



Environmental Toxicology Newsletter

"Published Occasionally at Irregular Intervals"

Arthur L. Craigmill
Extension Toxicologist

Vol. 23 No. 1 -- January 2003

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Study Affirms Water Effective for Reducing Trace Pesticide Residues on Produce, Finds Produce Washes Not Effective as They Claimed

A new study by Dr. Robert Krieger at the University of California, Riverside has found that produce washes are not as effective as they claim. The study looked to see if produce washes were more effective in removing pesticide residues than water. "Since we knew that water is effective in removing pesticide residues, it seemed very unlikely that produce washes would be as effective as they advertise," said Dr. Krieger. The study will be published in the February

2003 edition of *Bulletin Environ Contam Toxicol*.

In the first part of the study, crops that had been normally treated with captan were separated into three groups. The first group was rinsed with water, the second with water and produce wash, and the third was not rinsed. The unrinsed produce had a residue level of 6.7 parts per million. The group that was rinsed with water had a residue level of 4.1 parts per million, 39% less than the unrinsed group. The group that was rinsed with water and produce wash had a residue level of 3.7 parts per million, 45% less than the unrinsed group.

In the second part of the study, fruit that had been treated in the field with a tank mix of captan on methomyl were separated and treated as in the first part of the study. The fruit that was unrinsed had a residue level of 0.52 for captan and 0.87 for methomyl. The fruit that was rinsed with water had a residue level of 0.10 for captan and 0.71 for methomyl, 81% and 18% less than the unrinsed fruit respectively. The fruit that was rinsed with water and produce wash has a residue level of 0.053 for captan and 0.53 for methomyl, 90% and 39% less than the unrinsed fruit respectively.

The claims that produce washes are much more effective than water is not supported by this study. "Our concern about these claims is that they are misleading a public that is already concerned about pesticide residues," said Dr. Krieger. "Clearly, this study shows that there is no reason to spend extra money on these washes when water is just as effective."

Dr. Krieger also stressed that all of these residue levels are well below federal standards. "All levels of residues were well within the EPA levels and far below levels that could cause adverse health effects in rats or humans," said Dr. Krieger. "These studies simply reaffirm the effectiveness of water for trace pesticide residue reduction. In no case were residues of health significance."

REF: Alliance for Food & Farming, December 18, 2002.



Outbreaks of *Salmonella* Serotype Enteritidis Infection Associated with Eating Shell Eggs - United States, 1999-2001

A *Salmonella* serotype Enteritidis (SE) epidemic emerged in the 1980s, when increasing numbers of infections were detected in the Northeastern and Mid-Atlantic regions of the United States. In the early 1990s, while SE rates in the Northeast began to decline, the SE epidemic expanded to the Pacific region. Nationwide, the number of SE isolates reported to CDC peaked at 3.8 per 100,000 population in 1995. Although rates of culture-confirmed SE infection reported to CDC declined to 1.9 by 1999, rates did not decline further through 2001, and outbreaks continue to occur. Investigations of outbreaks and sporadic cases have indicated repeatedly that, when a food vehicle is identified, the most common sources of SE infection are undercooked and raw shell eggs. This report describes two SE outbreaks associated with eating shell eggs and underscores the need to strengthen SE-control measures.

South Carolina, 2001

During February-March 2001, outbreaks of gastroenteritis occurred among inmates in four prison facilities of the South Carolina Department of Corrections (SCDC). The first outbreak occurred in a men's facility (M1) on February 6.

The three other outbreaks, all occurring on March 2, affected a second men's facility (M2) and two women's facilities (F1 and F2). Among 2,317 inmates in the four prisons, 688 reported to prison infirmaries with gastrointestinal symptoms (e.g., abdominal cramps, diarrhea, and nausea). Stool specimens from ill inmates yielded SE phage types 2, 13a, and 23. No illness was reported among SCDC staff members.

A tuna salad served for lunch on March 2 was eaten by 88% of the male case-patients and by 89% of the female case-patients. The tuna salad was prepared with eggs that were reportedly hard-boiled by kitchen staff, who also were inmates. At the time of the outbreaks, all eggs used by the four involved SCDC facilities were supplied from a single vendor and eggs submitted from the farm tested positive for SE phage types 2, 13a, 22, 23, and 28. Phage type 2 was the predominant SE strain isolated from both ill patients and eggs from the farm. To protect the inmates, SCDC switched to pasteurized egg products in April 2001.

North Carolina, 2001

In June 2001, the Statistical Outbreak Detection Algorithm at CDC signaled an increase in SE cases reported from North Carolina. The Division of Public Health in North Carolina was alerted and began to review SE cases throughout the state. The North Carolina State Laboratory of Public Health reported 51 cases in July and 31 in August, compared with 11 cases in each of those months during 2000. Cases occurred throughout the state. Analysis of 53 patients and 78 controls indicated that illness was associated with eating eggs. A traceback of implicated eggs purchased from retail outlets in North Carolina was inconclusive for implicating a farm.

Editorial Note: During 1990-2001, state and territorial health departments reported 677 SE outbreaks, which accounted for 23,366 illnesses, 1,988 hospitalizations, and 33 deaths. Among the 309 outbreaks reported with a confirmed vehicle of transmission, 241 (78.0%) were associated with shell eggs, accounting for 14,319 illnesses. Of these, 10,406 illnesses occurred during 1990-1995, and 3,913 occurred during 1996-2001. The overall decrease in SE incidence and the decrease in the number of illnesses related to egg-associated SE outbreaks during the last decade might be attributed in part to the implementation of prevention measures, including on-farm control programs, egg refrigeration, and consumer and food worker education. However, reported cases did not decline during 1999-2001, and outbreaks associated with shell eggs continue to occur.

Eggs that reportedly were hard-boiled and used in a tuna salad were the implicated vehicle in the South Carolina outbreak. **A recent study demonstrated that unless SE-containing eggs are exposed to boiling water until the yolk is completely solidified, SE can survive the cooking process.** Cross contamination of the tuna salad by inmate food handlers also was possible.

The outbreak in South Carolina prisons was the largest SE outbreak in 2001. Because persons residing in institutions depend entirely on their institutions for meals, the supply of contaminated foods to these settings can place large populations at risk for developing foodborne diseases. **Persons residing in institutions, especially elderly persons in nursing homes or assisted living facilities, are at higher risk for dying from outbreak-associated SE infections.** During 1990-2001, a total of 83 SE outbreaks occurred in institutional settings, representing 12% of reported SE outbreaks. Of the 33 outbreak-associated deaths, 22 (67%) occurred in institutional facilities, underscoring the importance of using pasteurized egg products or in-shell pasteurized eggs for all recipes requiring pooled, raw, or undercooked shell eggs for institutionalized persons.

Additional information about preventing SE infections associated with eating raw or undercooked shell eggs is available at: http://www.cdc.gov/ncidod/dbmd/diseaseinfo/salment_g.htm

<http://www.cfsan.fda.gov/~dms/fs-eggs.html>

<http://www.cfsan.fda.gov/~dms/fs-eggs2.html>

<http://www.cfsan.fda.gov/~dms/fs-eggs4.html>

Information for retail and food service establishments and institutional facilities about handling and cooking shell eggs

is available in the Food Code at <http://www.cfsan.fda.gov/~dms/foodcode.html>

To read this entire article link to: <http://www.cdc.gov/mmwr>

REF: *Morbidity and Mortality Weekly Report*, 51(51);1149-1152, January 3, 2003



Outbreak of Botulism Type E Associated with Eating a Beached Whale - Western Alaska, July 2002

Botulism is a neuroparalytic illness caused by toxins produced by the bacterium *Clostridium botulinum*, an obligate anaerobe found commonly in the environment. Intoxication with toxin type E is associated exclusively with eating animal foods of marine (salt or fresh water) origin. Persons who eat raw or fermented marine fish and mammals are at high risk for botulism from type E toxin. On July 17, 2002, the Alaska Division of Public Health investigated a cluster of suspected botulism cases among residents of a fishing village in Alaska. This report summarizes the findings of the outbreak investigation, which linked disease to eating raw muktuk (skin and a pink blubber layer) from a beached whale. To avoid delays in treatment, health-care providers evaluating patients suspected of having botulism should base treatment decisions on clinical findings. Public health authorities should be notified immediately about any suspected botulism case.

During July 13-15, residents of a western Alaska village on the Bering Sea shore shared a meal consisting of muktuk harvested from a beached adult beluga whale found near their village. The villagers estimated that the whale had been dead for at least several weeks. They cut the whale fluke (tail) into pieces and stored them in zipper-sealed plastic bags in a refrigerator until they were eaten 1 or 2 days later. On July 17, after a physician from western Alaska reported three suspected cases of botulism among patients who had eaten the muktuk, the Alaska Section of Epidemiology began an investigation.

A case of foodborne botulism was defined as illness in a person who had eaten the muktuk and subsequently had symmetric descending flaccid paralysis of motor and autonomic nerves. Persons who ate muktuk were interviewed and examined, and their hospital records were reviewed. Serum, stool, and gastric contents from patients and leftover blubber were tested for botulinum toxin.

Of 14 persons identified who ate the muktuk, eight (57%) had an illness that met the case definition. Five of the eight patients were female; the median age was 73 years (range: 13-83 years). Symptom onset after ingestion of muktuk occurred within 36 hours in all patients. Five patients were hospitalized, four received antitoxin, and two required mechanical ventilation. Three stool, three gastric fluid, and seven serum samples from the eight patients and seven samples of muktuk were tested for botulinum toxin at CDC's National Botulism Surveillance and Reference Laboratory. The diagnostic laboratory received all laboratory specimens on July 26, and results were reported on August 1. Type E toxin was detected in stool from one patient. All seven samples of muktuk were positive for type *E botulinum* toxin.

Editorial Note: During 1973-1998, a total of 814 cases and an annual median of 24 cases (range: 14-94 cases) of foodborne botulism were reported to CDC; 236 (29%) of these cases occurred in Alaska. Although botulism is a rare disease, its presentation is distinctive (Because of the epidemic potential of foodborne botulism, every case should be reported and investigated immediately.)

The probable mode of contamination of the whale in this outbreak was either growth and toxin secretion by *C. botulinum* present in the intestinal tract of the whale or traumatic introduction of *C. botulinum* spores into the beached whale tissue from contact with sand, rocks, and driftwood, and subsequent germination and toxin production. *C. botulinum* type E has been found in Alaska coastline soil, and outbreaks of botulism associated with eating beached marine mammals are documented. A previous report on the accumulation of *C. botulinum* toxins in the North Sea coastal food chain associated with beached whales suggested the disposal of the carcasses as a preventive measure. However, because of the impracticality of frequent scanning of the vast Alaska shoreline and high costs associated with disposal, the U.S. Fish and Wildlife Service does not remove beached mammal carcasses regularly.

Persons should avoid eating beached marine mammal carcasses and boil raw or fermented Alaska Native dishes >10 minutes before eating to inactivate botulinum toxin. Additional information on botulism prevention is available at <http://www.phppo.cdc.gov/phtn/botulism/alaska/alaska.asp> and http://www.epi.hss.state.ak.us/pubs/botulism/bot_01.htm.

For the entire report link to: <http://www.cdc.gov/mmwr/>

REF: *Morbidity and Mortality Weekly Report*, January 17, 2003, 52(02);24-26.



Infant Botulism - New York City, 2001--2002

Infant botulism results from germination of swallowed spores of botulinum toxin-producing clostridia that colonize the large intestine temporarily. The annual incidence of infant botulism in the United States is two cases per 100,000 live births. Staten Island recorded 5,899 live births in 2000; incidence of infant botulism during this 12-month period was 68 cases per 100,000 live births. This report summarizes the investigation of these four cases; as expected with infant botulism, a common source of exposure was not identified. All four patients recovered after treatment and were discharged from local hospitals. State and local health departments should be notified promptly when infant botulism is suspected to arrange diagnostic testing.

Infant botulism is a reportable disease in NYC, and the NYC Department of Health and Mental Hygiene (DOHMH) investigates all suspected cases. Botulism should be suspected in an infant aged <12 months with symptoms including constipation, lethargy, poor feeding, weak cry, bulbar palsies, and failure to thrive. These symptoms might be followed by progressive weakness, impaired respiration, and sometimes death.

Summary of Cases: All four patients received antibiotics during hospitalization. None had ingested honey or had parents employed in occupations that might increase exposure to *C. botulinum* spores in soil and dust (e.g., construction, plumbing, and farming). All patients had uncomplicated gestational histories and vaginal deliveries. All resided within a 6-mile radius of each other. All parents reported recent construction in their neighborhoods during the period (range: 1-31 days) before illness onset. In the fourth case, the infant's home had been remodeled since the infant was born.

Editorial Note: Intestinal botulism is the most common form of human botulism in the United States, and approximately 100 cases are reported among infants in the United States annually (Intestinal botulism occurs rarely in

older children and adults). Intestinal botulism results from colonization and bacterial production of botulinum toxin in the colon. Swallowing ambient *C. botulinum* spores, which exist worldwide in soil and dust, has been proposed as the principal route of exposure; honey is an avoidable source of some causative spores. A common source of exposure generally is not identified; apparent clusters such as the four Staten Island cases are rare and often remain unexplained after investigations are complete. In a cluster of infant botulism cases identified previously in the mid-Atlantic region of the United States, no common source of exposure was identified.

Botulism should be suspected in previously healthy infants aged <12 months who are constipated and who exhibit weakness in sucking, swallowing, or crying; hypotonia; and progressive bulbar and extremity muscle weakness. Approximately half of patients require mechanical ventilation during hospitalization.

For the entire report link to: <http://www.cdc.gov/mmwr/>

REF: *Morbidity and Mortality Weekly Report* January 17, 2003 / 52(02);21-24



Turning Up the Heat on Acrylamide

A potentially cancer-causing agent used to manufacture certain chemicals, plastics, and dyes has recently been found to be a natural by-product of cooking certain foods. The [Food and Drug Administration](#) is taking a closer look at this white, odorless chemical, [acrylamide](#), to determine how much of it occurs in foods and whether it could pose a health risk. In April 2002, researchers in Sweden discovered that cooking at high temperatures could create acrylamide in many types of foods, particularly starchy foods such as french fries, potato chips, bread, rice, and processed cereals.

Scientists know that acrylamide causes cancer in laboratory rats. They also know that contact with large quantities of acrylamide can cause nerve damage in humans. But no one knows whether the tiny amounts of acrylamide in cooked foods can cause cancer or have any other harmful effects when ingested by people. "As soon as we heard about this problem, we took action and laid out a solid plan to learn more about acrylamide and to reduce exposure to it," says Terry Troxell, Ph.D., director of the FDA's Office of Plant and Dairy Foods and Beverages.

The FDA's draft action plan for acrylamide in food was presented in September 2002 at the first of a series of public meetings held to get feedback and to provide updates on FDA activities related to acrylamide. With the goal to prevent or reduce the potential risk of acrylamide in foods to the greatest extent feasible, the FDA's plan calls for developing laboratory methods to measure acrylamide and surveying the levels of acrylamide in foods. In addition, FDA scientists will study how acrylamide is formed so that the agency can identify ways to reduce it. "We really want to help industry understand what they might be able to do to reduce the formation of acrylamide," says Richard Canady, Ph.D., a toxicologist in the FDA's Center for Food Safety and Applied Nutrition.

What We Know So Far

Following the Swedish researchers' identification of acrylamide in foods, researchers in other countries, including Norway, the United Kingdom, Switzerland, Canada, and the United States, analyzed samples of foods and came up with similar findings. The FDA developed its own method to measure levels of acrylamide in foods and has tested more than 300 food items. Studies by the FDA and others found a wide variation in the levels of acrylamide among different types

of foods and even different brands. "Much more testing is needed to understand the scope of occurrence of acrylamide in food," says Troxell. The FDA's plan calls for testing about 1,500 more samples over the next year, and more testing may be added based on the findings.

Acrylamide was not found in uncooked or boiled food -- studies indicate that it appears to form during certain high-temperature (greater than 250 F) cooking processes, such as frying and baking, and that levels of acrylamide increase with heating time. Home-cooked foods, as well as pre-cooked, packaged and processed foods, have been found to contain acrylamide.

Acrylamide levels in 39 samples of potato chips ranged from less than 1.4 micrograms to 100 micrograms per ounce, according to a group of international food safety experts who met in June 2002 in Geneva to discuss the public health impact of acrylamide in foods.

This meeting of experts, including FDA scientists, was hosted by the United Nations Food and Agriculture Organization (FAO) and the World Health Organization (WHO). The FAO and WHO reported that the short-term dietary intake of acrylamide was found to be about 50 micrograms per day for the average adult -- an amount significantly below that known to cause nerve damage in laboratory animals.

The FAO and WHO experts concluded that more information was needed on acrylamide in food, but added that the substance was a "major concern." Based on high-dose experiments in animals, acrylamide is classified as a potential human carcinogen, as well as a genotoxicant, a substance that can mutate and damage genetic material.

Advice for Consumers

Based on the current knowledge about acrylamide, the FDA has re-emphasized its traditional advice to eat a balanced diet, choosing a variety of foods that are low in fat and rich in high-fiber grains, fruits, and vegetables. "As more information becomes available, we will consider additional messages, for example, recommendations related to cooking," says Troxell.

The FAO and WHO advise consumers that food should not be cooked excessively -- for too long or at too high a temperature. They also recommend cooking all food thoroughly, particularly meat and meat products, to destroy foodborne pathogens, such as bacteria and viruses.

The FDA reinforces that consumers should not overreact. "It's a bigger risk if you don't cook your food thoroughly and consume pathogens," says Troxell. It's also a nutritional risk to avoid foods rich in fiber such as cereals and whole-grain products.

Educating consumers will be an important part of the FDA's acrylamide action plan. "Once we have enough information, we want to help consumers understand the potential risks for acrylamide, how it gets into food, and what they can do to avoid it," says Canady.

Cooperative Research

There is a high level of cooperation and information-sharing among the FDA, other U.S. and international government agencies, research institutions, academia, and industry, says Canady. And it's starting to pay off. Five different labs throughout the world have announced that they discovered what may be a primary mechanism of how acrylamide can be formed in food. They identified a high-temperature reaction of two compounds found in potatoes and other carbohydrates: glucose (a sugar) and asparagine (an amino acid).

In October 2002, the Joint Institute for Food Safety and Applied Nutrition and the National Center for Food Safety and Technology held a workshop titled "Acrylamide in Food: Scientific Issues, Uncertainties, and Research Strategies." Intended to determine acrylamide research needs and facilitate coordination and collaboration among scientists worldwide, the workshop looked at all the components of acrylamide and the current research being done.

"People are working very hard in the agency and around the world to understand acrylamide levels and see why it's formed," adds Troxell. "Once we understand what causes it, we can better address how to reduce it."

REF: U.S. Food and Drug Administration, *FDA Consumer magazine*, January-February 2003.



MSG: A Common Flavor Enhancer

Although it has no distinct taste itself, monosodium glutamate (MSG) stimulates our taste buds and makes a variety of foods taste better. The flavor enhancer is commonly added to Asian cuisine, canned vegetables, soups, and processed meats. Made by a fermentation process using starch, beet sugar, cane sugar, or molasses, MSG is sold as a white crystal substance that resembles salt and sugar.

Many consumers often equate all "free glutamate" products with MSG, but it is only one of several forms of glutamate -- a major building block of proteins. Free glutamate, which results when glutamate is released during the breakdown of a protein molecule, occurs naturally in many foods, such as meat, milk, mushrooms, Parmesan cheese, and tomatoes.

In 1959, the Food and Drug Administration classified MSG as a "generally recognized as safe" food ingredient under the Federal Food, Drug, and Cosmetic Act. But the use of MSG in food has remained controversial. In the 1980s, research showed that glutamate plays an important role in the normal functioning of the nervous system, raising questions about whether glutamate in food could affect the nervous system.

The FDA also received numerous reports of MSG-related adverse events, including headaches, palpitations, vomiting, and nausea. While these voluntary reports were useful for drawing attention to potential problems, they were unconfirmed by controlled testing.

Because of concerns about the adverse event reports, the FDA sponsored several safety assessments which all concluded that MSG is safe when consumed at levels typically used in cooking and food manufacturing. In 1986, FDA's Advisory Committee on Hypersensitivity to Food Constituents found that MSG was generally safe, but that short-term reactions may occur in some people. Other reports from the American Medical Association's Council on Scientific Affairs and the European Community's Scientific Committee for Foods reported similar findings.

Then in 1992, the FDA contracted with the Federation of American Societies for Experimental Biology (FASEB), an independent group of scientists, to complete the most comprehensive review of available scientific data on glutamate safety to date. The 1995 FASEB report reaffirmed the safety of MSG when it is consumed at usual levels by the general population, and found no evidence of any connection between MSG and any serious long-term reactions. The report indicated that no evidence exists to suggest that dietary MSG or glutamate contributes to Alzheimer's disease, Huntington's disease, or any other long-term or chronic diseases. There was also no evidence suggesting that dietary MSG or glutamate causes brain lesions or damage to nerve cells in humans.

But the report did identify short-term reactions known as MSG Symptom Complex in two groups of people. The first group includes people who may have a reaction after eating large doses of MSG, particularly on an empty stomach. A large dose would be three grams or more per meal. A typical serving of glutamate-treated food contains less than 0.5

grams of MSG. The second group includes people with severe and poorly controlled asthma. MSG Symptom Complex can involve symptoms such as numbness, burning sensation, tingling, facial pressure or tightness, chest pain, headache, nausea, rapid heartbeat, drowsiness, and weakness. Asthmatics may experience these symptoms as well as difficulty in breathing. Additional studies in asthmatics under controlled conditions have not produced consistent results.

Glutamate is commonly found in food, primarily from protein sources. Foods and ingredients that contain glutamate as an inherent component are not required to list glutamate on the label. Examples include tomatoes, cheeses, meats, hydrolyzed protein products such as soy sauce, and autolyzed yeast extracts. These ingredients are declared on the label by their common or usual names.

It's when MSG is added to food that the FDA requires "monosodium glutamate" to be listed on the label. Other salts of glutamic acid -- such as monopotassium glutamate and monoammonium glutamate -- also have to be declared on labels and can't be lumped together under "spices," "natural flavoring" or other general terms.

For the entire article link to: <http://www.fda.gov/fdac/>

REF: U.S. Food and Drug Administration, *FDA Consumer magazine*, January-February 2003.



◆ Toxicology Tidbits ◆

~~ Household Contamination with *Salmonella enterica*

Household contamination with *Salmonella enterica* increases when occupational exposure exists (cattle farms with known salmonellosis in cattle, a salmonella research laboratory, or a veterinary clinic experiencing an outbreak of salmonellosis). Fifteen of 55 (27.2%) vacuum cleaner bags from households with occupational exposure to *S. enterica* were positive versus 1 of 24 (4.2%) without known exposure. Use of a carpet cleaner and several cleaners/disinfectants reduced, but failed to eliminate, *S. enterica* from artificially contaminated carpet.

Read the entire article at: www.cdc.gov

REF: *Emerging Infectious Diseases*, Vol. 9, No. 1, January 2003.



~~ New Website Compiles FDA's Spanish Publications

Dozens of [FDA's Spanish-language publications](#) are within easy reach on a new website that includes materials related to all products the agency regulates. Many of the publications are written for consumers; others provide guidance for FDA-regulated companies. Subjects found on the site include rare diseases, using medicine wisely, eating for a healthy heart, mammograms, and foodborne illness.

The site also links to general information about health conditions such as diabetes, the flu, and hearing loss.

REF: FSnet Dec. 20/02



~~ More on "Toxic Mold"

Neuropsychologist debunks "toxic mold" claims. Paul R. Lees-Haley, Ph.D., has written a lengthy article criticizing the diagnosis of ["mold neurotoxicity"](#) and the many lawsuits filed by alleged victims. He states that (a) there is no consistent pattern of symptoms or test results through which that diagnosis of "mold neurotoxicity" can be defined, and (b) there is no scientific evidence that breathing mold spores in household and commercial office settings causes neuropsychological impairment. The article concludes:

"Toxic tort attorneys and a handful of experts they favor would like you to believe that "toxic mold" is disabling people in epidemic proportions by damaging their brains. In order for this to be correct, the overwhelming majority of physicians, toxicologists, and mental health professionals who have studied this issue would have to be completely wrong, and doctors in day-to-day practice would have to be overlooking the diagnosis. . . .

"The mold neurotoxicity debate is not simply about health care and science -- a focus on money and litigation is pervasive in the communications of the toxic mold promoters. . . . The campaign being waged to convince people of the dangers of "toxic mold" is not merely an amusing example of folly in modern society. The people who are bypassing scientific evidence and engaging in wholesale dissemination of "toxic mold" rhetoric are not neutral forces. If it turns out that these exposures are neuropsychologically harmless, the hysterical claims and unfounded alarms sounded by lawyers, doctors, and others will nonetheless have harmed many victims. . . . Further exploration of the effects of inhaling mycotoxins and mold spores should be through high-quality, well controlled, scientific studies, not speculation in adversarial settings."



~~ World Health Organization's Website

The World Health Organization's Department of Food Safety website has a couple of topics that may be of interest to our readers. <http://www.who.int/fsf/>

Answers to the 20 Most Common Questions on [Genetically Modified \(GM\) Foods](#) (available in Arabic, Russian, Chinese in addition to English, French and Spanish) and [Acrylamide in Food](#) -- Frequently Asked Questions.



VETERINARY NOTES.....



Safety Guidance Issued for Using Raw Meat in Diets of Pets and Zoo Animals

Prompted by an increased use of raw meat for pets and zoo animals, FDA has issued a draft guidance for industry on the manufacture and labeling of [diets containing raw meat](#), or other raw animal tissue, for consumption by these animals. The guidance includes warnings about bacterial contamination and dental or gastrointestinal trauma, and it recommends measures to minimize contamination and disease transmission.

REF: *FDA News Digest*, December 23, 2002.

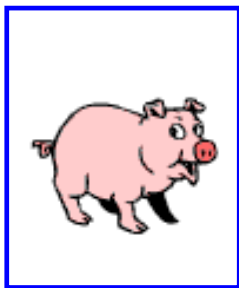


Revision of the Definition of the Term "No Residue" in the New Animal Drug Regulations

SUMMARY: The Food and Drug Administration (FDA) is amending its regulations regarding carcinogenic compounds used in food-producing animals. Specifically, FDA is deleting the operational definition of the term "no residue" and is making conforming amendments to other parts of these regulations. FDA is making these amendments in response to a legal opinion issued by the Department of Justice (DOJ), Office of Legal Counsel, which concluded that the operational definition of "no residue" is not legally supportable. This rule is effective January 22, 2003.

REF: *Federal Register*, Volume 67, Number 246, December 23, 2002

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