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Animal Health Centre

Ministry of Agriculture, Food and Fisheries

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Editor:

J.W. Coates

Mailing Address:

Ministry of Agriculture,
Food and Fisheries
Animal Health Centre
1767 Angus Campbell Road
Abbotsford, BC V3G 2M3

From the Director

R.J. Lewis:

I am certain there is no one reading this that is unaware of the current ongoing crisis affecting the Canadian cattle industry. The identification of a single beef animal in Alberta affected by bovine spongiform encephalopathy (BSE) or mad cow disease continues to cost the industry $11 million dollars daily, beginning May 20. In addition to the effects on the cattle industry, export of other livestock unaffected by BSE such as sheep, goats, llamas, and elk, have also been impacted due to this ban. Wildlife guides and outfitters are negatively affected since meat from wild ruminants also cannot be exported to the United States where many of the hunter-clients originate.

A great deal of information in the media relates to the very significant costs that are resulting from the discovery of this single affected animal. Although we hear of the large amounts of money that are being lost, most of us are unable to contemplate such large sums. The most significant impact falls outside of these frequently-quoted massive monetary losses but relates to *individual* large losses for hard-working individuals whose equity and livelihood is being destroyed.

Canada has an enviable global reputation for producing high quality and safe animal products. The thorough investigation that the Canadian Food Inspection Agency launched to ensure there are no additional cases has been lauded as exemplary by an independent scientific commission comprised of international BSE experts. The identification of this single affected animal demonstrates that our inspection system works.

Additional stringent measures and more extensive surveillance systems are being implemented to build upon an already high quality inspection system. We should be proud of our food system and remain confident that the risk to any of us relating to BSE is, at most, negligible. On behalf of the farm families and others suffering from this export ban, we need to continue to demonstrate our confidence in their products by supporting them any way we can.

Continued Page 2

TOLL FREE NUMBER AND WEB SITE:

Please note that the Animal Health Centre has a toll free number: 1-800-661-9903. Keep this in mind, if calling long distance. This Newsletter, and other information from the AHC, may also be found on the Internet at our web site : http:// www.agf.gov.bc.ca./croplive/anhlth/ahc

To date (August 1st), we have examined 1,438 corvids (crows, jays, ravens, magpies) submitted by local health authorities from most areas of BC - all remain negative for West Nile Virus (WNV). One hundred and seventeen WNV-positive birds have been identified this year from Alberta, Manitoba, and Saskatchewan. There are no positive human cases in Canada, thus far. There are seven positive horses in Canada, to date – Alberta (3), Manitoba (2), and one each in Sask. and Ontario. The virus has not been identified in any states bordering British Columbia in 2003.

Several interesting articles arising from submissions to the Animal Health Centre, as well as materials that have been excerpted from other sources, is included in this issue. We hope that you will find this information of value and, as always, we welcome any comments or suggestions you may have to help us improve this communication.

Short cuts from the Post Mortem Room:

J Coates

Bronchopneumonia in lambs aged six weeks:

A lamb was submitted for necropsy following the loss of five others at pasture; the number of animals within the flock was not reported. The lamb had severe bronchopneumonia involving the anterior lung lobes as well as large portions of the more posterior (diaphragmatic) lobes. Severe inflammation of the lung pleura (fibrinous pleuritis) was also present. Standard bacterial culture yielded *Mannheimia (Pasteurella) hemolytica* and *Arcanobacterium pyogenes,* which were considered the major players in the death of these animals. An additional test for *Mycoplasma spp* involvement proved positive, with the identification of *M ovipneumoniae* by the polymerase chain reaction test (PCR- molecular diagnostics).

In combination with pasteurellae in sheep*, M ovipneumoniae* may produce a proliferative and exudative pneumonia *(*Radostits et al, *Veterinary Medicine*, *9th ed)*. The organism can often be isolated from the lungs, trachea and nasal cavities, and occasionally from the eyes of sheep with pneumonia – though it is also found in the respiratory tract of healthy sheep.

The natural disease caused by *M. ovipneumoniae* has often been described. Some studies have suggested the existence of synergistic action with *Pasteurella multocida and/or Mannheimia hemolytica* in the production of pneumonias that mainly affect animals under one year of age. Mortality rarely exceeds 10% under experimental conditions, although economic losses are considerable due to delayed livestock growth and the culling of animals that otherwise might have been retained within the flock.

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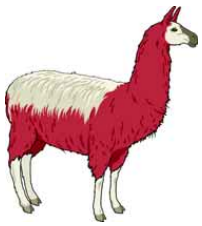

Multiple congenital anomalies in a newborn llama:

A newborn male llama was submitted for necropsy following euthanasia. Multiple anomalies of the skull were observed. The animal had marked elongation of the lower jaw (mandibular prognathism). In addition, there was marked 'wry-face', with twisting of the cranial premaxillary bones. There was also deformation of the nasal turbinate bones of the skull as well, a congenital malformation referred to as choanal atresia.

Choanal atresia refers to a membranous or bony blockage (as in this specimen) between the nasal turbinate sinuses (passage ways of the nose) and the pharyngeal area of the throat. Because nasal secretions flow or discharge continuously from the nose, they cannot be swallowed. Worse, there can be no breathing or inhalation by

the animal unless the mouth is opened. In this particular newborn, the combined deformations of wry-face and choanal atresia would have rendered normal suckling almost impossible, and its survivability very low.

Choanal atresia is very uncommon in other domestic animals and its precise etiology in any one case remains uncertain. In humans this rare condition appears sporadically, although in some instances there has been a suspected genetic link within family members.

Congenital defects are abnormalities of structure or function present at birth, and are of chromosomal, genetic, environmental, or unknown cause; some are caused by environmental-genetic interaction. Any body system can be affected, and defects vary from blemishes to severe malformations. The most commonly observed defects in all species involve brain, bone, muscle, and skin (*Leipold et al: Congenital Defects in the Llama; The Veterinary Clinics of North America, vol. 10, No. 2, 1994*).

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Vitamin E-related encephalomalacia in organically fed broilers:

A group of poor-doing organically-fed broilers, aged six weeks, was submitted for necropsy. Initial losses were reported as 3% per day and had risen to 15-30 birds per day, in a flock of 5000. Some birds were found recumbent on their backs.

The birds were small (300 to 600 grams) and emaciated, together with severe rickets characterized by markedly thickened growth plates within the proximal tibial-tarsal bones. Chronic-active cecal coccidiosis was also present, with frank or tarry blood deposited within cecal contents. Numerous coccidial parasites were obsrved microscopically within the damaged cecal mucosa and fecal flotation revealed high numbers of coccidial oocysts (eggs). Brain tissue revealed areas of subacute brain degeneration (encephalomalcia) that is typical of nutritional vitamin E deficiency, especially within the cerebellum.

The case was referred to the owner's veterinarian with recommendations to review the nutritional status of the flock as well as to address concerns with cecal coccidiosis.

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An unusual degenerative muscle lesion (myopathy) in broilers:

J. Coates

Recently, killed broilers have been submitted to the AHC for additional study following condemnation at a meat-processing plant for bacterial cellulitis. Cellulitis refers to inflammation of the tissue under the skin, commonly seen in association with infecting bacteria (*Dorland's Medical Dictionary, 28 th edition)*. Two groups of broilers approximately five weeks of age were submitted for examination. Both groups demonstrated severe muscle degeneration (necrosis) of primarily one specific muscle band over the dorsal back area. The muscle band, resembling a smaller version of the mammalian trapezius muscle, originates from the dorsal vertebral spinous processes of the mid-thoracic vertebrae and attaches to the scapula and adjacent forearm.

Degenerative changes were noted occasionally on microscopy in adjacent muscle groups but were consistently much milder, if present. Most cases demonstrated variable subcutaneous edema. In addition, several cases had concurrent lesions of muscle inflammation (myositis). However, bacterial cultures revealed minimal or very light mixed miscellaneous bacterial growth, with a noted absence of hemolytic *Staphylococcus sp*.

Additional follow-up on this interesting form of skeletal muscle degeneration in broilers will doubtless follow. The origin of the muscle lesion is speculative although the lesion resembles a form of exertional myopathy. Excessive 'wing flapping' by the birds might have initiated the muscle degenerative process. Other possible considerations are a link to reduced dietary vitamin E-selenium levels, ionophore toxicity, genetic predispositions, or unusual environmental stimuli/ irritants. In some of the sections examined microscopically, the inflammatory component was minimal to mild despite severe necrosis and proliferation of extensive 'reparative' muscle fibrous tissue. This observation suggested that any inflammation was a secondary response to primary muscle degeneration and necrosis.

– with thanks to Dr Ken Moll, CFIA veterinary inspector at Abbotsford, who submitted the birds for examination together with a detailed history. Ed.

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Acute septicemic coliform meningitis (inflammation of the brain membranes) was diagnosed in three week-old commercial ducklings, together with severe inflammation of the spleen and head sinuses (infra-orbital sinusitis). A moderate to heavy growth of *E coli* was cultured from affected tissues.

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Severe goiter (thyroid hyperplasia) from stillborn goat kids was diagnosed microscopically in thyroid tissue submitted to the laboratory by a veterinary practitioner. The owners had complained to their veterinarian that all their newborn kids were stillborn, or died shortly after birth. The practitioner observed enlarged thyroid glands while performing necropsies on submitted stillborns. Histopathology on the thyroid tissue confirmed the diagnosis. Goiter in newborns is usually linked to severe fetal deficiency of the essential element iodine.

In this writer's view, any fixed or fresh tissue submissions for histopathology from any fetus, newborn, or young animal are essentially incomplete at an elementary level, unless fixed thyroid is provided.

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Inflammation of brain tissues (meningoencephalitis) was diagnosed in a mature three year-old Shorthorn bull. The animal had been displaying vague nervous signs prior to its death, including stumbling and loss of balance. Because a skunk had been seen earlier in the barn, the owner was concerned about the possibility of rabies. Adequate portions of fresh brain were retained frozen for rabies virus investigation, pending the results of traditional laboratory investigation. However *Listeria monocytogenes*, a significant bacterial pathogen in many species including cattle and other ruminants, was cultured from brain tissue. In addition, the location and nature of the brain inflammatory reaction was also consistent with listerial encephalitis.

Practitioners are reminded to send in as much of the brain as possible when requesting laboratory tests for *L monocytogenes*. Since the brain lesions in listerial encephalitis are usually found in the brain stem, medulla and cerebellum, a submission limited to pieces of cerebral cortex may lead to inconclusive or negative bacterial culture and histopathological results.

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Bacterial embolic (blood-borne) inflammation of the brain tissue was diagnosed in a group of 14 day-old chicks. Coccoid organisms consistent with *Staphylococcus sp* were observed microscopically within the brain lesions. The primary source of the bacterial organism, or the environmental/husbandry factors enabling the bacteria to gain entry into the chicks' blood circulation, was not determined.

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Nutritional muscle degeneration (myopathy) in a four day-old purebred foal:

Extensive skeletal muscle degeneration and necrosis was observed microscopically in a four day-old purebred Newfoundlander filly. The animal had collapsed and died overnight following a brief illness characterized clinically as colic. Heart muscle lesions were not readily observed, and thyroid tissue was also unremarkable on microscopic examination. Liver analysis indicated selenium levels at 0.22 ppm, wet weight. Unfortunately, liver vitamin E levels were not determined, as the technique presently used in this laboratory for vitamin E measurement requires blood serum from the live animal. Blood gammaglobulin levels were high, indicating good colostral intake following birth. The muscle lesion that was observed microscopically represents a form of nutritional myopathy in this young animal. Inadequate provision of vitamin E and selenium to the developing fetus during gestation would be the likely principal factor. Ensuring sufficient dietary vitamin and trace mineral levels to pregnant livestock is essential in preventing this type of degenerative muscle syndrome in newborns, including foals.

A new method of bacteriological testing for livestock water analysis:

S Byrne

Good quality drinking water is critical for optimal livestock health and production. However, water may also be the vehicle for transmission of many diseases. Young animals are particularly susceptible to intestinal infections caused by *Salmonella*, *E coli* and *Cryptosporidium spp,* which are often found in feces-contaminated water. Therefore preventive steps should be taken to assure the safety of drinking water, such as restricting the access of animals to water sources such as dugouts, ensuring animals cannot defecate into drinking troughs, and avoiding entry of run-off surface water into wells.

Because low-level fecal contamination may not be visibly obvious, a key step in prevention is to perform bacteriological testing of water samples. Such testing uses the numbers of certain intestinal bacteria to indicate the degree of contamination. These indicators are the total coliform count, the fecal coliform count, and the *E.coli* count. **Total coliform bacteria** are widespread in nature; they are found in the intestine of animals, as well as in manure and soil. Their presence in water is a rather general indicator of contamination of the water supply by an outside source.

Fecal coliforms are a more limited group of bacteria. Most of these grow in the intestines of animals, and are used to indicate direct fecal contamination of water. However some fecal coliforms, such as *Klebsiella spp*, can survive in the environment. For this reason, **E.coli counts** are replacing fecal coliform counts as better indicators of recent fecal contamination.

It is important to remember that both total coliform counts and *E.coli* counts allow a determination of the potential risk for water borne disease. A high total coliform count

in water indicates contamination of the water from feces or soil runoff. On the other hand, because *E.coli* bacteria don't survive long in the environment, the *E.coli* count is a more stringent measure, and their presence indicates direct fecal contamination.

We are now performing bacteriological water testing using the 'IDEXX Colisure' method. This EPA-approved method is a first-line test that enables the determination of both the total coliform count and the *E coli* count in 100 ml of water.

No clear standards exist for coliform counts in drinking water provided to animals. However, for human consumption, both the total coliform and *E.coli* counts should be zero. These same high standards are also required in the wash water used in dairy operations to prevent contamination of food products originating from dairy milk. For livestock, the recommended **maximum** levels of fecal coliforms *(E coli*) in drinking water ranges from 10 organisms per 100 ml for calves, to 5,000 organisms per 100 ml for cows.

If you request perform bacteriological water testing, please submit at least 100 ml of water in a sterile container (available through the Animal Health Centre laboratory). If the water is chlorinated it is important to use bottles from this laboratory, as these contain a chemical to inactivate the residual chlorine in the water. Take care not to contaminate the water when sampling. To collect a sample, remove the aerator (if present) and run the water for two minutes to clear the line. Do not touch the inside of the bottle or lid. Fill the bottle to above the line and replace the lid. Label the bottle with the farm name and the water source. Transport the sample as soon as possible to the AHC on ice (but not frozen).

We are using this method to ensure good quality drinking and wash water for farm animals. Please be reminded that we are not in the business of certifying water for human consumption. If you have concerns about your own drinking water quality, please contact a private laboratory which performs water testing.

Did you know?

(a) That selenium is a component of thyroid gland hormones? Selenium is a component of type-1 iodothyronine deiodinase, which catalyzes the extra-thyroidal conversion of thyroxine (T4) to the more active form tri-iodothyronine. Sheep grazing pastures low in selenium frequently have higher circulating T4 and lower circulating T3 concentrations than sheep receiving selenium supplementations.

- from *Veterinary Medicine,* 9th edition, 2000; Radostits OM, Gay CC, Blood DC, Hinchcliff KW. WB Saunders, publishers.

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(b) That *Aeromonas hydrophila* has been reported as an occasional causative agent of bovine mastitis? Kirkbride also lists *Aeromonas hydrophila* as an agent occasionally linked to bovine abortion (*Laboratory Diagnosis of Livestock Abortion*, 3rd ed, 1990).

A hydrophila is a Gram-negative bacillus, and like *Vibrio* species, is a motile, facultative anaerobe. In this laboratory, the organism sometimes appears in bacterial cultures of stomach contents from aborted bovine fetuses, usually accompanied by *E coli*. Its significance in these cases is often uncertain, especially if no placenta has been submitted for examination.

In other species, *A hydrophila* is considered an opportunistic pathogen as it is directly linked to water-borne disease in fish and reptiles. These include swim-bladder

infection in pike, 'Red-leg disease' in frogs, and necrotic stomatitis/septicemia in snakes. The organism has also been reported to cause neonatal septicemia in canine puppies. Most strains of *A hydrophila* are considered harmless for humans, but some have been associated with intestinal disease and diarrhea.

– from *Clinical Veterinary Microbiology* 1999; Quinn PJ, Carter ME et al; Mosby publishers.

Scrapie surveillance in Alberta cull ewes:

"… The internationally recognized TSE-specific immunohistochemistry staining technique was used to test a specific target site of the brain stem (obex), tonsil and lymphoid tissue of the third eyelid for the scrapie prion protein. This is a very sensitive and specific test that allows detection of subclinically affected sheep.

All 350 cull ewes were negative for scrapie by immunohistochemistry staining. The prevalence of scrapie in Alberta's cull ewe population, estimated to be 22,000, is estimated to be between 0 and 0.83%, with a 95% confidence level..."

– Dr Brian Miller, Veterinary Pathologist, Agri-Food Systems Br., Food Safety Division, Edmonton, AB; from the March 2003 issue of *Animal Health Forum*.

Hemorrhagic bowel syndrome in cattle:

"…Hemorrhagic bowel syndrome (HBS) is a sporadic, acute intestinal disease of milking cows. HBS is also known as 'bloody gut' and 'jejunal hemorrhage syndrome', and is characterized by large blood clots in the intestine that result in obstruction and severe enlargement of the bowel.

Several reports suggested that *Clostridium perfringens* Type A might be involved in the etiology of HBS. In fact, *Clostridium perfringens* Type A has been found in 85% of HBS cases. However, because this type of Clostridium is found commonly in both the environment and in bovine gastrointestinal tracts, it has not been determined if the organism is involved in the primary disease process or proliferates as a secondary response. Other proposed HBS risk factors are: increasing milk production; feeding a high-energy total mixed ration (compared to a component diet); and a decrease in dietary fibre."

– *APHIS Info Sheet*, of the United States Department of Agriculture, Animal and Plant Inspection Service; May 2003.

Nursing sickness in mink:

" … Nursing sickness, the largest cause of mortality in adult female mink (*Mustela vison*), is an example of a metabolic disorder, which develops when the demands for lactation require extensive mobilization of body energy reserves. The condition is characterized by progressive weight loss, emaciation, and dehydration with high concentrations of glucose and insulin in the blood.

It is recommended that female breeder mink be maintained in moderate body condition to avoid obesity and emaciation and the potential development of acquired insulin resistance…..In order to alleviate the nursing burden on females, large litters

may be fostered... Early weaning may also be helpful in preventing the onset of the catabolic state... Lowering of the dietary protein and elevating the level of carbohydrates may alleviate both oxidative stress and heat stress on the nursing females. This dietary change also reduces the amount of water voided in the urine due to reduced protein catabolism aiding in the prevention and management of this complex metabolic syndrome.

In summary, it is postulated that the underlying cause of mink nursing sickness is the development of acquired insulin resistance. There are 3 possible key elements leading to this development in the female mink:1) obesity or lipodystrophy, 2) n-fatty acid deficiency, and 3) high protein diet. Given the widespread incidence of type 2 diabetes in carnivore companion animals it may be surmised that other, thus far unexplained, metabolic disorders seen in both male and female mink during other times of the production cycle may have their origins in the acquired resistance syndrome... "

– K Rouvinen-Watt: Nursing sickness in the mink – a metabolic mystery or a familiar foe? *Can Jour Vet Res* 2003;67:161-168.

Looking back - post mortem changes in normal bovine rumen content and in grain overloads:

"…One of the difficulties encountered by the veterinarian in the field and in the diagnostic laboratory is the evaluation of ruminal content in an animal that has been dead several hours. Changes in pH with time have been described in the live animal in the natural disease but reference to changes after death was not found in the literature. In order to assist in the evaluation of changes in ruminal content in animals that have been dead for some time the following investigation was carried out……

Rumens from apparently normal feedlot cattle were collected at slaughter over a period of several weeks in the summer and brought within one hour of death to the laboratory…

The results indicate that the pH values of ruminal content remain above 6.0 up to 24 hours after death. Depending on the history of the case, a level of 5 or lower should be present to indicate fatal acidosis in untreated cases. Evaluation by pH paper is sufficiently accurate to make a diagnosis in most instances. The pH values reported here may not represent exactly the condition in the rumens of dead and unopened carcasses. In addition, the pH values may change quickly when the content of the rumen is exposed to air. The protozoan activity falls to zero in 24 hours and might be less accurate but still satisfactory as a diagnostic aid. In most naturally fatal cases of the disease in cattle the protozoan activity is nil at time of death…"

The above findings will be of assistance in evaluating changes in rumen content in animals in which overload is a suspected or possible diagnosis..."

– Thomson, RG. Post mortem changes in rumen content. *Can Vet Jour* 1969;10:312-313.

Mosquito life cycle:

"…Mosquitoes have a four-stage life cycle: egg, larva, pupa and egg. Only female mosquitoes need a blood meal, which they require to develop their eggs. Both male

and females feed on nectar for their energy source. During its life, a female mosquito may take two or three blood meals and develop several hundred eggs each time. Mosquitoes can live four to eight weeks. All female mosquitoes lay eggs in and around water. Some species leave their eggs in spots that will flood later, such as mud at the edge of a drying pond, while others lay them in tree holes that flood in rains. The eggs hatch into larvae. At the water surface, the larva changes to a pupa before emerging as an adult mosquito. The entire life cycle can be completed in less than 10 days if the temperature is favourable. Most mosquito species survive the winter as dormant fertilized eggs. However the mosquito species of concern with respect to the spread of West Nile virus is *Culex pipiens*. *Culex* species are thought to be the primary bridging factor in the transfer of West Nile virus from infected birds to humans and horses. A fertilized *Culex* female can survive over winter in sheltered places such as animal burrows, cellars and sewers, emerging in the spring to take a blood meal prior to laying her eggs..."

– Bob Wright DVM, Ontario Ministry of Agriculture and Food; from a news bulletin on mosquitoes and West Nile virus, May 2003.

Recycling good information:

A brief review of the topics covered in this newsletter will reveal diverse sources of information as well as material from our own laboratory. Many of the persons reading *Diagnostic Diary* including veterinarians don't have access to the numerous and varied sources of information that arrive daily at the Animal Health Centre.

Redistribution of a few of these articles via our own newsletter (*Diagnostic Diary*) disseminates this information even further. A few represent different areas of scientific investigation, such as Gordon Reid's fascinating booklet entitled *Head-Smashed–In. Ed.*

Focus on Staff: Robert Davis, Toxicology Section.

Robert Davis has joined the staff of the Animal Health Centre as Senior Analyst in the Toxicology and Clinical Nutrition Lab. Mr Davis replaces the former senior analyst Bob Puls, who recently retired. Previously, Mr Davis was employed as an analyst at the Provincial Toxicology Centre located at Riverview Hospital, in Port Coquitlam. Prior to that, he worked in a mineral assay laboratory and briefly for the Ministry of Environment.

A native of Penticton, British Columbia, Robert has a Bachelor of Science degree in chemistry from the UBC. He has extensive chromatography experience performing analysis on specimens from humans and looks forward to expanding his knowledge to include the many species tested here.

The clinical nutrition aspect of the job is new to him, but there are many similarities to his previous work. In time, he looks forward to the possibility of expanding and/or improving the testing presently offered by the Toxicology section.

A resident of Abbotsford, Robert is married and has a ten month-old son. We sincerely welcome Robert Davis to the AHC, and wish him well in his new role as senior analyst in the Toxicology Section.

Head-Smashed-In:

"…. Head-Smashed-In Buffalo Jump in Alberta is one of the oldest, largest, and best preserved buffalo jump sites in North America and was declared a World Heritage Site in 1981…

One might assume that the name "Head-Smashed-In" refers to the unfortunate buffalo, but according to George Dawson, who worked for the Geological Survey of Canada in the 1880s, the name comes from an allegedly true story concerning a Peigan brave who wanted a closer look at the plummeting buffalo. The brave situated himself against the cliff wall, beneath the lip over which the bison charged. He watched from his privileged position as one after another the bison hurtled to their deaths. He was safe for a while, but the pile of bison carcasses grew much higher than he anticipated, eventually pinning him against the cliff wall. When his fellow tribesmen came to butcher the kill, they were shocked to discover the young brave with his head crushed by the weight of dead and dying buffalo. In the Blackfoot language, this place is called *Estpah-Sikikini-Kots*, meaning "where he got his head smashed in."

One bison supplied enough meat to feed a tribe of approximately three hundred people for one day. Imagine how much food and leather a kill of two to three hundred buffalo would have supplied…

Leg bones were smashed to bits and thrown into the cooking pot to draw out grease. It was a laborious task, producing only a cup of grease per cooking pot. For days fires and boiling pots were used throughout the processing area….

It was once estimated that the Indians had over three hundred separate uses for a buffalo. Hides were made into clothing or used to cover teepees and lodges. It took over … sixteen to twenty skins for a large teepee…

The list of items a buffalo carcass provided is long and varied, but included glue, whistles, shields, bowstrings, ropes, snowshoes, saddles, blankets, and robes. Buffalo robes protected the natives from cold prairie winds… Buffalo robes and horses were the natives' two most important bartering items.

Trained buffalo horses were among the fastest, most intelligent animals on the plains. They readily learned to charge the much-larger bison and to swerve as arrows flew from hunters' bowstrings. Blackfoot braves rode bareback, controlling these "buffalo runners" by exerting pressure with their knees. Manoeuvring a buffalo horse required extraordinary skill, as wounded bulls were extremely dangerous to approach while gopher holes that randomly appeared in the prairie dirt threatened to throw both man and mount... Stories were told of daring braves who jumped on the backs of wild, running bison, speared select animals, and miraculously clambered back onto their mounts….."

– from the booklet *Head-Smashed-In Buffalo Jump*, by Gordon Reid; published 2002 by Fifth House Ltd, Calgary, Alberta

The Last Word:

"It must have been at an early age that I decided I would be a scientist. But I foresaw one snag. By the time I grew up… everything would have been discovered."

Francis Crick, co-discoverer of the helix structure of DNA and Nobel laureate. Quoted from *The Quotable Scientist*, published 2000 by McGraw Hill, USA, but originally taken from his book *What Mad Pursuit*, a personal view of scientific discovery, published 1988.

Bacterial resistance to antibiotics – a compilation by species

S Byrne

Antibiotic resistance of Gram negative bacteria isolated from bovine sources (Nov.1, 2002 - May 1, 2003)

	Tilmicosin	Enrofloxacin	Excenel	Gentamicin	Neomycin	Penicillin	Sulbactam-Amp	SXT*	Tetracycline	Florfenicol
Haemophilus somnus										
# of isolates	1	1	1	1	1	na	1	1	1	1
# resistant	0	0	0	1	1	na	0	0	0	0
% resistance	0%	0%	0%	100%	100%	na	0%	0%	0%	0%
Hemolytic *Escherichia coli*										
# of isolates	15	27	29	29	29	1	27	29	29	27
# resistant	15	0	0	1	1	1	1	1	3	0
% resistance	100%	0%	0%	3%	3%	100%	4%	3%	10%	0%
Non-hemolytic *E.coli*										
# of isolates	68	169	169	169	169	na	169	171	171	170
# resistant	66	9	9	25	69	na	67	69	110	44
% resistance	97%	5%	5%	15%	41%	na	40%	40%	64%	26%
Mannheimia haemolytica										
# of isolates	7	15	15	15	15	12	15	15	15	15
# resistant	2	1	1	2	5	3	4	2	3	0
% resistance	29%	7%	7%	13%	33%	25%	27%	13%	20%	0%
Pasteurella multocida										
# of isolates	4	6	6	6	6	5	5	5	6	6
# resistant	1	0	0	1	2	0	1	1	0	0
% resistance	25%	0%	0%	17%	33%	0%	20%	20%	0%	0%
Salmonella sp.**										
# of isolates	1	5	5	5	5	na	5	5	5	5
# resistant	1	0	2	0	3	na	4	2	4	3
% resistance	100%	0%	40%	0%	60%	na	80%	40%	80%	60%

* Sulfamethoxazole/trimethoprim
** includes S.dublin, S.typhimurium

Antibiotic resistance of Gram positive bacteria isolated from bovine sources (Nov. 1, 2002- May 1, 2003)

	Tilmicosin	Enrofloxacin	Erythromycin	Gentamicin	Lincomycin	Penicillin	SXT*	Tetracycline	Florfenicol
Arcanobacterium pyogenes									
# of isolates	2	9	9	9	9	9	9	9	9
# resistant	0	3	1	2	0	0	7	5	0
% resistance	0%	33%	11%	22%	0%	0%	78%	56%	0%
Listeria sp.**									
# of isolates	na	2	2	2	2	2	2	2	2
# resistant	na	2	2	0	2	2	2	2	2
% resistance	na	100%	100%	0%	100%	100%	100%	100%	100%
Staphylococcus aureus									
# of isolates	1	2	2	2	2	1	2	2	2
# resistant	1	0	2	0	2	1	0	2	0
% resistance	100%	0%	100%	0%	100%	100%	0%	100%	0%

* Sulfamethoxazole/trimethoprim
** includes L.innocua, L.monocytogenes

Antibiotic resistance of Gram negative bacteria isolated from bovine milk samples (Nov.1, 2002 - May 1, 2003)

	Cephalothin	Cloxacillin	Excenel	Neomycin	Penicillin	SXT*	Tetracycline	Pirlimicin	Pen-Novobiocin
Escherichia coli									
# of isolates	6	13	13	13	13	13	13	13	13
# of resistant	5	12	1	2	12	1	4	13	12
% resistance	83%	92%	8%	15%	92%	8%	31%	100%	92%
Pasteurella multocida									
# of isolates	1	3	3	3	3	3	3	3	3
# of resistant	0	2	0	2	0	0	0	3	0
% resistance	0%	67%	0%	67%	0%	0%	0%	100%	0%
Serratia sp.*									
# of isolates	na	3	3	3	3	3	3	3	3
# of resistant	na	3	0	1	3	1	2	3	3
% resistance	na	100%	0%	33%	100%	33%	67%	100%	100%

* Sulfamethoxazole/trimethoprim
** includes *S. marscesens* and *S. liquefaciens*

Antibiotic resistance of Gram positive bacteria isolated from bovine milk samples (Nov.1, 2002 - May 1, 2003)

	Cephalothin	Cloxacillin	Excenel	Neomycin	Penicillin	SXT*	Tetracycline	Pirlimicin	Pen-Novabiocin
Aerococcus viridans									
# of isolates	4	8	8	8	8	8	8	7	7
# resistant	2	6	2	8	6	8	7	5	3
% resistance	50%	75%	25%	100%	75%	100%	88%	71%	43%
Arcanobacterium pyogenes									
# of isolates	3	6	6	6	6	6	6	5	5
# resistant	0	2	1	3	1	5	2	0	0
% resistance	0%	33%	17%	50%	17%	83%	33%	0%	0%
Enterococcus sp.*									
# of isolates	2	3	3	3	3	3	3	3	2
# resistant	2	3	3	3	3	3	3	3	2
% resistance	100%	100%	100%	100%	100%	100%	100%	100%	100%
Streptococcus sp.**									
# of isolates	10	12	12	11	12	12	12	12	11
# resistant	2	3	2	11	3	12	9	8	2
% resistance	20%	25%	17%	100%	25%	100%	75%	67%	18%
Staphylococcus aureus									
# of isolates	5	11	11	11	11	11	11	11	11
# resistant	0	0	0	0	5	0	0	1	1
% resistance	0%	0%	0%	0%	45%	0%	0%	9%	9%

* Sulfamethoxazole/trimethoprim
** includes E.faecalis, E.faecium, E.saccharolyticus
*** includes S.dysgalactiae, S.mutans, S.uberis

Antibiotic resistance of Gram negative bacteria isolated from porcine sources (Nov 1, 2002 - May1, 2003)

	Apramycin	Excenel	Gentamicin	Lincomycin	Neomycin	Penicillin	SXT*	Tetracycline	Tri-sulfa
Actinobacillus suis									
# of isolates	8	8	8	8	7	8	8	8	8
# resistant	2	0	1	8	1	0	0	6	3
% resistance	25%	0%	13%	100%	14%	0%	0%	75%	38%
Hemolytic *Escherichia coli*									
# of isolates	11	11	11	11	11	11	11	11	11
# resistant	1	1	2	11	3	11	2	11	11
% resistance	9%	9%	18%	100%	27%	100%	18%	100%	100%
Non-hemolytic *E.coli*									
# of isolates	57	57	57	56	56	57	55	57	56
# resistant	14	3	10	55	17	57	17	53	51
% resistance	25%	5%	18%	98%	30%	100%	31%	93%	91%
Pasteurella multocida									
# of isolates	11	11	11	10	11	11	11	11	11
# resistant	11	3	6	10	4	4	5	2	11
% resistance	100%	27%	55%	100%	36%	36%	45%	18%	100%
Salmonella sp.*									
# of isolates	3	3	3	3	3	3	3	3	3
# resistant	0	1	0	3	1	3	1	2	2
%resistance	0%	33%	0%	100%	33%	100%	33%	67%	67%

* Sulfamethoxazole/trimethoprim
** includes *S. derby* , *S. enterica* and *S. typhimurium*

Antibiotic resistance to Gram positive bacteria isolated from porcine sources (November 2002 - May 2003)

	Apramycin	Excenel	Gentamicin	Lincomycin	Neomycin	Penicillin	SXT*	Tetracycline	Tri-sulfa
Staphylococcus aureus									
# of isolates	2	2	2	2	2	2	2	2	2
# resistant	0	0	0	2	0	2	0	1	2
% resistance	0%	0%	0%	100%	0%	100%	0%	50%	100%
Staphylococcus hyicus									
# of isolates	2	2	2	2	2	2	2	2	2
# resistant	0	0	0	2	0	1	0	2	2
%resistance	0%	0%	0%	100%	0%	50%	0%	100%	100%
Streptococcus suis II									
# of isolates	19	19	19	18	17	19	19	19	19
# resistant	19	6	19	18	17	11	17	19	18
% resistance	100%	32%	100%	100%	100%	58%	89%	100%	95%
Streptococcus sp.*									
# of isolates	9	9	9	9	9	9	9	9	9
# resistant	9	0	9	9	9	1	9	9	9
% resistance	100%	0%	100%	100%	100%	11%	100%	100%	100%

* Sulfamethoxazole/trimethoprim
** includes *S.bovis* , *S.dysgalactiae-equisimilus* , *S.equinis* , *S.equisimilus* , *S.infantarius* , *S.porcinus* , *S.suis I* , *S.suis I/II*

Antibiotic resistance of Gram negative bacteria isolated from poultry* (Nov.1, 2002 - May 1, 2003)

	Apramycin	Enrofloxacin	Neomycin	Penicillin	Tetracycline	Tri-sulfa	Romet
Actinobacillus salpingitidis							
# of isolates	5	5	5	1	5	5	5
# resistant	0	0	1	1	3	5	2
%resistance	0%	0%	20%	100%	60%	100%	40%
Mannheimia haemolytica							
# of isolates	18	18	18	8	18	18	18
# resistant	0	1	2	2	14	12	6
%resistance	0%	6%	11%	25%	78%	67%	33%
Non-hemolytic *Escherichia coli*							
# of isolates	234	237	232	na	238	233	238
# resistant	10	4	44	na	173	198	69
%resistance	4%	2%	19%	na	73%	85%	29%
Pseudomonas aeruginosa							
# of isolates	13	13	12	na	13	13	13
# resistant	9	2	9	na	13	7	13
%resistance	69%	15%	75%	na	100%	54%	100%
Ornithobacterium rhinotracheae							
# of isolates	8	8	8	1	8	8	8
# resistant	7	0	6	1	5	6	8
%resistance	88%	0%	75%	100%	63%	75%	100%
Salmonella sp. **							
# of isolates	7	7	6	na	7	7	7
# resistant	0	0	2	na	6	7	1
%resistance	0%	0%	33%	na	86%	100%	14%

* 250 chicken isolates, 46 turkey isolates
** includes Group B: *S.heidelberg* , *S.typhimurium* ; Group C1: *S.thompson* ; Group E1: *S.uganda*

Antibiotic resistance to Gram positive bacteria isolated from poultry* (Nov.1, 2002 - May 1, 2003)

	Enrofloxacin	Erythromycin	Lincomycin	Penicillin	Tetracycline	Romet
Staphylococcus aureus						
# of isolates	49	47	48	48	48	46
# resistant	9	17	43	13	38	1
%resistance	18%	36%	90%	27%	79%	2%

* 48 chicken isolates, 2 turkey isolates