Risk on the Table

Food Production, Health, and the Environment

Edited by

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CHAPTER 5

Risk on the Negotiating Table

Malnutrition, Mold Toxicity, and Postcolonial Development

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In September 1977, delegates from forty countries and sixteen international organizations gathered in Nairobi to tackle an urgent environmental problem of the increasingly interconnected world. Raj Malik, an official of the Food and Agriculture Organization (FAO), observed that "the world has become too interdependent to entertain a notion that any country or its produce are immune to the ill effects of mycotoxins."[1] These toxic substances, which were excreted by molds that grew on peanuts, maize, and other crops, could cause acute poisoning, cancer, and death. James Charles Nakhwanga Osogo, Kenya's minister of health, explained in his opening address that the conference took place "in one of the areas of the world where mycotoxins can represent a real hazard to the quality and quantity of human food and feed," suggesting that the substances did not pose the same hazard to every country.[2] Moreover, mycotoxins were not only a threat to food safety and security in Kenya and other tropical countries. Osogo added that the mycotoxin problem was "another specific deterrent to international trade in the produce of developing countries, since exports containing mycotoxins are sometimes found and this results in the destruction of specific shipments and often loss of entire markets and valuable foreign earnings."[3] Mycotoxins jeopardized developing countries' capacities to feed their populations and capabilities to export produce during a period when these countries burst onto the global scene, demanding a New International Economic Order (NIEO). This proposal challenged the hegemony of the North Atlantic powers and sought to reshape global trade in favor of the Global South.[4] The problem of mycotoxins was a new factor in the postcolonial struggle between

industrialized and developing countries, putting health and incipient environmental politics on the table.

Scientists, food and feed manufacturers, and administrators at national and international agencies had struggled over the best solution to the problem of mycotoxins since 1960, when veterinarians isolated a previously unknown substance that caused the death of hundreds of thousands of turkeys on British farms. British and US scientists discovered that *Aspergillus* molds on peanuts in the birds' feed produced this substance and named it aflatoxin. Scientists around the globe started to study aflatoxin's effects on humans and animals and to develop detection methods for aflatoxin in crops, making aflatoxin the best studied mycotoxin. They found aflatoxin not only in peanuts but also in corn, cottonseed, and many other crops that were important foodstuffs, animal feed, and export commodities in the recently decolonized nations in Africa and Asia, creating the very problems that Osogo described at the Nairobi conference. At that meeting, Peter Thacher, a representative of the United Nations Environmental Program (UNEP), proposed a strategy to resolve the mycotoxin crisis: "UNEP wishes to assess the risks in more precise terms so that governments can have a solid basis on which to make decisions within their own values as to the level of risk they wish to accept. We believe that by helping to improve assessments of the risks we will reduce risks, and can reduce interferences to international trade and consequent economic hardship. We are very anxious to assist the exporting developing countries."[5] UNEP concentrated on risk assessments to solve the aflatoxin crisis. The UNEP proposal limited UNEP's role in conducting these assessments, while leaving decisions about the supposedly objective level of risks to be taken to national governments. UNEP thus proposed a separation of risk assessment, delegated to the international level, from value-laden political decisions about implementation left in the realm of the nation state. UNEP assumed that its work of improving risk assessments would on its own reduce health hazards and economic hardship. UNEP drew on the work of scientists and regulators who had advanced risk as the primary way to understand and solve the aflatoxin problem since 1960. However, the proposed remedy, risk assessment, was itself laden with moral assumptions.

This chapter examines how scientists and officials came to see aflatoxin through the lens of risk, from the discovery of aflatoxin in 1960 to the

aftermath of the Nairobi conference, describing changing assumptions and limitations of the risk framework. I show how experiments with laboratory animals made aflatoxin into a perceptible hazard and how colonial and Cold War preoccupations with hunger and malnutrition influenced risk calculus. The risk framework espoused a zero-sum logic that justified the continued exposure of vulnerable populations in Africa to aflatoxin, sidelined developing countries' grievances about inequitable trade relations, and failed to protect vulnerable populations in Kenya. Relations between Global North and South played a central role in the calculus of aflatoxin risk. I argue that risk was decisively shaped by the international politics of decolonization during the 1960s and 1970s, when the "Third World" challenged the existing global trade system and the industrialized countries experienced "the shock of the global."[6] Researchers and officials at international organizations advanced risk as an answer to this challenge, ultimately re-entrenching global inequalities and furthering economic hardship for the developing countries.

This chapter thus challenges the narrative that risk played a role only in affluent industrial societies.[7] Sociologist Ulrich Beck argued in *Risk Society*, published in 1986, that risk became the defining feature of the new era of reflexive modernity, which began in the second half of the twentieth century.[8] In Beck's narrative, modernity, dominated by a logic of wealth production through techno-economic "progress," was succeeded by reflexive modernity, when skepticism about industrialization's scientific and technological foundations abounded, and the focus of politics shifted to risks that these very foundations generated.[9] Beck writes, "The driving force in the class society can be summarized in the phrase: *I am hungry!* The movement set in motion by risk society, on the other hand, is expressed in the statement: *I am afraid!*"[10] However, aflatoxin has contaminated foodstuffs essential for the hungry person's survival and for economic development through exports, and aflatoxin gave rise to fears about food contamination. This history of aflatoxin collapses the distinction between the "hungry world" and risk society.[11]

Aflatoxin became a problem for both worlds, intertwining them in new ways that facilitated the erasure of developing countries' economic demands. Risk combined health and wealth in one framework, which enabled scientists to move between the two domains and reshuffle them. Political demands for market access and stable commodity prices were

reframed as questions of standards and acceptable risk levels, thus transforming political disputes about wealth and power into technical questions about seemingly objective levels of risk to health. These technical questions were to be answered by scientists and experts. They formed "epistemic communities" that detected and assessed risks to make what were ultimately political decisions, becoming a "fifth branch" of government. However, epistemic communities did not necessarily give unanimous advice.[12] Drawing on Sheila Jasanoff's insights into the political context of risk assessment and the displacement of political struggles into the realm of technical advisory boards, the chapter describes the process of reshuffling scientific questions and political demands on the level of international institutions in the wake of decolonization.[13]

Risk facilitated a division of responsibilities between international organizations and nation states, which differed vastly in their capacities to deal with toxicity in food and the environment.[14] For example, international organizations conducted risk assessments of aflatoxin, which left the implementation to nation-states and shifted the burden of global trade regulations onto the developing countries by excluding questions of economic justice. By considering aflatoxin risk across affluent risk societies and the hungry world, this chapter shows how the risk framework was also a process in scale-making that shaped political possibilities on the levels of nation-state and international organizations.[15] In addition to the FAO and UNEP, all major agencies of the United Nations (UN)—the World Health Organization (WHO) and the United Nations International Children's Emergency Fund (UNICEF)—as well as the General Agreement on Tariffs and Trade (GATT) and its successor, the World Trade Organization (WTO), have been concerned with aflatoxin at some point. The international institutionalization of risk resembles the conceptual structure of international trade law, such as the GATT, which enshrined markets and movement of capital, protecting capital from demands for redistribution that national mass democratic movements and anti-colonial nationalists might voice.[16] Similarly, the international legal framework of human rights excluded questions of economic justice.[17] The lens of risk through which scientists and international agencies viewed aflatoxin circumscribed the possibilities of toxicity control in narrow ways, supporting a global system that paved the way for the expansion of markets for industrial chemicals within Europe and from the Global North, driving proliferation and

harmonization of international legal regimes to regulate toxic risks in food and the environment since the 1950s.[18]

This chapter advances its argument about the centrality of North-South relations in shaping the theory and practice of aflatoxin risk in three main parts. Each part considers a different commodity that aflatoxin contaminated: peanuts as remedies for malnutrition, peanuts as export goods, and maize as staple food. The first part shows how malnutrition became a political concern that the UN Protein Advisory Group (PAG) sought to solve with peanuts as an additional source of protein. The PAG responded to the discovery of aflatoxin by determining a contamination level that allowed the continued use of peanuts, developing a calculus of aflatoxin risk. Second, I discuss how industrialized countries introduced restrictions on the import of goods potentially contaminated with aflatoxin. Developing countries challenged these measures at the UN Conference on Mycotoxins in 1977 in Nairobi, bringing together two pressing developments in international politics during the 1970s: the NIEO and environmentalism. Finally, I show how the risk-based regulatory approach failed to protect populations most vulnerable to aflatoxin, when an outbreak of acute aflatoxin poisoning in the staple food maize killed twelve people in 1981. It would not be the last outbreak of acute aflatoxin poisoning in Kenya. Together, these histories suggest that the risk-based approach ultimately evaded questions of economic justice and accountability, failing to protect the health and livelihoods of the most vulnerable people in Africa and Asia.

Sentinels for Toxicity, ca. 1960

In May 1960, farmers in southern England noticed that their turkeys suffered from a strange illness. A feed company sent veterinarian William Blount, who observed that "the birds move slowly, do not feed or drink, sink to the ground, fall quietly over on to one side, stretch out their limbs and die. It is as though the birds were in a gas chamber, and dying from the effects of some poisonous, lethal gas!"[19] Blount compared the sites of the outbreak to find common factors and thereby identify the cause of "Turkey X" disease. He discovered that the disease was caused by feed that had been produced in a specific feed mill from specific peanut batches imported on specific vessels from Brazil. Peanuts—which were imported not only from

Brazil, but also from other subtropical and tropical Asian, African, and American countries—constituted the protein-rich part of the feed. These protein-rich feeds made birds gain more weight and gain it faster, enabling the rapid expansion of poultry farming in postwar Great Britain. Veterinarians, chemists, and mycologists studied the contaminated peanut batches, ultimately learning that *Aspergillus* molds excreted a poisonous molecule, which they named aflatoxin.

The researchers discovered aflatoxin in the highly artificial environment of the industrialized poultry farm. The peanuts of the contaminated feed had been stored and transported across the Atlantic before being processed into homogenous poultry feed. This process spread the toxic substance, which might have been on a few single peanuts, across large batches of feed, in a dose sufficiently high to kill a turkey, which, as a species, happened to be highly susceptible to aflatoxin. The birds became an ideal "sentinel" for a toxic substance so concentrated and distributed as to cause a visible hazard.[20] If a handful of birds had ingested a few toxic peanuts, farmers might have dismissed the dead birds as a common occurrence in poultry-keeping, in which some mortality was expected. In 1960, feed manufacturers accidentally spread aflatoxin across feed batches and industrialized farms in a way that exposed an entire avian population to levels of toxicity so high as to make their mass mortality an abnormal phenomenon, which farmers noticed and which scientists investigated with epidemiological methods. It was not only scientific methods but also industrialized agriculture and the global commodity trade that led to the discovery of aflatoxin.

The Shock of the Malnourished World

The discovery of a toxic substance in peanuts alarmed UNICEF, which was promoting peanut flour as a remedy for infant malnutrition. Aflatoxin threatened the use of this remedy in the intensely political fight against malnutrition. Malnutrition was the latest manifestation of hunger, which had transformed from an inevitable natural condition into an object of statecraft in early nineteenth-century Britain.[21] In the late nineteenth century, nutrition science began to recast hunger in the language of molecules, calories, and other calculable entities; the discourse of nutrition was embraced by states, colonial rulers, and anti-colonial activists alike,

offering a new scientific language to advance and criticize government programs.[22] Nutrition became an object of interwar colonial and international statecraft for its potential to unsettle colonial rule and international relations amidst mounting fears about unfettered population growth.[23] In the interwar years, British nutrition scientists "discovered" malnutrition, the lack of specific nutrients, in East Africa.[24] British colonial rulers, the Rockefeller Foundation, and other organizations embraced the concept of malnutrition, shifting the focus from outwardly political questions of poverty and undernourishment to educational and technical interventions to train the population of the malnourished world in proper dietetics.

Hunger, food, and malnutrition did not disappear with World War II. In the context of the Cold War, the US government, supported by the Rockefeller and Ford Foundations, promoted a program of agricultural reform in Asia—later called the Green Revolution—to increase food production, fill the stomachs of hungry peasants, win their hearts and minds, and close their ears to the siren songs of communism.[25] In the late 1940s, the recently founded United Nations and its specialized agencies began to focus on malnutrition, especially kwashiorkor, in Africa. Kwashiorkor had been first observed by physician Cicely Williams in West Africa in 1933. She found that milk could cure the disease of infant malnutrition, leading her to speculate that lack of protein played a role in causing kwashiorkor.[26] Kwashiorkor remained a topic of discussion at meetings of a joint committee of nutrition experts of the World Health Organization and Food and Agriculture Organization.

In 1955, the World Health Organization established a new expert group, the Protein Advisory Group (PAG), to advise the organization on its protein-rich foods program and on new sources of protein in the fight against protein malnutrition.[27] Many prominent food and nutrition scientists from around the globe, including Nevin Scrimshaw (US), Coluchur Gopalan (India), and Benjamin S. Platt (UK), joined the PAG.[28] The PAG received funding from different US philanthropic organizations, including the Rockefeller Foundation, which funded a subgroup in protein malnutrition.[29] The PAG met regularly, developed recommendations, and issued bulletins, attempting to make the case to the international organizations that the problem of protein malnutrition was "a primary factor in susceptibility to infection and impaired growth and development of

children in many developing countries" and "a primary deterrent to national social and economic development."[30] The PAG sought to fill the "protein gap," the perceived scarcity of protein, by tapping unconventional protein sources.

Peanuts featured prominently, and controversially, in this plan. Peanuts had been important cash crops in British and French African colonies since the late nineteenth century, including the infamous Groundnut Scheme in Tanganyika (present-day Tanzania), which the British government and Unilever advanced in the 1950s.[31] The PAG estimated that peanuts could provide 10 percent or more of the optimum human protein requirement (40 to 70 grams per day) in West Africa.[32] Hunger and malnutrition were thus subject to a calculus of food. While the use of groundnuts was not without critics—for example, Gopalan considered peanuts inferior to milk and vegetable proteins that UNICEF had used in nutrition programs in postwar Europe—the group continued to focus on peanuts for filling the protein gap, fighting protein malnutrition, and thus removing what they believed to be the primary obstacle to development.[33] The causes and remedies of the hungry world themselves were up for grabs.

Saving the Peanut

In fall 1961, the PAG learned that there was "a fungus among us."[34] The fungus excreted a poisonous substance and contaminated peanuts, which the PAG was promoting as a remedy for malnutrition. At the PAG meeting in 1962, members deliberated about the consequences of this discovery.[35] They set out to study aflatoxin's effects on human bodies, to develop detection methods, and to devise ways to eliminate aflatoxin contamination. The PAG wanted to be in a position to advise "the agencies as to desirable procedures and precautions to be observed in utilizing foods derived from materials subject to this kind of fungal attack."[36] The PAG would primarily use published and unpublished reports to determine aflatoxin hazards and strategies to limit exposure.

Moreover, the PAG sought to determine the hazard that aflatoxin caused in animals. They found that "insufficient information was available to allow setting of a tolerance level."[37] Instead, the PAG recommended that only peanuts that were free of toxicity should be used for human nutrition. They recommended using a duckling test that British veterinarians had first

developed. A researcher would inject a sample in a duckling's esophagus and observe the reaction. If the duckling died immediately, the sample was strongly toxic. If the duckling did not die, the researcher would kill the bird after several days and examine the liver for lesions, whose presence indicate a weaker toxic potency. This test made aflatoxin visible as a direct physical harm to an animal, a duckling of the Khaki-Campbell breed. If the sample harmed a duckling, the batch contained aflatoxin and was unfit for human consumption. This test assumed that it could either detect the smallest possible amount or that ducklings were at least as sensitive as humans, permitting the continued use of peanuts in malnutrition programs.

There were alternatives to the PAG approach of justifying the continued use of peanuts. In fact, two companies that produced peanut-based nutrition supplements followed a different strategy: eliminating peanuts from food. The British pharmaceutical company Glaxo, which collaborated with the PAG, withdrew the peanut-based nutritional supplement "Amana" in late 1961. Glaxo gave the supplement's disappointing sales as the official reason. In fact, Glaxo found that most of the peanut supply was contaminated by aflatoxin and admitted privately to nutrition experts that uncertainty about the safety of the raw materials was the real reason that they withdrew the product. They said that the company "as a responsible manufacturer [was] not prepared to continue its manufacture and supply."[38] Similarly, Nestlé changed the formula of its high protein food in 1962, replacing peanuts with soy to avoid the presence of aflatoxin.[39] While industry pursued an alternative strategy, suggesting that peanuts were not an economic necessity, the PAG remained wedded to peanuts, developing a risk-based justification to continue this strategy.

In 1963, the PAG reaffirmed its recommendation that peanuts that passed the biological test could be used in nutrition programs. British researchers developed a physiochemical test for aflatoxin, using the recently introduced thin-layer chromatography (TLC). TLC was an analytical method to determine the molecular composition of a solution. Molecules were separated on a strip of adsorbent material according to physical and chemical properties. Each type of molecule produced a distinct spot, which was compared to a reference spot produced by the known substance.[40] However, the PAG was concerned that the test was not specific enough, and any batch of peanuts that tested positive on the TLC had to be subjected to

the duckling test.[41] The decisive test was still whether a sample produced visible harm or lesions in an animal.

Scientists' animal experiments raised questions about how to extrapolate these findings to humans. Researchers at the Unilever Research Laboratory observed in 1961 that rats fed with contaminated peanut meal did not develop acute poisoning but liver tumors.[42] This observation prompted researchers to consider whether there was a link between aflatoxin exposure and the high liver cancer rates that scientists had noticed in Africa since the 1920s.[43] While cancer researchers generally assumed that carcinogens in even the smallest amounts could cause cancer, the available records show no discussions about this question at PAG meetings. In 1963, the PAG maintained that "there is no proof that these compounds have any harmful effect on human beings."[44] Michael Latham, an American nutritionist who did research in Tanganyika in 1963, criticized this lack of knowledge. He lamented that aflatoxin research focused on animal feed, imported products, and peanut trade—economically important for European countries—but failed to study possible effects on people in developing countries.[45]

The PAG researchers relied on animal studies to make their recommendations. They quantified aflatoxin's effects by correlating amounts of aflatoxin to specific bodily effects in ducklings and monkeys. For example, they observed that no effects were observed in ducklings for amounts smaller than 0.005 parts per million (ppm) in the diet, and results tended to be negative for aflatoxin concentration below 0.1 ppm. In monkeys, there were no "gross effects" at 0.1 ppm, 0.6 ppm caused some deaths, and higher levels would kill all monkeys.[46] These studies revealed some of the limits of such animal tests. The duckling test could not detect amounts of aflatoxin that were smaller than 0.03 to 0.05 ppm.[47] Moreover, the scientists observed that test results depended on many factors, such as the choice and preparation of a sample, or the breed, species, and age of the animal.[48] Using a seemingly simple test with live ducklings to eliminate contaminated peanut batches appeared increasingly questionable.

In the mid-1960s, the PAG members realized that aflatoxin contamination was much more widespread than initially assumed. There were hardly any sources of uncontaminated peanuts. Convinced of peanuts' nutritional value to many people, the PAG concluded that "therefore, some level of aflatoxin in the protein-rich foods and food mixtures with which the Group is concerned must be accepted if these supplements are to continue

to play a role in improving human nutrition."[49] The PAG was determined to set a level of acceptable aflatoxin contamination because the importance of peanuts outweighed the hazard of aflatoxin.

The PAG proposed a level of 0.03 ppm of aflatoxin in human diets. This number corresponded exactly to the detection limit of the biological duckling test. The PAG provided a biological justification for this figure based on experiments with monkeys, which Glaxo conducted in its laboratories. These experiments revealed a sharp difference between the amount of aflatoxin that produced a change in a monkey's liver and the amount that produced no observable changes:

> The "no effect" level in monkeys is 0.3 ppm, which is the equivalent of a daily intake of 0.015 mg aflatoxin/kg body weight. Applying a safety factor of 50, a dose of 0.0003 mg/kg body weight would be an ineffective daily dose for people. This calculation means that for an infant weighing 10 kg, 0.003 mg aflatoxin per day would be a dose likely to be without effect on the liver. From this it can be calculated that a level of aflatoxin in the protein supplements of which 100 g (or 0.1 kg) is eaten should not exceed 0.03 mg/kg or 0.03 ppm.[50]

The researchers emphasized that "it is not possible to draw any conclusions from these experiments about the susceptibility of man to the toxic or carcinogenic action of aflatoxin," but they did exactly that by choosing a safety factor that confirmed the initial PAG recommendation of not using any food that tested positive in the duckling test.[51]

The PAG had maneuvered itself into such a position where it faced the stark choice between solving the problem of protein malnutrition through contaminated supplements or not having enough protein for malnourished children. The PAG justified its choice of the former:

> Although the Group would prefer to impose a lower level of aflatoxin in the foods and food mixtures concerned in order to provide a wider margin of safety, it believed that there was an even more urgent need to provide extra protein in some parts of the world so as to prevent malnutrition and starvation. These considerations outweighed the desirability of introducing measures for reducing a hypothetical health hazard by limitations which were difficult to enforce under current agricultural practices and techniques of food processing.[52]

However, the reaction by other actors, such as Glaxo or Nestlé, which substituted groundnut with other sources of protein, suggested that this was a false dilemma. There were other sources of protein. Nonetheless, the PAG stood by its recommendation, reconfirming it in 1967 and again in 1968, 1969, and 1972.[53]

In 1973, the PAG declared its approach a success in hindsight, because the "pioneering evaluation of the toxicological hazards of aflatoxin to young children in relation to their urgent need for dietary protein apparently became the basic philosophy for much regulatory action in this area subsequently by a number of governments."[54] Judging from the available records, the PAG did not discuss actual amounts of peanuts needed, but assumed that stricter limits would reduce this amount so much as to trigger a protein crisis, a crisis of malnutrition that was always already looming. Historians and anthropologists have shown how colonial rule, settlement schemes, agricultural programs, and postcolonial policies have shaped food supply and crop choice in Kenya.[55] The protein crisis perhaps justified exposing vulnerable populations to aflatoxin through a calculus of risk.

The risk assessment of aflatoxin in nutritional supplements was grounded as much in the material culture of laboratory research and animal experiments as in the late colonial and early postcolonial projects of nutritional studies and aid. The calculus of risk assessment justified the exposure of populations in the developing countries to toxic substances for their supposed benefit. The PAG, however, eschewed taking responsibility for its recommendations, stating that "the ultimate responsibility for the use of these products in human nutrition programs must rest with the governments of the territories concerned."[56] The PAG provided a justification for giving contaminated food to children and infants who were already suffering from malnutrition. This strategy set a precedent for UNEP and other international agencies that would provide risk assessment, ridden with assumptions of what counted as hunger, malnutrition, and food resources. If countries with unequal resources and expertise were left to draw their own conclusions from these assessments, they would reproduce and even exacerbate existing inequalities.

Aflatoxin and Risk Assessment in the US Federal Government

This kind of zero-sum reasoning was not limited to the PAG and the context of peanuts as nutritional supplement for the global poor. The US Food and Drug Administration (FDA) in 1978 conducted its first formal risk assessment on aflatoxin. This risk assessment justified not lowering the threshold level of acceptable aflatoxin contamination, because there would be little effect on health, and lowering the threshold would result in high

economic losses. The FDA employed a *de minimis* approach for aflatoxin and other contaminants that it considered to be unavoidable food additives. This approach permitted contamination up to a certain threshold even by substances known to be carcinogenic in the tiniest amounts.[57] In 1965, the FDA set an action level of 30 parts per billion (ppb) total aflatoxin, based on the sensitivity and reliability of the available detection methods.[58] In 1969, this action level was lowered to 20 ppb. In 1974, the FDA proposed a tolerance of 15 ppb considering "the consequences of possible levels above zero."[59] The FDA took into consideration higher prices, unavailability of peanuts, which were "generally considered a highly nutritious and useful food," manufacturers' capability, and lack of "direct evidence that aflatoxin causes cancer in man or of what may be the level of no effect."[60] The FDA assumed without quantitative analyses that manufacturing costs were higher and that peanuts were an almost irreplaceable food.

In 1978, the FDA employed a quantitative risk assessment to justify the tolerance level of 15 ppb. Aflatoxin was one of the first toxic substances in food to be subjected to such a formal exercise.[61] This assessment determined the theoretical effects of lowering the threshold by 5 ppb on rates of human liver cancer. This risk assessment relied on the correlation between aflatoxin exposure and liver cancer occurrence that British researchers had established in a study in Kenya in the late 1960s.[62] Ultimately, the FDA withdrew its proposal for a tolerance level, a regulatory action that required a formal administrative hearing, and has maintained the action level—a less rigorous, informal regulation—of 20 ppb to the present day.[63]

For the PAG and the FDA, risk assessments hinged on classifying aflatoxin as an unavoidable additive. For the PAG, this classification legitimized the continued use of peanuts contaminated at low but possibly harmful dosages. For the FDA, this classification enabled a regulatory intervention under the current law. While the FDA considered manufacturers and consumers, the PAG imagined the nutritional needs of people in the developing world. Risk assessments were not limited to places without material scarcity.

Mycotoxins, Environments, and the Third World Moment

Many national and supranational agencies set aflatoxin regulations in the late 1960s and 1970s with consequences for global trade, especially for developing countries' ability to export peanuts to Europe. For example, the International Agency for Research on Cancer (IARC), an agency of the WHO, found in 1972 that "increased frequency of liver cancer has been recorded in populations consuming diets contaminated by aflatoxins and possibly other mycotoxins, but no causal relationship has been established," giving credence to the thesis that aflatoxin was a possible human carcinogen.[64] In 1973, the European Economic Community (EEC), a major peanut importer, introduced a directive "on the fixing of maximum permitted levels for undesirable substances and products in feedingstuffs."[65] Such regulations restricted trade between Global North and South during a period in which these relations were intensely politicized.

Peanuts were one of the most important export commodities for countries such as Senegal, The Gambia, and Nigeria. Peanut commerce constituted 60 percent of the national incomes of The Gambia and Niger, 20 percent for Senegal and Sudan, and 5 percent for Mali and Nigeria. In Senegal, more than 70 percent of the population worked in the groundnut industry. The countries formed the African Groundnut Council (ACG) in 1964 in order to, among other things, "ensure reasonable prices for their products," and stabilize "prices of groundnut in the world market at remunerative level."[66] Peanut exports were threatened not only by aflatoxin but also by the competition from other oilseeds, such as soy. In April 1971, Senegal's president Léopold Senghor gave a speech at an EEC-African summit on the peanut problem, arguing that "peanut oil . . . is constantly meeting stiffer competition from various kinds of vegetable oil powerfully supported by different types of national guarantee and support systems. The production in poorer countries cannot and for a long time will not be able to have the same advantage."[67] Senghor viewed the economic trade situation not just as one of peanuts but of all oil seeds, some of which were produced and promoted by other countries, such as soy from the United States. In France, groundnut feed was being replaced by more expensive soybean cakes, which were primarily produced in the United States. Aflatoxin contamination had played a role in this shift.[68] The strict EEC directive of 1973 caused problems for many oilseed exporting countries.

A coalition of these decolonized nations had been developing a proposal for a New International Economic Order (NIEO) since the mid-1960s.[69] The

NIEO sought to overhaul the global liberal economic order by giving decolonized states sovereignty over their natural resources, control over raw material prices and commodity exports, access to markets in developed countries, and sufficient food supply.[70] These radical reforms would enable the postcolonial countries to gain real independence. The end of the Bretton Woods system in 1971 and the oil embargo by the Organization of Petroleum Exporting Countries (OPEC) in 1973 empowered the countries of the "Third World" to make a decisive push for the NIEO at the sixth special session of the UN General Assembly, which adopted the proposal. The program of action included a call to "promote exports of food products of developing countries through just and equitable arrangements, inter alia, by the progressive elimination of such protective and other measures as constitute unfair competition."[71] These measures included tariffs as well as nontariff barriers, which, for example, restricted import for health reasons.

Concern about invoking environmental pollution as a pretext to restrict market access had already been raised at the UN Stockholm Conference on the Human Environment in 1972. The conference passed a resolution that "the burdens of the environmental policies of the industrialized countries should not be transferred, either directly or indirectly, to the developing countries."[72] In the event that stricter environmental standards would result in restricting trade or negatively affecting exports, compensations should be worked out "within the framework of existing contractual and institutional arrangements."[73] Moreover, the conference suggested "internationally coordinated programmes of research and monitoring of food contamination by chemical and biological agents" be established by the FAO and WHO. The results from these programs should be used to provide early information about rising contamination levels, especially those "that may be considered undesirable or may lead to unsafe human intake."[74] The conference established the UN Environment Programme, which would pursue this goal. Further, it was recommended that the Codex Alimentarius Commission, an international body for harmonizing food regulations, be supported to develop international standards for pollutants in food and a code of ethics for international food trade.[75] FAO and UN should assist developing countries in the field of food control.

The problem of mycotoxins emerged at the intersection of the NIEO and environmental governance, both key areas of international politics of the 1970s.[76] The UN Conference on Mycotoxins in 1977 in Nairobi was the

central forum for discussing this problem. Aflatoxin regulations by the EEC, Japan, and other importers had ramifications for all participants in the supply chain, including primary producers, middlemen, national exchequers, and consumers. Internationally, Osogo said, "potential problems would be the increased difficulty involved in arriving at sound international agricultural production adjustments and commodity and food security agreements."[77] The aim of the conference was to "establish an effective system of internationally agreed monitoring and control that would protect the consumer from exposure and that would safeguard the producer from unexpected, and sometimes unjustified, deprivation of the fruits of his labor and his investments."[78] While its concrete recommendations were mostly technical in nature, the conference made explicit the relationship between health and wealth that the risk calculations sought to obscure. Ultimately, the conference failed in both regards: consumers in Africa were unprotected, and producers in Africa could not sell their produce. The responsibility for the losses was ascribed to the exporter countries: "It was suggested that the risk of sending contaminated shipments was the exporter's and that it was a matter of contract and agreement between trading partners which analysis and sampling procedures were acceptable."[79] Risk was privatized.

Moreover, the conference called for regulatory harmonization through the Codex Alimentarius Commission because developing countries often lacked effective food control systems. It was concluded that "in the long run therefore, exporting countries must improve their food control systems, and surveillance and monitoring programmes to reduce contaminations." Still, the solution of the problems of balance of payments and the protection of public health with regard to mycotoxins would need to seem appropriate within "the context of the New International Economic Order and to ensure a fuller, safer, more wholesome food supply for mankind [*sic*]."[80] This approach, however, was never found or promoted. The final recommendations of the conference focused exclusively on technical assistance that would increase the "marketability" of food commodities and products through improved storage, monitoring and control, better training, and further research. Ultimately, the NIEO was not translated into actual changes on the level of policy, such as the formulation and control of risk. By 1981, the NIEO, which was already dissipating, received its final blow from US president Ronald Reagan, who refused to discuss any further

changes to the global economic structure.[81] Aflatoxin remained a problem for trade, leaving the burden of regulation on the producing countries of the Global South.

Risk on the Ground

While peanuts were used as nutritional supplements and as export commodities, maize was the central staple in East Africa.[82] Researchers found that aflatoxin could also contaminate maize. On the level of policy implementation, the UN agencies provided technical assistance to improve the system of food control in Kenya and other countries. However, this system neither reduced contamination in export goods nor protected a population, which relied primarily on subsistence farming and local markets. Setting threshold levels and enforcing them depended on systematic testing, which was difficult in an agricultural system without central grain collection. In 1981, the long rain season began on 17 March in Machakos, just east of Nairobi, Kenya. "The rest of the month," the crop officer wrote, "remained very wet with low temperatures."[83] Some areas of Machakos, mostly populated by the Akamba people, had been badly hit by food shortages. Soon, doves began to die, a family's dog perished, and the fish in the Athi River also died.[84] A few months later, in early June, a family was admitted to Makueni District Hospital. The family's eight members had fallen ill at the same time with fever, vomiting, loss of appetite, and weakness. After a week, they developed jaundice. All of them, except two unweaned infants, died.[85] Kenyan researchers visited the family's homestead, where they collected foodstuff, including corn, cowpeas, sorghum, millet, and vegetables. The grains had been stored by the "traditional Akamba method" in gourds and clay pots inside a granary that was raised above the ground.[86] The samples were brought to National Public Health Laboratory Service in Nairobi, where the researchers detected 3,200 ppb of aflatoxin in the corn samples. The researchers noted that "the previous year was extremely dry and, as a result, the harvest was poor, necessitating the storage of grains that might normally have been discarded as spoiled."[87]

After the Stockholm Conference, the FAO and UNEP had organized a program to address the problem of food contamination in Kenya, especially relating to aflatoxin. They trained local laboratory staff, prepared analytical

manuals, and established a library. Yet, one WHO consultant warned early on that "whereas economic interest in food production, especially food for export, will have dictated the necessary preventive measures, subsistence farmers likely to be at highest risk due to their primitive food storage conditions, are badly in need of protection against the hazards arising from aflatoxin intake."[88] The Kenyan government's focus had been on exports, so many of the efforts of aflatoxin control and sampling were centered on the ports.[89] Programs to monitor toxic substances in food and workplaces were often spotty, understaffed, and underfunded. The director of medical services appealed to his regional officers to "please help the *Wananchi* [citizens] with preventive aspects by taking regular grain samples in your Provinces—this should include also maize meal from the shops and from the maize mills themselves—and send these to the National Public Laboratory Services for analysis."[90] This laboratory, however, lacked essential chemicals. For example, in 1986, the Mycotoxin Laboratory of the National Public Health Laboratory Service analyzed a total of 109 food and feed samples, but "due to lack of essential reagents and chemicals which are required for aflatoxin analysis work has presently come to standstill."[91] As such the laboratory could illuminate the "edges of exposure" but not conduct a comprehensive surveillance program of food.[92] After the NIEO fizzled out in the early 1980s, international donor organizations imposed structural adjustment programs on African countries, resulting in the further defunding of health and other public services.[93] The highly abstract system of threshold levels, which were determined by risk assessment, required a complex laboratory infrastructure to take hold in the world. Without such infrastructure, the system reached its limits in the poor countries before it ever really began. For international regulators, the markets and consumers in the rich countries seemed to have mattered more than the decolonized nations and their farmers.

Conclusions: Risk under the Table

The history of aflatoxin regulation across rich and poor countries in the 1960s and 1970s reveals a complex landscape of expertise, international relations, and commodity trade. Aflatoxin with its twofold toxicity of acute poisoning and carcinogenicity became visible to scientific research in the artificial environment of the industrialized farm. The carcinogenic and toxic

effects of aflatoxin on humans remained an object of protracted epidemiological studies. The nutrition researchers of the PAG employed an approach of quantitative risk assessment to justify the continued use of potentially contaminated goods in food and nutrition programs in the face of a perceived scarcity of protein. This approach had ramifications for the global trade of potentially contaminated commodities that were primarily exported by developing countries.

This chapter shows that toxicity and food contamination were not limited to risk societies in affluent industrialized countries. Beck wrote that "the struggle [of the 'Third World'] against hunger and for autonomy forms the protective shield behind which the hazards, imperceptible in any case, are suppressed, minimized and, by virtue of that, amplified, diffused and eventually returned to the wealthy industrial countries via the food chain."[94] This chapter reveals a more complex story, in which hazards have not merely been suppressed and minimized in the "Third World," but the process of making visible, assessing, and quantifying risk crisscrossed Global North and South. For Beck, risks know no social or national differences. Wealth and health hazards cannot be contained within a nation-state in an "interdependent" or "globalized" world. Ultimately, Beck believes that only the UN would be in a position to tackle risks. However, this empirical study of the efforts of the UN and its agencies to tackle aflatoxin risk dampen this hope. Risk-based global regulation required a level of abstraction that removed questions of inequality of trade relations and that was impossible to translate into public health programs in resource-poor settings. The risk approach failed to increase exports and to protect the health of people in Kenya and other places, where grains were produced and consumed locally. The history of aflatoxin shows how the concept of risk has tended to mask the global distribution of wealth and health, raising the question whether risk itself, as a quantitative category, should remain on the table at all.

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Notes

1. Food and Agriculture Organization of the United Nations, *Report of the Joint FAO/WHO/UNEP Conference on Mycotoxins*, 3.
2. Ibid., 48.
3. Ibid.
4. Gilman, "New International Economic Order."
5. Food and Agriculture Organization of the United Nations, *Report of the Joint FAO/WHO/UNEP Conference on Mycotoxins*, 49.
6. Ferguson et al., *Shock of the Global*.
7. Building on Gabrielle Hecht's work on the market for nuclear materials and postcolonial power relations, this chapter shows how risk assessment in the United States and beyond was shaped not only by Cold War nuclear weapon planning but also by postcolonial North-South relations. See Hecht, *Being Nuclear*; Nash, "From Safety to Risk."
8. Beck, *Risikogesellschaft*.
9. Historians have called into question the sharp transition from class to risk society, adducing examples of earlier concerns about environmental destruction. See Bonneuil and Fressoz, *Shock of the Anthropocene*.
10. Beck, *Risk Society*, 49.
11. Cullather, *The Hungry World*.
12. The literature on the roles of scientific experts in politics is far too expansive to do justice to it here. The works of political scientist Peter Haas and STS scholar Sheila Jasanoff are starting points of this literature. Haas introduces the concept of "epistemic communities" to describe how experts shape governments' multilateral coordination and foreign policy decisions that appear opposed to state interests at first sight. Jasanoff introduces a nuanced analysis that shows that the scientists forming epistemic communities are neither as disinterested, nor acting necessarily as coherently, as Haas suggests. See Haas, "Introduction"; Jasanoff, *The Fifth Branch*; Jasanoff, "Science and Norms." For the role of lay knowledge in risk society, see Wynne, "May the Sheep Safely Graze?"
13. See, for example, Jasanoff, "Science, Politics, and the Renegotiation"; Jasanoff, "Songlines of Risk."
14. Vogel, *The Politics of Precaution*; Tousignant, *Edges of Exposure*.
15. Hecht, "Interscalar Vehicles."
16. Slobodian, *Globalists*.
17. Moyn, *Not Enough*.

18. The literature on standards, risk, and economic interests has taken different approaches to understand these interrelations. Most authors agree that risk-based standards implicitly configure a moral economy of relations. Proctor, Oreskes, and other show that industry actively produces ignorance by hiring "faux experts," who distort facts and cast doubt on scientific studies. Others, such as Nathalie Jas and Michelle Murphy, argue that measurements and standards have always already been shaped by specific regimes of perceptibility. For example, Jas describes how European scientists developed the measure of the "acceptable daily intake" (ADI) in the 1950s to take into consideration economic development in setting health standards. Graham Burnett has emphasized the role of epistemic and disciplinary allegiances in the regulation of whaling. This chapter draws on the last two approaches to understand aflatoxin regulation and standard setting as a reconfiguration of relations between countries and people. See Busch, "Moral Economy"; Murphy, *Sick Building Syndrome*; Boudia and Jas, "Introduction"; Proctor and Schiebinger, *Agnotology*; Oreskes and Conway, *Merchants of Doubt*; Burnett, *Sounding of the Whale*; Jas, "Adapting To 'Reality'"; Jas, "Gouverner les substances chimiques dangereuses"; Stoff, *Gift in der Nahrung*.
19. William Blount, "A New Disease of Turkeys," June 1960, Disease of Turkey Poults and Groundnut Toxicity (Aflotoxin) [*sic*], Ministry of Agriculture and Fisheries, The National Archives, Kew, UK: MAF 287/41.
20. Lakoff and Keck, "Preface."
21. Vernon, *Hunger*.
22. Cullather, "Foreign Policy of the Calorie"; Amrith, "Food and Welfare in India."
23. Amrith, *Decolonizing International Health*; Amrith and Clavin, "Feeding the World"; Bashford, *Global Population*.
24. Worboys, "The 'Discovery' of Colonial Malnutrition."
25. While the Cold War shaped US foreign policy, including funding for food and nutrition projects, the Cold War was not the primary lens through which experts in postcolonial Great Britain and African countries viewed agricultural and scientific problems. These experts were much more concerned with the problems that the division between Global North and South posed. For the history of US food policy in the Cold War, see Cullather, *The Hungry World*; Connelly, "Taking Off the Cold War Lens."
26. Tappan, *Riddle of Malnutrition*.
27. Ruxin, "United Nations Protein Advisory Group."
28. "Current and Past Members of PAG," PAG Bulletin, No. 7, April 1967, FAO Archives, Rome: 12-ESN-536.
29. At the same time, the Rockefeller Foundation promoted a molecular approach to the study of life that "aimed to map the pathways in the molecular labyrinth of the human soma and psyche in order to control biological destiny." Scientists studied the biological structure and function of proteins as part of these efforts. See Kay, *The Molecular Vision of Life*, 48.
30. FAO/WHO/UNICEF Protein Advisory Group, "The PAG: Its History, Function and Work Programme," 28 September 1971, Accession No. 18378, FAO Library.
31. See chapter 1 in Mueller, "Toxic Relationships."
32. Max Milner, "Peanut as Protein Resource in International Feeding Programs," 31 January 1962, Accession No. 060838, FAO Library.
33. C. Gopalan to C. G. King, 2 February 1960, in WHO Protein Advisory Group, NU 13/4, FAO Archives: 12-ESN-495; "Progress Report on Peanut Flour," PAG Meeting, June 1961, Accession No. 0608009, FAO Library.
34. Milner, "There's a Fungus among Us."
35. PAG. "Memorandum. Proposed Agenda for the next PAG Meeting," 15 September 1961, NU 13/4 –Meetings Vol. II, FAO Archives: 12-ESN-498.

36. Milner, "There's a Fungus among Us."
37. "Minutes of Meeting, Protein Advisory Group," 26–29 March 1962, NU 13/4—Meetings, Vol. III, FAO Archives: 12-ESN-498.
38. Minutes of Marketing Policy Committee, 7 December 1961, 2/6/13/2, GSK Heritage Archives, Brentford, Middlesex, UK.
39. Zbinden, *History of Nestlé's Infant and Dietetic Preparations*, n.d., 313, Nestlé Archives, Vevey, Switzerland.
40. Wintermeyer, *Die Wurzeln der Chromatographie*.
41. "Aflatoxin Recommendation," 3 August 1963, NU 13/4 "Meeting 1963," FAO Archives: 12-ESN-498.
42. Lancaster, Jenkins, and Philp, "Toxicity Associated with Certain Samples."
43. Le Breton, Frayssinet, and Boy, "Sur l'apparition d'hépatomes 'spontanés' chez le Rat Wistar"; Pirie, "Hepatic Carcinoma in Natives"; Mueller, "Cancer in the Tropics."
44. "PAG Minutes. (1963 Meeting). Progress Report," NU 13/4 "Meeting 1963," FAO Archive: 12-ESN-498.
45. Latham, "Hazards of Groundnuts."
46. L. J. Teply, "Memo for the Record," 26 August 1963, NU 13/4 "Meeting 1963," FAO Archive: 12- ESB-498.
47. "Minutes. Meeting—WHO/FAO/UNICEF Protein Advisory Group," 5–7 July 1965, FAO Archives: 12-ESN-498.
48. E. M. DeMaeyer to V. N. Patwardhan et al., 17 September 1963, FAO Archives: 12-ESN-498.
49. "Recommendation on Aflatoxin, Meeting of the WHO/FAO/UNICEF Protein Advisory Group, 17–19 August 1966," 30 September 1966, NU-13/4 PAG (Meeting 1966), FAO Archives: 12-ESN-498.
50. Ibid.
51. W. F. J. Cuthbertson, "Effect of Groundnut Meal Containing Aflatoxin on Cynomologus Monkeys," October 1967, Accession No. 105383, FAO Library.
52. "Recommendation on Aflatoxin, Meeting of the WHO/FAO/UNICEF Protein Advisory Group, 17–19 August 1966," 30 September 1966, NU-13/4 PAG (Meeting 1966), FAO Archives: 12-ESN-498.
53. "Report on the FAO/WHO/UNICEF PAG Meeting," 1967, Accession No. 105402, FAO Library.
54. Protein Advisory Group, "PAG Activities: Retrospect and Prospect," 15 May 1973, FAO Archives: 12-ESN-536.
55. For Kenya, see Mackenzie, *Land, Ecology, and Resistance in Kenya*; McCann, *Maize and Grace*; Moskowitz, *Seeing Like a Citizen*. For an example of cash crops in Africa, see Cooper, "'Ray of Sunshine on French Tables.'"
56. "PAG New Bulletin No. 3," February 1964, NU 13/4 "Meeting 1963," FAO Archives: 12- ESB-498.
57. Vogel, *Is It Safe?*
58. Daniel Banes, "Aflatoxin in Peanut Products," 29 January 1965, Folder 428 Peanut Aflatoxin, Box 3694, Records of the Food and Drug Administration, Record Group 88, General Service Files 1938—1974, National Archives at College Park, MD.
59. Schmidt, "Aflatoxin in Shelled Peanuts and Peanut Products Used as Human Foods," 42750.
60. Ibid.
61. Bureau of Foods, FDA, *Assessment of Estimated Risk*.
62. Mueller, "Cancer in the Tropics."
63. The US Supreme Court upheld the FDA's approach of using action levels instead of tolerance levels for aflatoxin regulation when challenged in 1986. See Hutt, Merrill, and Grossman, *Food*

and Drug Law, 507–21.

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65. "Council Directive of 17 December 1973 on the Fixing of Maximum Permitted Levels for Undesirable Substances and Products in Feedingstuff," *Official Journal of the European Communities*, no. L 38/31 (February 11, 1974).
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68. Food and Agriculture Organization of the United Nations, *Perspective on Mycotoxins*, 159.
69. Murphy, *The Emergence of the NIEO Ideology*; Prashad, *The Poorer Nations*.
70. Ogle, "State Rights against Private Capital"; Gilman, "The New International Economic Order"; Moyn, *Not Enough*.
71. UN General Assembly, Resolution 3202, Programme of Action on the Establishment of a New International Economic Order, A/RES/S-6/3202 (1 May 1974), http://www.un.org/en/ga/search/view_doc.asp?symbol=A/RES/3202(S-VI).
72. United Nations, *Report of the United Nations Conference on the Human Environment*.
73. Ibid.
74. Ibid., 21.
75. Winickoff and Bushey, "Science and Power in Global Food Regulation."
76. The link between the NIEO and global environmental governance has received little attention. See Conca, *An Unfinished Foundation*.
77. Food and Agriculture Organization of the United Nations, *Perspective on Mycotoxins*, 145.
78. Food and Agriculture Organization of the United Nations, *Report of the Joint FAO/WHO/UNEP Conference on Mycotoxins, Held in Nairobi, 19–27 September 1977*, 2.
79. Ibid., 8.
80. Food and Agriculture Organization of the United Nations, *Perspective on Mycotoxins*, 166.
81. Gilman, "The New International Economic Order."
82. McCann, *Maize and Grace*.
83. "Crop and Food Situation Report for the Month of March 1981," 22 April 1981, Kenya National Archives, Nairobi (KNA): KD 4/1.
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86. Ngindu et al., "Outbreak of Acute Hepatitis Caused by Aflatoxin Poisoning in Kenya."
87. Ibid.
88. Director to Regional Director, AFRO, "Food Safety Programme," 3 February 1977, KNA: BY/25/18, KNA.
89. "Food and Drugs Control," 1979—1985, KNA: BY/14/72.
90. W. Koinage to Provincial Medical Officers, 6 July 1981, KNA: BY 4/255.
91. "Ministry of Health, National Public Health Laboratory Services, Annual Report, 1986," Library of the National Public Health Laboratory Services, Nairobi, Kenya.
92. Tousignant, *Edges of Exposure*; Mutongi, *Matatu*.
93. Geissler, "Introduction."
94. Beck, *Risk Society*, 42.

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