin contamination in foodstuffs in the Chinese province Guangxi. Recent evidence indicates that both aflatoxins and hepatitis B virus are involved in the very high incidence of primary liver cancer in parts of Africa, Southeast Asia and China (11).

Viral hepatitis is a major worldwide public health problem. It is estimated that over 300 million individuals are chronically infected with hepatitis B (HBV) and perhaps 100 million with hepatitis C (HCV). HBV is prevalent in the developing parts of the world, and HCV is emerging as a major cause of hepatocellular cancer in Japan and Western societies (12).

The Joint FAO/WHO Expert Committee on Food Additives (JECFA), which is the Codex Committee responsible for assessing the toxicology of chemicals in foods, has summarised this position: "Risks from specific exposures to aflatoxins are difficult to estimate and predict... Many questions remain regarding the independence of aflatoxin as a human carcinogen, [and] the extent to which hepatitis B, hepatitis C and other factors modify the effect of aflatoxin...." (13).

Dose response analyses

To help answer these questions, JECFA reviewed dose response analyses performed on aflatoxins. Observations concerning the interaction of hepatitis B and aflatoxins suggest that two separate aflatoxin potencies exist, one in populations in which chronic hepatitis infections are common and the second where such infections are rare. In consequence, JECFA divided potency estimates for analyses based on toxicological and epidemiological data into two basic groups, applicable to individuals with and without hepatitis B infection. Mean potency values for these two groups were chosen, of 0.3 and 0.01 cancers per year per 100000 population per ng aflatoxin ingested per kg body weight per day, respectively (13).

Population risks

The fraction of the incidence of liver cancer in a population attributable to intake of aflatoxins is derived by combining aflatoxin potency estimates (risk per unit dose) as described above with estimates of aflatoxin intake (dose per person). In one such calculation, JECFA assumed a population with a European diet, from which all lots containing over 20 µg/kg aflatoxin had been removed. The mean aflatoxin intake for this population was 19 ng per person per day. Assuming a 60 kg person, the mean cancer risk for that population was 0.004 cancers per 100000 population per annum.

At the other end of the scale, *Lubulwa and Davis* (14) estimated deaths from aflatoxins in Indonesia, a country of high risk. They used data of *Pitt and Hocking*

(15) and Pitt *et al.* (16) on the incidence of aflatoxin in Southeast Asian commodities, and estimated that the liver cancer rate from aflatoxins in Indonesia was 10 per 100 000 population per annum, a rate 1000 times higher than the JECFA estimate for European populations. Peanuts accounted for most of the ingested aflatoxins. Given the Indonesian population approaches 200 million people, those figures indicate 20 000 deaths per annum from liver cancer due to aflatoxins in Indonesia.

Risk Management

Because aflatoxins are well known chemical hazards (albeit from a microbial source), risk management has taken a different path from that expected for bacteria or bacterial toxins. In the years following their discovery, the limits set for aflatoxins in foods at first amounted to the limit of detection by chemical assay. In importing countries, this was at first 5 µg/kg, then in some cases reduced to as low as 1 µg/kg (4). However, it soon became clear that producing countries could not meet such limits: the USA set 25 µg/kg and Australia 15 µg/kg as practical limits, which would reduce exposure to aflatoxins as far as possible without destroying the peanut industries in those countries.

Epidemiological and animal studies which followed established that aflatoxins were genotoxic carcinogens. In consequence, limits continued to be set more on the basis of perceived risk in importing countries (4), while those set in developed exporting countries continued to be based on those attainable by the producing industry. Limits established by regulation in less developed producing countries were often nominal and seldom met in practice (15).

Acceptable level of protection

The development of an Acceptable Level of Protection (ALOP) for aflatoxins has proved very difficult, both because a no effect level for aflatoxins in foods has not been established, and because of the perceived synergy of aflatoxins with HBV and HCV.

During the mid 1990s, JECFA carried out a thorough reexamination of the toxicity of aflatoxins, by setting up hypothetical standards for maximum allowable aflatoxin concentrations in foods. In one example, the distribution of aflatoxin contamination in US maize was used, known from thousands of US Department of Agriculture analyses. Application of a hypothetical 20 µg/kg standard to that distribution resulted in rejection of 4 % of the maize crop and a mean aflatoxin level in US maize of 0.91 µg/kg. Imposing the stricter hypothetical standard of 10 µg/kg resulted in rejection of 6.2 % of the samples to achieve a mean aflatoxin contamination of 0.58 µg/kg, a drop of only 0.33 µg/kg.

To calculate overall population risks based upon the prevalence of hepatitis B infection in various regions, JECFA took two examples of different populations for comparison (13).

In one example, an area with low contamination of food by aflatoxins and with a population having a small prevalence of carriers of hepatitis B was chosen: aflatoxin levels based on European monitoring of aflatoxin B₁ in peanuts, maize and their products were used, and a population with 1 % carriers of hepatitis B was assumed. From the potencies given earlier, this yielded an estimated average population potency of 0.013 cancers per year per 100000 population per ng aflatoxin per kg body weight per day. Based on European monitoring, if all lots with contamination above 20 µg/kg are removed and it is assumed that these foods are ingested according to the "European diet", the mean estimated intake of aflatoxin is 19 ng per person per day. Assuming an adult human weight of 60 kg, the estimated population risk is 0.0041 cancers per year per 100000 people. If a 10 µg/kg hypothetical standard is applied, the average aflatoxin intake is 18 ng per person per day, resulting in an estimated population risk of 0.0039 cancers per year per 100000 people. Thus, reducing the hypothetical standard from 20 µg/kg to 10 µg/kg yielded a drop in the estimated population risk of approximately two additional cancers per year per 10⁹ people, well beyond the level of detection (13).

The second example pertained to areas with higher contamination. For these purposes, Chinese data on aflatoxin B₁ in peanuts, maize and their products were used and areas with a larger population fraction as carriers of hepatitis B (in this case, a population with 25 % hepatitis B carriers was assumed). The estimated potency for this population is 0.083 cancers per year per 100000 people. Using 20 µg/kg and 10 µg/kg hypothetical standards and the "Far Eastern" diet, the average estimated intake was 125 ng aflatoxin per person per day yielding an average population risk of 0.17 and 0.14 cancers per year per 100000 people respectively. Thus, reducing the hypothetical standard for this population from 20 µg/kg to 10 µg/kg yielded a drop in the estimated population risk of 0.03 cancers per year per 100000 people (13). This is a greater decrease in risk, but still barely detectable.

Food Safety Objective

In the case of a chemical toxin such as aflatoxin, the limits set by a country for aflatoxins in foods can be logically considered also to have the status of a Food Safety Objective (FSO). If it is accepted that a maximum permitted level established within a country is equivalent to an FSO, then by 1990 each major country importing or exporting peanuts had established a *de facto* FSO set, not on the basis of risk analysis, but on more pragmatic approaches.

The examples shown above, and others, enabled JECFA to show that reducing the permitted concentration of aflatoxins in foods, from 20 µg/kg to 10 µg/kg, had only a very marginal effect on the level of cancers produced, i.e. that little or no case existed for levels of aflatoxins in international trade being set below 20 µg/kg. Eventually it was recommended to the Codex Alimentarius Commission that the maximum permitted level for total aflatoxins in foods in international trade should be set at 15 µg/kg. If Codex adopts this recommendation, and it is accepted that the

FSO in this case is equal to that limit, then an FSO of 15 µg/kg has been established for peanuts in international trade.

This example has shown the kind of deliberation, which took place in, firstly, carrying out risk assessments for aflatoxins in peanuts, secondly, developing an ALOP for aflatoxins for various populations and, thirdly, establishing an FSO (although this term has not been used for it). This FSO is considered to be technologically achievable by major exporting countries, including the United States, China and Australia, but is currently out of reach of a number of producing countries in the tropics.

Meeting the Food Safety Objective

The Food Safety Objective is expressed as: $H_o - \Sigma R + \Sigma I \leq FSO$
(See text of Martin Cole in this journal.)

Initial level of contamination (H_o)

In bad seasons, i.e. seasons with severe drought stress in the 2–3 weeks before harvest, aflatoxin may be formed in peanuts before they are pulled from the soil. In regions where dry land farming is practised and irrigation impossible, good farm management cannot overcome this problem. Good agricultural practice can assist in limiting aflatoxin formation, but not in complete prevention. Aflatoxin is also formed while peanuts are being dried, which is usually carried out in the field, and can also continue during farmer storage if drying has been inadequate. Control of the initial level of contamination (H_o) is not possible with current knowledge.

Increase during storage and processing (ΣI)

Provided peanuts are adequately dried on the farm, and maintained in the dried state during transport and storage, the fungi which produce aflatoxins cannot grow, so $\Sigma I = 0$. However in some tropical countries, where high humidity conditions prevail and storage conditions are inadequate, aflatoxins can continue to be formed. A positive value for ΣI is to be avoided.

Reduction in levels during processing (ΣR)

The major method used for reducing aflatoxin levels in peanuts is colour sorting. In this procedure, nuts are inspected individually by electronic or laser sorting systems, and discoloured nuts removed. The rationale for aflatoxin reduction by colour sorting is that the growth of a fungus in a peanut results in discolouration, so removal of discoloured nuts sorts out those containing aflatoxins as well. In the United States and Australia, it is standard commercial practice that every individual shelled peanut entering commercial streams has been colour sorted. If the colour sorting process is ineffective, as can occur when severe drought stress causes peanuts to commence drying in the soil before harvest, it is common practice to blanch

peanuts to remove their skins, then roast and colour sort them again. This accentuates the darkening process and facilitates colour sorting.

Performance objective

The performance objective in peanut processing is to use colour sorting and other procedures as necessary to reduce the levels of aflatoxins in peanuts so that assays on representative samples indicate an acceptable level of $<15 \mu\text{g/kg}$ has been achieved consistently. In Australia, the Performance Objective used by one peanut shelling company is to sort peanuts until the mean aflatoxin content of samples from any one lot does not exceed $3 \mu\text{g/kg}$; this provides 95 % confidence that any lot will meet the $15 \mu\text{g/kg}$ FSO.

Summary

Aflatoxins in peanuts provide an example of how a Food Safety Objective can be derived for a chemical hazard (of biological origin) in foods. Aflatoxins are potent liver carcinogens, treated as food contaminants by regulatory agencies for many years. This paper shows the many steps used to set maximum limits for aflatoxins in foods in international trade. These steps are applied here to the development of a Food Safety Objective, linked to the existing maximum limit recommended by the Codex Alimentarius Commission.

Zusammenfassung

Aflatoxine stellen ein Beispiel dar, wie ein Food Safety Objective für eine chemische Gefahr (biologischen Ursprungs) in Lebensmittel abgeleitet werden kann. Aflatoxine sind potente Leberkarzinogene welche seit langem vom Gesetzgeber als Lebensmittelkontaminanten angesehen werden. Dieser Beitrag beschreibt die verschiedenen Schritte, die durchlaufen werden, um Grenzwerte für Aflatoxine im internationalen Handel festzulegen. Diese Schritte werden hier verwendet, um ein Food Safety Objective zu entwickeln, welches mit der vom Codex Alimentarius empfohlenen, existierenden Empfehlung verbunden ist.

Résumé

Les aflatoxines représentent un exemple de l'établissement d'un Food Safety Objectives pour un danger chimique (d'origine biologique) dans une denrée alimentaire. Les aflatoxines sont des carcinogènes puissants considérés depuis longtemps par les Autorités comme contaminants alimentaires. Cette contribution décrit les différentes étapes retenues lors de l'établissement de valeurs maximales pour le commerce international. Ces étapes sont utilisées ici pour développer un Food Safety Objective, lié aux valeurs maximales telles que recommandées par le Codex Alimentarius.

Key words

Aflatoxins, food safety objectives, performance objective, limits

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Address of correspondent: John I. Pitt, Food Science Australia, PO Box 52, North Ryde, NSW 1670, Australia, john.pitt@foodscience.afisc.csiro.au