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Arthur L. Craigmill
Extension Toxicologist

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Introduction

I recently attended a conference held at Michigan State University and hosted by their National Food Safety Center. The primary focus of the conference was microbial pathogens and foodborne disease, but they also had several presentations which focused on chemical residues in both plants and animal-derived foods. Throughout the conference there were three words used many times, by almost every speaker. These words were HAZARD, RISK and SAFETY. It became apparent to me during the conference that people were using these words as if we all had similar definitions for them, however it was clear that we did not. During a discussion session, I asked three of the speakers (a physician from the Centers for Disease Control and Prevention, a toxicologist, and a sociologist [both from Michigan State University]), to please define these words. The toxicologist went first, and gave what I thought was the best rendition, or should I say, that which is closest to the operational definition I use. He used the National Academy of Sciences (NAS) paradigm for risk assessment, which begins with hazard determination (which is determining if a chemical (or pathogen) can cause a harmful effect), followed by dose-response assessment, and then exposure assessment. When this is all determined, these are put together to calculate a risk probability. The final step in the process is deciding if that is acceptable.

The physician said that routinely he used the words interchangeably, and considered them to be the same. The sociologist had a similar understanding to that of the toxicologist and felt that the public considered safety to be the absence of risk.

It seems to me that people working in the realm of risk assessment and food safety need to have similar understandings of these terms and processes if we are going to communicate effectively with one another, and even more so if we are going to communicate with non-scientists.

Years ago when I team taught a University Extension course with Dr. Lee Shull, Lee taught the risk assessment portion of the course. He had very good definitions for HAZARD and RISK. HAZARD is the POSSIBILITY that a chemical can cause harm, and that is related to its toxicity and physical/chemical properties. RISK is the PROBABILITY of an adverse effect, and for chemicals it is determined by EXPOSURE and toxicity dose-response. SAFETY can be defined as a probability to, that is; 1-RISK. Thus if the risk of an adverse outcome is 1 in 100, 0.01, then the SAFETY is 99 out of 100, or 0.99. While this is a useful mathematical definition, another definition is probably more useful, and that is that SAFETY is ACCEPTABLE RISK. Using this definition, SAFETY then becomes a non-scientific determination since the acceptability of risk will be a decision made at the personal and societal levels. It will relate more to public policy than to science, however, risk assessment can and must be based on good data and good science.

An element which is often not fully appreciated when we talk about risk assessment, and particularly about chemical risk assessment is that predictive risk (extrapolation from animal data to humans, for example) always carries with it uncertainty, and that this uncertainty can be quantified. For example, when performing risk assessment for carcinogens, dose-response data and exposure data are combined to calculate a probability of cancer due to exposure to the particular chemical in question. Also calculated are confidence intervals (CI) or limits on these data, which are really measures of uncertainty. We usually use the 99% CI which essentially says that 99 times out of 100 the true value for the prediction will lie within these limits. Often for cancer risk assessment the CI may be 3-5 orders of magnitude, which means the lower value will differ from the upper value by a factor of 100 to 10,000 or more.

It is also necessary to appreciate that the risks predicted are POPULATION risks, not individual risks. To illustrate this, there is a cartoon which appeared in the NAS publication on Risk Communication. It shows a winding road going into a series of mountains, with a sign at the entrance which says: "Fatality Risk Next 10 Miles, 5×10^{-6} (5 in a million)". This wonderful cartoon illustrates several principles of risk. The first is that the risk of driving that road can actually be measured by counting the people who go in one end alive, and the number of people which come out alive. This is an ACTUARIAL RISK. It is the type of risk measurement which is used for establishing insurance rates.

Another concept that it illustrates exceptionally well is that the risk figure is a population risk, and that the risk to any individual driving that road is NOT five in a million. An individual's risk will be more or less based on the person's car, mental condition, and behavior behind the wheel. It also illustrates that risk is the probability of an adverse effect under very specific conditions (driving the road, chemical exposure, eating pathogen contaminated food, etc.). Right now it is impossible to predict INDIVIDUAL RISK, but it is possible to minimize factors which contribute to risk (risk factors).

Because predictive risk assessment has uncertainty, I think it is imperative that this uncertainty be communicated to non-risk assessors and non-scientists so that they can better understand the process. Too often, regulatory agencies and special interest groups will publish risk estimates and only mention the upper-bound estimate. Indeed, exposure to chemical toxicants IS usually regulated using this upper-bound 99 CI, but the choice of this value is a RISK MANAGEMENT decision, NOT a risk assessment decision.

Another problem which is often encountered with predictive risk assessment is the tendency of agencies (governmental and special interest) to take the risk estimates, multiply them by the potentially exposed population, and come up with "body counts." This is a form of "math abuse."

Lastly, when communicating with non-scientists, we must be certain that we use well-defined terms, or at least understand what definitions others use for similar words. This will make effective communication possible. Good communication begins at home!

~~~ **Art Craigmill**

Peanut Allergy and the Detection of Peanut Allergens in Human Breast Milk

In a recent article in the *Journal of the American Medical Association*, researchers in Toronto have shown that peanut protein allergens can be excreted in human breast milk. Peanut allergy is estimated to affect about 1% of US school children, and is a serious problem for the allergic child since it begins early, and lasts throughout life (you usually don't grow out of it). Allergic reactions are often severe and life threatening, and it is widely accepted that peanuts account for the majority of allergic reaction deaths.

An unusual aspect of peanut allergy is that in about 10-80% of the children affected, the first known exposure to the food produces an allergic reaction, indicating that an unknown (occult) exposure must have taken place to provoke the sensitivity. Breast milk has been suspected to be such a route for peanuts, and has been previously shown to be a route of exposure to egg, milk and wheat allergens.

In the recent study only 48% of the women showed excretion of the peanut allergen in milk after eating about 2 ounces of dry roasted peanuts. This is similar to the percentage of women who excrete egg (59-74%) and milk (53-63%) allergens in breast milk. The authors suggest that transfer of peanut allergens in breast milk may predispose children who are at-risk of sensitization, to acquire peanut protein allergy.
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REF: *JAMA*, 285(13), April 4, 2001.

Fatal Pediatric Lead Poisoning --- New Hampshire, 2000

Fatal pediatric lead poisoning is rare in the United States because of multiple public health measures that have reduced blood lead levels (BLLs) in the population. However, the risk for elevated BLLs among children remains high in some neighborhoods and populations, including children living in older housing with deteriorated leaded paint. This report describes the investigation of the first reported death of a child from lead poisoning since 1990. The investigation implicated leaded paint and dust in a home environment as the most likely source of the poisoning. Lead poisoning can be prevented by correcting lead hazards, especially in older housing, and by screening children at risk according to established guidelines.

On March 29, 2000, a 2-year-old girl was seen at a community hospital emergency department with a low-grade fever and vomiting of approximately 1 day's duration. The child had been well since arriving in New Hampshire from Egypt with her Sudanese refugee family 3 weeks earlier. Laboratory findings included a microcytic anemia (hemoglobin: 7.6 g/dL; lower limit of normal: 11.5 g/dL) with occasional basophilic stippling of red blood cells. A throat swab streptococcal antigen screening test was positive. She was discharged from the emergency department with prescriptions for an antibiotic and antiemetic to treat presumed strep throat. However, her vomiting worsened, and she was admitted to the same hospital on April 17, and then transferred to a tertiary-care hospital the next day. On April 19, approximately 5 hours after the transfer, she became unresponsive, apneic, and hypotensive. She was intubated and placed on a ventilator. Computerized tomography of the head showed diffuse cerebral edema and dilated ventricles. Later that day, the results of a blood test drawn on April 18 showed a BLL of 391 µg/dL and an erythrocyte protoporphyrin level of 541 µg/dL. Chelation therapy was initiated. Despite a decrease in her BLL to 72 µg/dL, she remained comatose. She was pronounced brain dead on April 21. An autopsy found diffuse cerebral edema. A hair sample lead concentration was 31 µg/g in the distal centimeter and 67 µg/g in the proximal centimeter, indicating a large increase in lead exposure during the preceding month. Radiographs of the left knee were equivocal for growth arrest lines that can occur in chronic lead poisoning.

After living in Egypt for approximately 18 months, on March 9, 2000, the family had moved to Manchester into an apartment constructed before 1920. A wall in a sibling's bedroom had multiple holes from which the patient had been seen removing and ingesting plaster. Two of seven samples of plaster with the adhering surface paint contained lead at levels of 5% and 12%. Peeling paint (35% lead) was present on the balusters and floor (3% lead) of a porch outside the apartment entrance where the patient sometimes had played. She also had played near and looked out of a living room window that occasionally was opened during meal preparation. A wipe sample of dust from the window well showed 6732 µg lead/ft2, well above the hazardous level of 800 µg/ft2.

BLLs in the mother and three siblings (ages 5, 11, and 15 years) ranged from 4-12 µg/dL. The family did not use or possess nontraditional remedies, food supplements, cosmetics, or ceramic eating or drinking containers acquired abroad. No one in the household was employed or had lead-related hobbies. Measurements of stable lead isotopes in selected environmental samples and the patient's blood showed that the isotopic lead composition of the porch paint and window

well dust in the her Manchester apartment matched the composition of lead in her blood more closely than did the isotopic composition of other samples, including those from her previous residence in Egypt.

Editorial Note: Lead encephalopathy is a life-threatening complication of lead poisoning that can occur in young children who have very high BLLs (>70-100 µg/dL). Nonspecific symptoms (e.g., lethargy, sporadic vomiting, and constipation) can occur at BLLs >50-70 µg/dL and may precede the abrupt onset of frank encephalopathy characterized by persistent vomiting, ataxia, altered consciousness, coma, and seizures. In this report, the child's anemia with basophilic stippling also suggested lead poisoning. However, symptoms or signs cannot be used to reliably diagnose or exclude lead poisoning; a BLL must be measured whenever lead poisoning is suspected. In young children, BLLs >70 µg/dL or elevated BLLs with symptoms suggesting encephalopathy require prompt inpatient treatment with chelating agents to rapidly reduce BLLs. Providing appropriate intensive care for children with encephalopathy can prevent death, although severe permanent brain damage can occur despite treatment.

During the 1950s and 1960s, acute, often fatal, lead encephalopathy was a common cause of pediatric admissions to urban hospitals. The subsequent decline in fatal lead poisoning cases is attributable to reduced lead exposure from multiple sources, institution of lead screening programs, and improved treatment of lead poisoning. Despite the reduction in severe lead poisoning, in some U.S. counties, >20% of young children tested have BLLs >10 µg/dL, high enough to adversely affect learning and development.

The likely sources of lead poisoning for the child in this report (deteriorated leaded paint and elevated levels of lead-contaminated house dust) are found in an estimated 24 million U.S. dwellings, 4.4 million of which are home to one or more children aged <6 years. Lead hazards are especially common in homes built before 1960 (58%). Although the patient's pica and iron deficiency probably contributed to the severity of her lead poisoning, by increasing ingestion and absorption of lead, all children living in homes with lead hazards are at increased risk for developing elevated BLLs.

REF: Morbidity and Mortality Weekly Report, 50(22), June 08, 2001.

Trends in Blood Lead Levels Among Children --- Boston, Massachusetts, 1994--1999

Data from the National Health and Nutrition Examination Survey and national childhood blood lead surveillance data from 19 states indicated that average blood lead levels (BLLs) in young children decreased during the late 1990s. The proportion of children tested who had BLLs >10 g/dL declined from 10.5% in 1996 to 7.6% in 1998, although the proportion was higher in certain counties. To determine whether a similar decline had occurred in Boston, Massachusetts, where a high proportion of children are tested each year, and whether any changes were similar in high- and low-risk neighborhoods, CDC, in collaboration with the Boston Childhood Lead Poisoning Prevention Program (BCLPPP) performed an analysis of BLLs among children aged 6-72 months in Boston during 1994-1999. The results indicate that BLLs in Boston declined during this period, but because of the geographic variation in lead exposure, continued surveillance will be necessary to eliminate childhood lead poisoning.

During 1994-1999, the overall prevalence of children with BLLs >10 g/dL declined 45%, from 9.3% to 5.1%. The prevalence of children with BLLs >20 g/dL declined 66%, from 1.5 % (545) to 0.5% (140). Neighborhoods with prevalence rates in the upper tertile in 1994 had a higher percentage of children living in poverty, Spanish-speaking households, and vacant parcels than neighborhoods with lower prevalence rates. Overall, about two thirds of houses were built before 1950, and no substantial differences were found in the proportion of houses built before 1950 between the highest and lowest risk neighborhoods. The prevalence of elevated BLLs declined from 1994 to 1999 in all 16 neighborhoods, with the highest absolute average decline (4.8%) in neighborhoods with the highest prevalence in 1994. In 1999, six high-risk neighborhoods accounted for 80% of children with elevated BLLs, approximately the same as in 1994 (83%).

The decline in prevalence of elevated BLLs during 1994-1999 was similar across age groups: 51% among children aged <12 months, 42% among children aged 12-36 months, and 46% among children aged 36-72 months.

Editorial Note: The findings in this report indicate that the prevalence of elevated BLLs in Boston declined consistently during 1994-1999, similar to declines reported from 19 states. Building of new houses and remodeling of older houses that removed lead painted building components, such as windows, may have contributed to this decrease.

Although BLLs have declined in all Boston neighborhoods, levels remained higher in 1999 in the areas with the highest levels in 1994. These high-risk neighborhoods are characterized by higher proportions of minority children, children living in poverty, and vacant properties; a high proportion of old housing, likely to have leaded paint, is found in all neighborhoods. Low socioeconomic status and associated deterioration of older housing are major contributors to lead exposure in Boston .

REF: *Morbidity and Mortality Weekly Report*, 50(17), May 04, 2001.

Antibiotic-Resistant Genes Traced From Farms to Groundwater

Champaign, IL. Genes resistant to tetracycline have been found in groundwater as far as a sixth of a mile downstream from two swine facilities that use antibiotics as growth promoters.

Researchers from the University of Illinois and Illinois State Geological Survey used a DNA-amplification technique (polymerase chain reaction or PCR) to analyze samples from lagoons, wells and groundwater on and near two Illinois facilities. Their research appeared in the April issue of *Applied and Environmental Microbiology*.

For more information on this news release link to: http://www.eurekalert.org/

REF: Eureka Alert, May 1, 2001.

FDA Warns Consumers to Discontinue Use of Botanical Products that Contain Aristolochic Acid

Based on new information, the Food and Drug Administration (FDA) is advising consumers to immediately discontinue use of any botanical products containing aristolochic acid. These products may have been sold as "traditional medicines" or as ingredients in dietary supplements. Aristolochic acid is found primarily in the plant *Aristolochia*, but may also be present in other botanicals. Consumption of products containing aristolochic acid has been associated with permanent kidney damage, sometimes resulting in kidney failure that has required kidney dialysis or kidney transplantation. In addition, some patients have developed certain types of cancers, most often occurring in the urinary tract.

Previously, in May 2000, FDA alerted health care professionals and the dietary supplement industry of two patients in the United Kingdom who had experienced serious, permanent kidney damage following the use of botanical products containing aristolochic acid. These cases, along with the ones previously reported from Belgium and France, resulted in FDA imposing an import alert to detain botanical ingredients that are either labeled as "Aristolochia" or, for other reasons, are suspected to contain aristolochic acid. The ingredient will only be allowed to enter the U.S. when adequate testing shows that the suspect ingredients are free of aristolochic acid.

Recently, FDA has received reports of two patients in the U.S. who developed serious kidney disease associated with the use of botanical products that were shown by laboratory analysis to contain aristolochic acid. In addition, the agency analyzed a sample of 38 botanical products available in the U.S. that were labeled as containing aristolochia or other herbs that might contain aristolochic acid and found that 18 of these products contained aristolochic acid. Based on these analytical results, FDA has requested that the involved U.S. manufacturers or distributors initiate recalls of these products.

Due to the potential serious public health risk, the agency is now advising consumers to stop using any products that may likely contain aristolochic acid. This includes products with the words **"Aristolochia," "Bragantia" or "Asarum"** listed as ingredients on the label, or any of the products FDA has found to contain aristolochic acid (see list of the 18 products analyzed by FDA - www.cfsan.fda.gov/~dms/ds-bot3.html). Consumers cannot be assured that products containing these ingredients are free of aristolochic acid unless they have been tested in a laboratory.

FDA advises consumers who have taken any of these products of concern to contact their health care provider immediately. Even if these products have not been used recently, consumers should still inform their health care provider about which product they took, so that an appropriate evaluation may be conducted.

Current and past FDA activities and actions associated with aristolochic acid may be found at: www.cfsan.fda.gov/~dms/ds-bot.html.

For more recent information on Dietary Supplements see http://www.cfsan.fda.gov/~dms/supplmnt.html

REF: FDA Center for Food Safety and Applied Nutrition, Consumer Advisory, April 11, 2001.

Nitrate in Drinking Water Increases Bladder Cancer Risk

Nitrate in drinking water is associated with an increased risk for bladder cancer, according to a University of Iowa study that looked at cancer incidence among nearly 22,000 Iowa women.

The study results suggest that even low level exposures to nitrate over many years could be problematic in terms of certain types of cancer, said Peter Weyer, Ph.D., associate director of the UI Center for Health Effects of Environmental Contamination (CHEEC) and one of the study's lead authors. The study will be published in the May issue of the journal *Epidemiology*.

The researchers assessed nitrate exposure from drinking water for 21,977 women who were participants in the Iowa Women's Health Study. The women, who were between 55 and 69 years of age in 1986 (at the start of the study) resided in a total of 400 Iowa communities and had used the same drinking water supply for more than 10 years. Approximately 16,500 of the women received their water from municipal water supplies; the remaining women used private wells. Since no individual water consumption data were available, the researchers assigned each woman an average level of exposure to nitrate based on data collected between 1955 to 1988 on nitrate levels in her community's water supply. No nitrate data were available for women using private wells.

Using cancer incidence data from the Iowa Cancer Registry for 1986 to 1998, and after adjusting for factors such as smoking and nitrate in the diet, the researchers found a greater risk for bladder cancer as the nitrate levels in the communities' water supplies increased. Women whose average drinking water nitrate exposure level was greater than 2.46 milligrams (mg) per liter (nitrate-nitrogen) were 2.83 times more likely to develop bladder cancer than women in the lowest nitrate exposure level (less than 0.36 mg per liter).

While nitrate is produced naturally within the body, environmental sources include food (including many vegetables), contaminated drinking water, cigarette smoking and certain medications. Drinking water can account for a substantial proportion of the total nitrate intake. Up to 20 percent of ingested nitrate is transformed in the body to nitrite, which can then undergo transformation in the stomach, colon and bladder to form N-nitroso compounds. These compounds are known to cause cancer in a variety of organs in more than 40 animal species, including higher primates.

"The U.S. Environmental Protection Agency drinking water standard is 10 mg per liter nitrate-nitrogen. Our study suggests that nitrate levels much less than that could be a serious health concern," Weyer said. Weyer emphasized that additional studies are needed to look at possible links between nitrate levels in drinking water and cancer, particularly with respect to refining exposure assessments. "From a public health perspective, source water protection is a main concern. Sources of nitrate which can impact water supplies include fertilizers, human waste and animal waste," he said.

"All of us, rural and urban residents alike, need to be more aware of how what we do as individuals can impact our water sources and potentially our health."

The Iowa Women's Health Study, a long-term epidemiological study based in the UI College of Public Health, is funded by the National Cancer Institute.

REF: University of Iowa Health Center , April 30, 2001.

USDA/ERS Estimates Food-Borne Disease Costs

USDA's Economic Research Service has recently issued reports on food-borne illness. Food-borne diseases are caused by ingesting bacteria, fungi, parasites, or viruses through contaminated food or water or through person-to-person contact. Each year, microbial pathogens cause as many as 76 million cases of foodborne disease, including 5,200 deaths. ERS has estimated the annual U.S. economic costs incurred for the major bacterial pathogens: *E. coli* O157 and other STECs (and associated hemolytic uremic syndrome), *Campylobacter* (and associated Guillain-Barré syndrome), *Listeria monocytogenes*, and *Salmonella*. In addition, ERS has developed outcome trees for the illnesses caused by those pathogens, showing the costs incurred and the number of cases by the severity of disease: no physician visit, physician visit, hospitalization, premature death, and chronic complications.

ERS research estimates that the costs associated with five major pathogens alone amount to at least $6.9 billion annually. These costs include medical costs, productivity losses from missed work, and an estimate of the value of premature deaths, but exclude travel costs in obtaining medical care, lost leisure time, and so forth. ERS recently adjusted its procedure so as to account for the age distribution of those taken ill. The Centers for Disease Control and Prevention (CDC) has provided updated case, hospitalization, and death estimates for these foodborne pathogens. ERS has also revised its methodology to take account of age in valuing premature deaths. Under the age-adjusted approach, the assumed cost of each death ranges from $8.9 million for children who die before their first birthday to $1.7 million for individuals who die at age 85 or older. Because of changes in case estimates and the economic valuation of deaths, the ERS estimates are not strictly comparable with earlier ERS estimates of foodborne disease costs.

- ERS estimates that ERS estimates that, each year in the United States, foodborne *E. coli* O157:H7 disease costs $659.1 million to society and foodborne *E. coli* non-O157 STEC disease costs $329.7 million for a combined total of $988.8 million.
- ERS has estimated that, each year in the United States, the costs of the acute illness from foodborne *Listeria* are $2.3 billion.
- ERS has estimated that the annual economic costs due to foodborne Salmonella infections are $2.4 billion.
- ERS has estimated that, each year in the United States, the costs of the acute illness from foodborne *Campylobacter* (all serotypes) are $471.7 million and that the costs of GBS associated with foodborne *Campylobacter* are $358.8 million. Combining acute and chronic costs, foodborne *Campylobacter* causes an estimated $1.2 billion in costs to the United States each year.

For more on this report link to: http://www.ers.usda.gov/briefing/FoodborneDisease/

REF: Economic Research Service website, May 21, 2001.

Home invaders! Fight them without "chemical warfare"

They buzz and bite. Sprout to choke and blight. From mosquitoes and weeds to ants and fleas, pesky invaders are turning California homes, lawns, and gardens into battlefields this spring. How will you fight back?

The California Department of Pesticide Regulation urges Californians to avoid "chemical warfare" with pesticides whenever possible. Instead, DPR recommends integrated pest management (IPM) that works with nature to make it difficult for pests to survive, while encouraging beneficial plants, insects, and animals to flourish.

"Californians who want to protect our environment can start in their own backyard," said DPR Director Paul E. Helliker. "Our research shows that good pest management decisions at home can preserve the environment many miles away. For example, suburban Sacramento residents who minimize pesticide use and apply a few simple IPM techniques are protecting sources of water that flow into San Francisco Bay."

DPR recently released new fact sheets to help consumers manage pest problems with IPM. Basic IPM tips include:

- Pull the welcome mat. Install screens on windows and doors, seal cracks and crevices around your residence to keep pests out.
- Don't feed and water them. Fix leaky plumbing, eliminate standing water inside and out. Store food in tightly sealed containers, and keep your kitchen clean.
- Lighten up on the lawn. Mowing high helps grass, keeps weeds down. Spread organic mulches around shrubbery and hand-weed the lawn instead of using herbicides. If lawn and garden pesticides must be applied, make spot treatments and delay watering to prevent runoff.
- Set realistic goals. Pests have been around longer than humans. Learn to live with a few weeds and bugs with the objective of limiting their damage to acceptable levels. California consumers have more IPM resources than ever to help them fight "green and clean" this spring. Consumers and community organizations have free online access to the "H20 Home to Ocean Workbook." DPR created this online guide to educate the public on pesticide use and disposal issues. Flyers, brochures, and other resource materials in both English and Spanish are available free at <www.home2ocean.org>.

More sources for IPM information:

- For DPR advice about least-toxic controls for Argentine ants, fleas, and lawn grubs, see <www.cdpr.ca.gov/docs/schools/pm0102.pdf >.

- The University of California's IPM Online site (< www.ipm.ucdavis.edu/ >) offers extensive information on specific pests and pest management.
- The U.S. Environmental Protection Agency's "Healthy Lawn, Healthy Environment" booklet and other information may be found at < www.epa.gov/pesticides >.
- UC Cooperative Extension offices and County Agricultural Commissioners are knowledgeable on local pest management strategies. They are listed in the local government pages of the telephone directory. Phone listings are also available online from DPR at < www.cdpr.ca.gov/dprpestlnk.htm >.

REF: *Department of Pesticide Regulation Release*, 01-09, April 11, 2001.

Surveillance for Fatal and Nonfatal Firearm-Related Injuries United States, 1993--1998

Firearm-related injuries are the second leading cause of injury-related death in the United States.

Since 1993, firearm-related injuries and deaths have been declining steadily. However, in 1998, firearm-related injuries remained the second leading cause of injury death in the United States, accounting for approximately 31,000 deaths. The majority of these fatal and nonfatal firearm-related injuries result from interpersonal violence and intentionally self-inflicted gunshot wounds, but approximately 15,000 unintentional gunshot wounds are treated in U.S. hospital emergency departments (EDs) each year. Although firearm-related injuries represent <0.5% of injuries treated in hospital EDs, they have an increased potential of death and hospitalization compared with other causes of injury. **In 1994, treatment of gunshot injuries in the United States was estimated at $2.3 billion in lifetime medical costs, of which $1.1 billion was paid by the federal government. These factors emphasize the importance of firearm-related injuries as a public health concern.**

Results: During 1993-1998, an estimated average of 115,000 firearm-related injuries (including 35,200 fatal and 79,400 nonfatal injuries) occurred annually in the United States. Males were seven times more likely to die or be treated in a hospital ED for a gunshot wound than females. The proportion of firearm-related injuries that resulted in death increased from younger to older age groups. Approximately 68% of firearm-related injuries for teenagers and young adults aged 15-24 years were from interpersonal violence, and 78% of firearm-related injuries among older persons aged >65 years were from intentionally self-inflicted gunshot wounds. Black males aged 20-24 years had the highest average annual fatal (166.7/100,000 population) and nonfatal (689.4/100,000 population) firearm-related injury rates during the 6-year period. Although 51.4% of intentionally self-inflicted nonfatal wounds were to the head or neck, 71.8% of unintentional and 45.8% of assault-related nonfatal wounds were to the extremities. During the 6-year period, estimates are that quarterly fatal firearm-related injury rates declined 29.3%, and quarterly nonfatal firearm-related injury rates declined 46.9%. Firearm-related injury rates declined for intentionally self-inflicted, assault, and unintentional causes.

Interpretation: Data in this report regarding trends in firearm-related injury rates during 1993-1998 indicate that both

mortality and morbidity from gunshot wounds declined substantially in the United States. However, firearm-related injury continues to be a public health concern accounting for approximately 31,000 deaths and 64,500 nonfatal injuries treated in hospital EDs in 1998.

The entire report can be found at: http://www.cdc.gov/mmwr/

REF: Morbidity *and Mortality Weekly Report*, 50(SS02), April 13, 2001.

Baler and Compactor-Related Deaths in the Workplace United States, 1992--2000

Equipment that compacts and bales loose solid waste materials into denser, more easily transported units is common in refuse disposal and recycling and is used routinely at recycling centers, manufacturing facilities, and retail and wholesale stores to compress paper, textiles, metals, plastic, and other material. Persons operating balers and compactors can become caught by the powered rams of the compression chambers while using these machines. Risk factors resulting from these incidents have been identified through surveillance findings and results of investigations conducted by CDC's National Institute for Occupational Safety and Health (NIOSH) Fatality Assessment and Control Evaluation (FACE) program and the Bureau of Labor Statistics Census of Fatal Occupational Injuries (CFOI), a nationwide multisource reporting system for occupational deaths.

Case Reports

Case 1. In July 2000, a 16-year-old produce market worker died from crushing injuries when he was caught in the vertical downstroke baler he was operating. He was working alone in the market's basement and was using the baler to crush cardboard boxes when he was caught by the machine's ram. Investigations determined that the machine's safety interlock had been bypassed, allowing the machine to operate with the loading door open. The worker may have reached into the compression chamber while the machine was operating and was caught by the ram during its downstroke.

Case 2. In May 1997, a 34-year-old paper products worker died after falling into an operating baler. The worker and a co-worker were loading scrap paper into the baler through a belt conveyor when the material jammed in the baler's feed chute. The co-worker shut down the conveyor but not the baler's automatic controls, and the worker ascended to a platform between the end of the conveyor and the feed chute. When he leaned over the platform rail to clear the jam, he fell through the feed chute and into the compression chamber. His presence tripped the automatic control sensor, and the baler's ram was activated.

CFOI identified 34 deaths related to compactors and balers during 1992-1998; 29 (85%) occurred when a worker was caught or crushed by the compacting ram of the machine. Decedents were age 17-72 years (median: 37 years): six were <25 years, 10 were 25-34 years, nine were 35-44 years, and nine were >45 years. Twelve worked in the wholesale trade

industry; nine in manufacturing; eight in transportation/communications/public utilities; and the remainder in retail and services industries. Six deaths occurred during the processing of cardboard; five workers were processing paper; five were processing trash; and five were processing cans, scrap metal, cotton, or plastic wrap. For eight deaths, the material being processed was not specified.

Field investigations that identified injury risks were conducted for 11 incidents. Nine involved **failure to implement effective power supply shutdown and ram pressure dissipation procedures, six involved failure to follow standard procedures for clearing material jams, six involved attempting to clear material jams without shutting down the machine's automatic controls, five involved operating machines with bypassed or defective safety interlocks, and three involved workers' operating a machine without determining the location of co-workers.**

Editorial Note: On the basis of information collected from FACE investigations, the following measures are recommended to reduce the risk for worker injury in compactors and balers: 1) employers should train workers to recognize the hazards of operating or working near balers and compactors; 2) before jams are cleared, authorized employees should verify that the machine's electrical power has been disconnected, the disconnecting device has been locked and tagged, and the ram pressure has been dissipated (3); employers should implement appropriate power supply shutdown procedures whenever repair or maintenance is needed (4); 3) employers should implement standard procedures for managing common events such as material jams; 4) balers and compactors should be equipped with machine guards and safety interlocks to prevent worker injury and interlocks should be designed so that they cannot be bypassed; and 5) employers should require machine operators to account for the location of co-workers before activating compactor or baler rams.

REF: *Morbidity and Mortality Weekly Report*, 50(16), April 27, 2001.

State Health Director Advises Consumers to Scrub Cantaloupe Before Eating

Sacramento - Following a recent outbreak of *Salmonella* foodborne disease associated with eating cantaloupe in California and seven other states, State Health Director Diana M. Bontá, R.N., Dr.P.H., reminded consumers to always thoroughly wash the skin of all fruits and vegetables that are eaten raw before consuming them.

"Contamination can occur when a consumer cuts through a cantaloupe rind that has not been scrubbed with a brush under cool, running water immediately before eating," Bontá said. To reduce the chance of contamination, Bontá said consumers should also wash their hands before and after handling the fruit and refrigerate unused cut portions immediately.

California reported 17 illnesses and the death of a Riverside woman from contaminated cantaloupes between April 6 and April 24. Thirteen other cases of illness were reported in Arizona, Missouri, New Mexico, New York, Oregon, Tennessee and Washington in the same outbreak.

An uncommon type of *Salmonella*, known as *Salmonella Poona*, caused the outbreak in California. Five individuals in Los Angeles, three in Orange, two each in Riverside, San Bernardino and Ventura and one each in Alameda, San Benito and San Diego counties became ill. Six of the ill individuals, including the woman who died, were over 60 years of age and five were children under 5 years. While most of the individuals ate melons that were purchased whole and cut at home, some ate pre-cut cantaloupes purchased from supermarkets or were served cantaloupes in restaurants.

The source of the contamination is under investigation. California Department of Health Services investigators said it is likely that contaminated fruit was imported into the United States. Domestic production of cantaloupes has not begun in California and Arizona, and production has only recently begun in Texas.

Cantaloupe has been implicated in previous *Salmonella outbreaks*, including a multistate outbreak of more than 46 cases (26 in California) due to *Salmonella Poona* in 2000, 400 cases due to S*almonella Poona* in 1991 and an outbreak in California of more than 20 cases due to *Salmonella Saphra* in 1997.

Because cantaloupes are grown on the ground, their skin can become contaminated in the field by human or animal waste, or during distribution prior to sale.

The symptoms of *Salmonella Poona* include fever, abdominal cramps and diarrhea. The symptoms generally develop one to three days after eating contaminated food. While most individuals who become ill from *Salmonella Poona* recover in three to five days without medical intervention, the infection can be life threatening to young children, the elderly and those with compromised immune systems. Consumers should consult with their physician if they have these symptoms.

REF: California Department of Health Services < http://www.dhs.ca.gov > Office of Public Affairs Press Releases (37-01), May 15, 2001.

Fatal Occupational Injuries - United States, 1980-1997

The Centers for Disease Control and Prevention monitors deaths from occupational injuries through the National Traumatic Occupational Fatalities (NTOF) surveillance system. This report provides an overview of traumatic occupational deaths among civilian workers from NTOF from 1980 through 1997, the most recent year for which data are available. The data presented in this report indicate a decrease in occupational deaths over this period with mining, agriculture/forestry/fishing, and construction having the highest death rates; motor-vehicle crashes were the leading cause of injury-related deaths for U.S. workers. State health departments and others involved in prevention of occupational injuries can use the data to prioritize intervention programs.

National Estimates

During 1980-1997, 103,945 civilian workers died in the United States from occupational injuries, an average of 16 work-related deaths per day. The annual number of traumatic occupational deaths declined 28%, from 7343 in 1980 to

5285 in 1997. The rate for occupational injury deaths for all workers decreased 45%, from 7.4 per 100,000 workers in 1980 to 4.1 in 1997.

Males accounted for 93% of all deaths, with a death rate approximately 11 times that of females. Although 85% of civilian workers who died were white, blacks had a higher fatality rate (5.6 per 100,000 workers) than whites (5.0). Workers aged 25-34 years accounted for the largest number of occupational injury deaths, and workers aged >65 years had the highest age-specific death rate.

Since 1980, motor-vehicle crashes accounted for 24% of deaths and were the leading cause of injury-related death for U.S. workers. In 1990, homicides became the second leading cause of occupational injury deaths (14%), surpassing machine-related deaths (13%). Deaths caused by falls and electrocutions accounted for 10% and 7% of work-related deaths, respectively.

The industries in which the largest numbers of deaths occurred were construction (19% of reported deaths), transportation/communications/public utilities (17%), and manufacturing (15%). Industries with the highest death rates were mining (30 per 100,000 workers), agriculture/forestry/fishing (19), and construction (15).

The risk for specific causes of death varied by industry. Machinery was the leading cause of death in agriculture/forestry/fishing, mining, and manufacturing. Falls were the most prevalent in construction, followed by motor-vehicle crashes, and machinery. Motor-vehicle crashes were the leading cause of death in transportation/communications/public utilities, wholesale trade, and public administration. Homicide was the leading cause of death in retail trade, finance/insurance/real estate, and services.

The occupation categories in which the largest number of deaths occurred were precision production/craft/repairers (21%), transportation/material movers (18%), and farmers/foresters/fishers (13%). Occupation categories with the highest death rates were farmers/foresters/fishers (21.4 per 100,000 workers), transportation/material movers (21.3), and handlers/equipment cleaners/helpers/laborers (13.4).

State Estimates

The greatest number of fatal occupational injuries occurred in California (10%), Texas (10%), Florida (6%), Illinois (4%), and Pennsylvania (4%). Fatal occupational injury rates were highest in Alaska (22.7 per 100,000 workers), Wyoming (15.8), Montana (11.8), Idaho (10.4), and West Virginia (10.1). The leading causes of death varied for each of these five states. For example, water transport accounted for the most deaths in Alaska (33%), compared with approximately 2% for the United States, and air transport was the second or third leading cause of death in four of the five states, compared with being the seventh overall cause of death nationally.

Editorial Note: The findings in this report indicate a general decrease during 1980-1997 in the annual number of deaths and the annual rates of occupational deaths in the United States. In addition, the leading causes of death have changed through the 1990s. Although surveillance data cannot identify reasons for these temporal trends, changes in the workplace (e.g., increased and better targeted regulations, improved hazard awareness, new technology, and mechanization) are possible factors. In addition, changes in the economy, the industrial mix, and the distribution of the workforce and improvements in acute trauma care for injured workers may have contributed to these decreases.

REF: *Morbidity and Mortality Weekly Report*, 50(16), April 27, 2001.

Motor-Vehicle Occupant Injury: Strategies for Increasing Use of Child Safety Seats, Increasing Use of Safety Belts, and Reducing Alcohol-Impaired Driving

Summary

The Task Force on Community Preventive Services has conducted systematic reviews of interventions designed to increase use of child safety seats, increase use of safety belts, and reduce alcohol-impaired driving. The Task Force strongly recommends the following interventions: laws requiring use of child safety seats, distribution and education programs for child safety seats, laws requiring use of safety belts, both primary and enhanced enforcement of safety belt use laws, laws that lower the legal blood alcohol concentration (BAC) limit for adult drivers to 0.08%, laws that maintain the minimum legal drinking age at 21 years, and use of sobriety checkpoints.

Background

Motor-vehicle-related injuries kill more children and young adults (i.e., those aged 1-24 years) than any other single cause in the United States and are the leading cause of death from unintentional injury for persons of all ages. Approximately 41,000 persons in the United States die in motor-vehicle crashes each year. Moreover, crash injuries result in approximately 500,000 hospitalizations and 4 million emergency department visits annually.

Viewed from an economic perspective, crash injuries and deaths are a burden to society. Motor-vehicle-related deaths and injuries cost the United States approximately $150 billion annually, including $52.1 billion in property damage, $42.4 billion in lost productivity, and $17 billion in medical expenses. Alcohol-related crashes contribute substantially to these costs, with a direct economic impact of approximately $45 billion in 1994 alone.

Reducing motor-vehicle injury remains a formidable public health challenge, despite sharp declines in motor-vehicle-related death rates since 1925. Use of child safety seats and safety belts and deterrence of alcohol-impaired driving are among the most important preventive measures to further reduce motor-vehicle occupant injuries and deaths. This report provides recommendations on interventions to increase use of child safety seats, increase use of safety belts, and reduce alcohol-impaired driving.

To read the entire report link to: http://www.cdc.gov/mmwr/preview/mmwrhtml/rr5007a1.htm

REF: *Morbidity and Mortality Weekly Report*, 50(RR07), May 18, 2001.

Toxicology Tidbits

Preliminary Risk Assessment Available for Atrazine

The Environmental Protection Agency (EPA) has posted the preliminary human health risk assessment for atrazine on its web page (www.epa.gov/pesticides/). A member of the class of chemically similar compounds known as the triazines, atrazine is one of the most widely used agricultural pesticides in the U.S. About 80 million pounds of atrazine active ingredient are applied annually to control broadleaf weeds in field corn and sorghum in the Midwest; in lawns and turf; and in wheat, pineapples, sugarcane, and macadamia nuts. EPA's preliminary health risk assessment evaluates risks associated with atrazine, including potential hazards to children. While exposure through food is not of concern, the assessment indicates exposure to children through drinking water may be of concern in some areas, as well as exposure from treated lawns.

Atrazine is the most commonly detected pesticide in ground water and surface water. EPA's Cancer Assessment Review Committee classified atrazine as "Not Likely to be Carcinogenic to Humans" in accordance with the draft Guidelines for Carcinogen Risk Assessment (July 1999). The FIFRA Scientific Advisory Panel's consensus supports EPA's cancer classification..

For more information link to: < http://www.epa.gov/oppfead1/cb/csb_page/updates/atrazine.htm >

NTP Final Report, Endocrine Disruptors Low Dose

Summary

The National Toxicology Program (NTP)/National Institute of Environmental Health Sciences (NIEHS) organized and conducted a scientific peer review at the request of the US Environmental Protection Agency (EPA) to evaluate reported low-dose reproductive and developmental effects and dose-response relationships for endocrine disrupting chemicals. The NTP is soliciting public comment prior to transmitting the final report to the US EPA. Public comments received in response to this solicitation will be included in the final transmittal. The final report is available on the NTP

web site at http://ntp-server.niehs.nih.gov

REF: *Federal Register*, 66(95), May 16, 2001.

Information on Arsenic

The 1996 amendments to the Safe Drinking Water Act requires the EPA to revise the existing drinking water standard for arsenic. The **EPA's Office of Ground Water & Drinking Water - Arsenic's website** < http://www.epa.gov/safewater/arsenic.html > contains the latest information about the recent changes in the rulemaking efforts for this standard. As more information becomes available, the site will continue to be updated. The site also contains information about the history of the EPA's rulemaking efforts for this standard including the project plan, implementation guidelines, the proposed timeline, strategies, meeting summaries, upcoming events, *Federal Register* notices, and how the public can comment on the proposed regulations.

Most arsenic found in ground water is a result of minerals that have dissolved from weathered rocks and soils. The **U.S. Geological Survey (USGS)** has collected and analyzed arsenic levels from 18,850 wells in 595 counties across the United States. Arsenic concentrations in these wells are similar to those found in nearby public supplies, thus producing a more accurate view of arsenic concentrations. On the USGS's website < http://co.water.usgs.gov/trace/arsenic/ >, you can find a map created from this data that shows where and to what extent arsenic occurs in ground water across the country. The highest amounts were found in the West, but also in the Midwest and Northeast. The site also contains a fact sheet, a faq, and other publications and data on this map project as well as links for both arsenic and drinking water resources.

The **University of California, Berkeley's Arsenic Research Program** is run by staff at the School of Public Health. The research done here began over ten years ago and originally focused on the risk of cancer. International projects are currently ongoing in Argentina, Chile, India, and Bangladesh. In the United States, projects are ongoing in California and Nevada. Funding sources include the National Institutes for Environmental Health Sciences, the U.S. Environmental Protection Agency, and the California Cancer Research Program. The website < http://socrates.berkeley.edu/~asrg/ > contains information on the various research projects and findings, publications, and the researchers at UC-Berkeley as well other institutions in the US, Chile, Argentina, and India.

REF: *Internet Newsbrief*, April 13, 2001.

Bushwhacked by Arsenic? Part I: Toxic Terror & Treated Wood

--- Dr. Allan S. Felsot, Environmental Toxicologist, WSU

Treated wood and drinking water don't have much in common, but both contain arsenic. This ubiquitous, naturally occurring element follows the laws of toxicology like everything else, so no doubt overexposure can lead to some nasty health effects. But what is the likelihood (i.e., risk) that you will be bushwhacked by arsenic from exposure in water or treated wood?

To read this entire article link to: Agrichemical and Environmental News

REF: *Agrichemical and Environmental News, 182*, June 2001.

FDA Consumer Warnings

Possible Health Risk Associated with Certain Candy Lollipops from Mexico: FDA is warning consumers not to purchase or consume tamarind candy lollipops labeled Dulmex brand "Bolirindo" because of high levels of lead. Consuming high levels of lead can cause serious damage to the central nervous system, especially in children.
http://www.fda.gov/bbs/topics/ANSWERS/2001/ANS01079.html

Consumers Warned About Mislabeled Poisonous Autumn Monkshood Plant: FDA is warning consumers not to consume a plant called Autumn Monkshood. The plants were distributed to nurseries in British Columbia, Washington State, Idaho, and possibly elsewhere. The packages were mistakenly labeled "All parts of this plant are tasty in soup" but the plant is actually poisonous.
http://www.fda.gov/bbs/topics/ANSWERS/2001/ANS01080.html

Consumers Warned About VIVA Brand Imported Cantaloupe: FDA is advising consumers of an outbreak of foodborne illness associated with cantaloupe from two Mexican companies, S.P.R. De R.I. Legumbrera San Luis and S.P.R. De R.I. Los Arroyes, and imported by Shipley Sales Service of Nogales, AZ. This outbreak of *Salmonella poona* has involved numerous illnesses and two deaths in Arizona, California, Connecticut, Georgia, Hawaii, Massachusetts, Minnesota, Missouri, New Mexico, Nevada, New York, Oregon, Tennessee, and Washington State.

http://www.fda.gov/bbs/topics/NEWS/2001/NEW00760.html

FSIS Releases Summer Food Safety Info

USDA's Food Safety and Inspection Service (FSIS) has put together important information on how consumers can protect themselves during the warm season. Access this important information on the FSIS website at www.fsis.usda.gov/oa/pubs/illpeaks.htm . Also available is information on Barbecue Food Safety. FSIS has gathered simple, yet important guidelines for grilling food safely to prevent harmful bacteria from multiplying and causing foodborne illness. Access this information online at www.fsis.usda.gov/oa/pubs/facts_barbecue.htm .

Parasitic Pathways - Swimming Pools/Recreational Water

Swimming is one of the most popular activities in the country. Millions of people swim safely each year. However, disease transmission can occur while swimming. The following link from the Centers for Disease Control and Prevention provides information and guidelines to help you avoid getting an infectious disease or getting injured while swimming in "recreational water," such as, swimming pools, waterparks, lakes, rivers, and the ocean.

< http://www.cdc.gov/ncidod/dpd/parasiticpathways/swimming.htm >

Water Treatment Devices

The California Department of Health Services (DHS) evaluates and certifies residential drinking water treatment devices for reduction of health related contaminants. These include contaminants such as lead, cryptosporidium (protozoan cysts), pesticides, herbicides, solvents, heavy metals, bacteria and virus. The Department does not regulate devices that make aesthetic claims. Aesthetic claims include improvement in taste, odor and appearance. Also, the Department does not regulate backpacking or camping filters, sports bottles, shower filters or water softeners.

DHS Prevention Services has a Drinking Water Program that lists a 2001 Directory of Certified Water Treatment Devices and can be found at: www.dhs.ca.gov/ps/ddwem/technical/certification/device/table.htm.

ConsumerLab.com Fails One-third of Tested St. John's Wort Products

ConsumerLab.com says one-third of the St. John's wort products it tested failed for not containing the amount of labeled ingredients or exceeding the level of cadmium it believes is safe. "

The independent testing firm said four of the 21 products it tested contained only 77% to 85% of the labeled amount of the chemical marker hypericin or, if not labeled, of the amount used as a minimum level that is "consistent with most clinical research." One of two products claiming to contain the marker hyperforin was found to have only 21.7% of the claimed amount.

REF: *Food Chemical News*, Volume 43(9), April 16, 2001.

VETERINARY NOTES.....

USDA Prepares Mandatory Inspection of Squab and Ratites

USDA is preparing to begin mandatory inspection of squab and ratites, following through with a congressional order to include the birds among the creatures that the government must inspect before they can be sold for human food. Until now, plants slaughtering and processing the animals have had to pay out of pocket for federal inspection services because they are not covered under inspection laws. Most have paid for the services because it is good for business to the have the inspection stamp of approval. Congress mandated that the birds be included last year. The inspection change will take effect April 26, and USDA has been given some extra funding to carry out its expanded inspection task.

FSIS Announces Mandatory Inspection of Ratites and Squabs

The Food Safety and Inspection Service announced that an interim final rule to include ratites and squabs under mandatory poultry inspection regulations has gone on public display and will be published in the *Federal Register* on May 1, 2001. Ratites are flightless birds such as ostriches, emus, and rheas. Squabs are young pigeons that have not yet flown. Some ratites and squabs have been inspected under the agency's voluntary poultry inspection program. Under this voluntary inspection, establishments pay a fee for inspection services. However, as part of the FY 2001 Agriculture Appropriations Act, $2.5 million was appropriated for their mandatory inspection. As a result of this action, U.S. establishments slaughtering or processing ratites or squabs for distribution into commerce as human food will now be subject to mandatory requirements of the Poultry Products Inspection Act and will no longer need to pay a fee for inspection.

Operators who wish to continue to slaughter or process ratites and squabs must apply to FSIS for a grant of inspection for mandatory inspection service. Establishments that slaughter and process ratites and squabs will be required to implement and validate sanitation standard operating procedures and Hazard Analysis and Critical Control Point systems, as required by mandatory poultry inspection regulations.

http://www.fsis.usda.gov/OA/news/2001/ratitesquab.htm

"Antimicrobial Drug Use and Veterinary Costs in U.S. Livestock Production"

Antimicrobial drugs are fed to animals at low levels to treat diseases, to promote growth, and to increase feed efficiency. Incorporating low levels of antimicrobial drugs in livestock feeds has been shown to be a factor stimulating the development of antimicrobial drug-resistant bacteria in livestock. Since many of the drugs used to treat livestock are the same as or are related to drugs used in human health care, there is concern that resistant organisms may pass from animals to humans through the handling of animals or food derived from animals. The movement of pathogens from animals to humans, and vice versa, has been documented, but the extent to which it has occurred or could occur is unknown. Although it is estimated that as little as 10 percent of the problems of drug-resistant pathogens in humans originate in livestock health care practices, there is currently considerable debate about the frequency and costs of human disease outbreaks resulting from animals infected with drug-resistant pathogens. Several European countries have banned the growth-promoting use of antimicrobial drugs in livestock production as a precautionary measure to prevent resistant organisms from passing from animals to humans. This report present preliminary estimates suggesting that discontinuing the use of antimicrobial drugs in hog production would initially decrease feed efficiency, raise feed costs, reduce production, and raise prices to consumers. Longer term effects were not examined.

This USDA Economic Research Service publication can be found at: http://www.ers.usda.gov/publications/aib766/

Pathogen Load Report Available

FDA's Center for Veterinary Medicine (CVM) contracted with Exponent of Alexandria, Virginia to conduct a review of published literature on the effect of using antimicrobials in food-producing animals on pathogen load. This review has been completed, and the report is available at http://www.fda.gov/cvm/antimicrobial/antimicrobial.html .

In November 1998, FDA's CVM published in the *Federal Register* a notice of availability for the draft guidance document entitled " Consideration of the Human Health Impact of the Microbial Effects of Antimicrobial New Animal Drugs Intended for Use in Food-Producing Animals " (Guidance for Industry #78). In this guidance document, CVM stated that the Agency intended to consider the potential human health impact of the microbial effects associated with all uses of all classes of antimicrobial new animal drugs intended for use in food-producing animals. The guidance said that in order to assess this impact, it may be necessary to evaluate the following two separate, but related aspects: 1) the rate and extent of development of antimicrobial drug resistant enteric bacteria formed in the animal's intestinal tract following exposure to the antimicrobial new animal drug (resistance); and 2) changes in the number of enteric bacteria in the animal's intestinal tract that cause human illness (pathogen load).

On February 22-24, 2000, CVM held a public scientific meeting on pre-approval studies in antimicrobial resistance and pathogen load. The purpose of this workshop was to discuss the appropriate designs for pre-approval studies to evaluate the potential microbial effects associated with the use of antimicrobial drugs in food-producing animals. The slide presentations from this meeting as well as the complete transcripts are available at (http://www.fda.gov/cvm/antimicrobial/oldmeet.htm). At the February 2000, meeting, CVM received numerous comments questioning the relevance of conducting studies to try to assess the impact of drug effects on pathogen load.

CVM recognizes that scientific information in this area is limited and acknowledges the concerns raised at the February 2000 public meeting. Therefore, in an attempt to gather additional information on the topic, CVM contracted with Exponent to conduct this literature review entitled "Effect of the use of antimicrobials in food-producing animals on pathogen load: Systematic review of the published literature." This report represents just one component of CVM's ongoing effort to complete a thorough review of the pathogen load issue and to develop appropriate policy in this area. CVM is planning to seek further scientific input on this issue and will announce its intentions in the near future.

http://www.fda.gov/cvm/index/updates/pathload.htm (May 2, 2001)

"Mad Cow" Page Updated

FDA has added several information sources to its Web page dedicated to bovine spongiform encephalopathy (BSE), including April 4 congressional testimony from FDA Center for Veterinary Medicine Director Stephen Sundlof.

Sundlof testimony: http://www.fda.gov/ola/2001/bse0401.html
BSE page: http://www.fda.gov/oc/opacom/hottopics/bse.html
and http://www.fda.gov/oc/oca/roundtable/bse

Animal Toxicity Cases

Oleander toxicosis caused listlessness for two days prior to the death of a Barbados eye in Southern California. Oleandrin is the toxic glycoside found in all portions of the dry and green plant. In sheep, ingestion of two to three leaves can be fatal. Clinical signs involve the gastrointestinal tract and heart, and can include abdominal pain, vomiting, anorexia, rapid and weak pulse, cardiac arrhythmias, hypotension and hypothermia. Death can occur suddenly without signs.

Oleander toxicosis was diagnosed in nine horses from seven premises. The affected horses either died suddenly or had anorexia, dehydration, diarrhea, ventricular tachycardia and fever. In one case, the source of oleander was accidental contamination of hay. One affected animal was a foal with minimal lesions.

Several goats died from acute **avocado toxicity** with signs of lethargy and drinking excessive water. The rumen contained avocado leaves, and histopathology revealed acute cardiac edema, hemorrhage and necrosis.

Strychnine was found in the stomach contents of a 4-year-old Quarter house gelding on pasture. The horse was noted

to be very stiff, suddenly fell down, developed difficulty breathing and died within 15 minutes. Strychnine is the active ingredient in many rodent, gopher and coyote baits. Clinical signs included a stiff, sawhorse stance, violent tetanic seizures and death from anoxia.

REF: *Lab Notes* (UC Davis), 14(2), Spring 2001.

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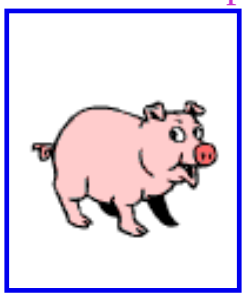

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