Veterinary Medicine

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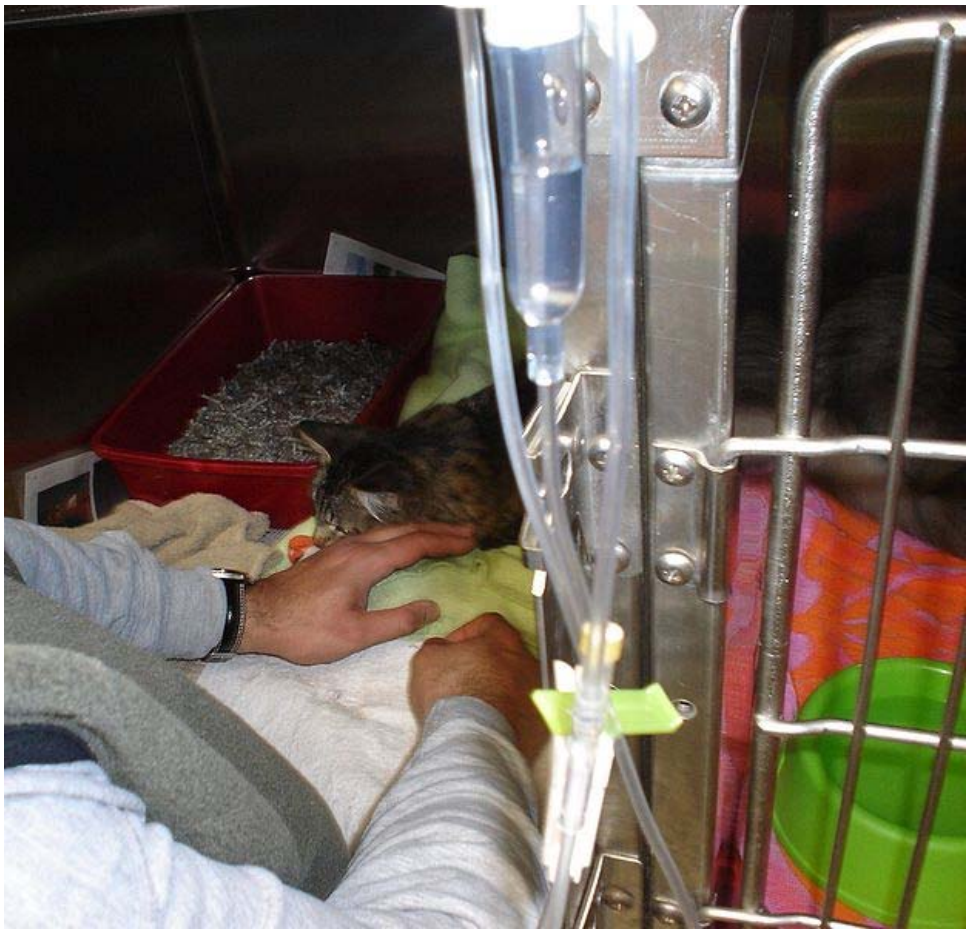
Chapter 1

Veterinary Medicine



A cat after surgery. Antibiotics and morphine are delivered via various intravenous drips.

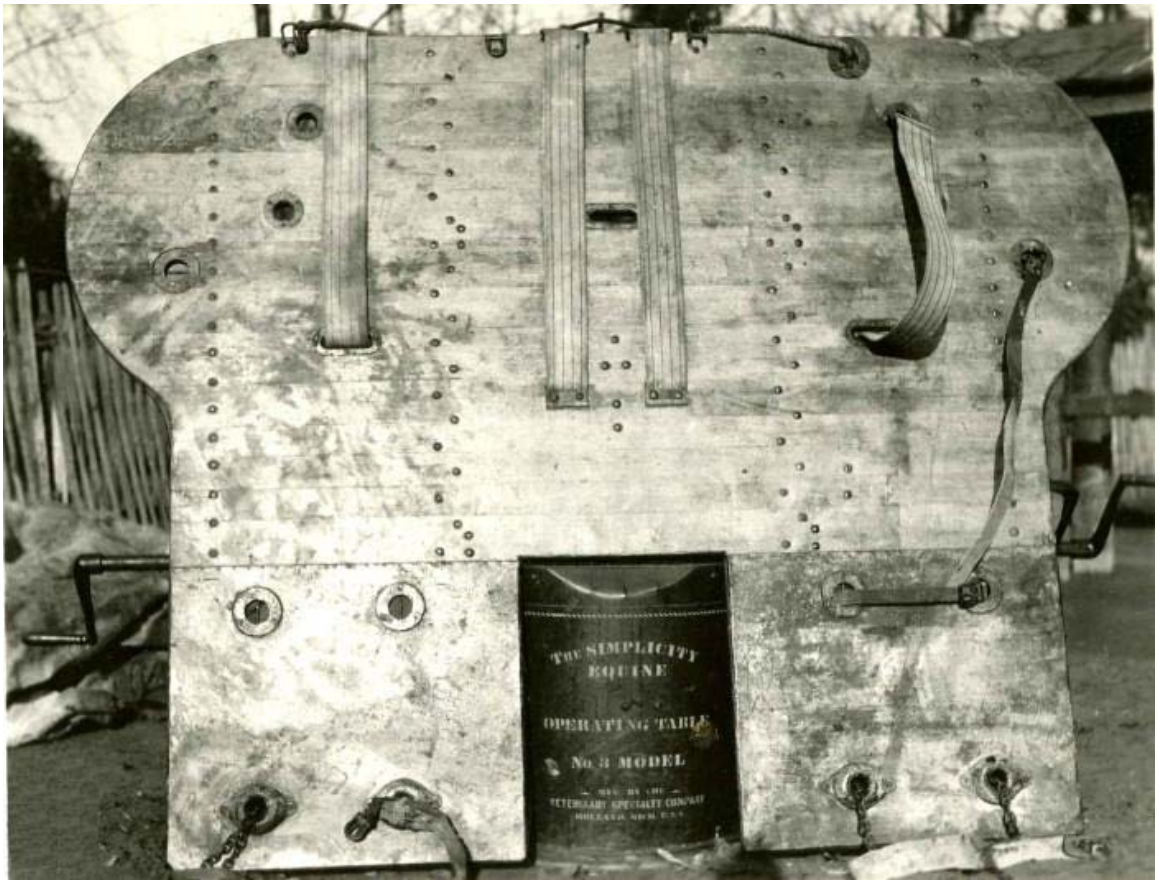
Veterinary medicine is the branch of science that deals with the application of medical, surgical, public health, dental, diagnostic, and therapeutic principles to non-human

animals, including wildlife and domesticated animals, including livestock, working animals, and companion animals. Practitioners of veterinary medicine are known as veterinarians. In most developed countries, veterinarians are highly qualified professionals with advanced educations.

Veterinary science helps human health through the monitoring and control of zoonotic disease (infectious disease transmitted from non-human animals to humans) and veterinary scientists often collaborate with epidemiologists.

History

A pillar in Vaishali, India, displaying edicts of Emperor Asoka (272—231 BCE); the pillar records King Asoka building hospitals for both humans and animals.



"The Simplicity Equine," a portable operating table for horses used by the field veterinarians of the US Army Signal Corps in World War I

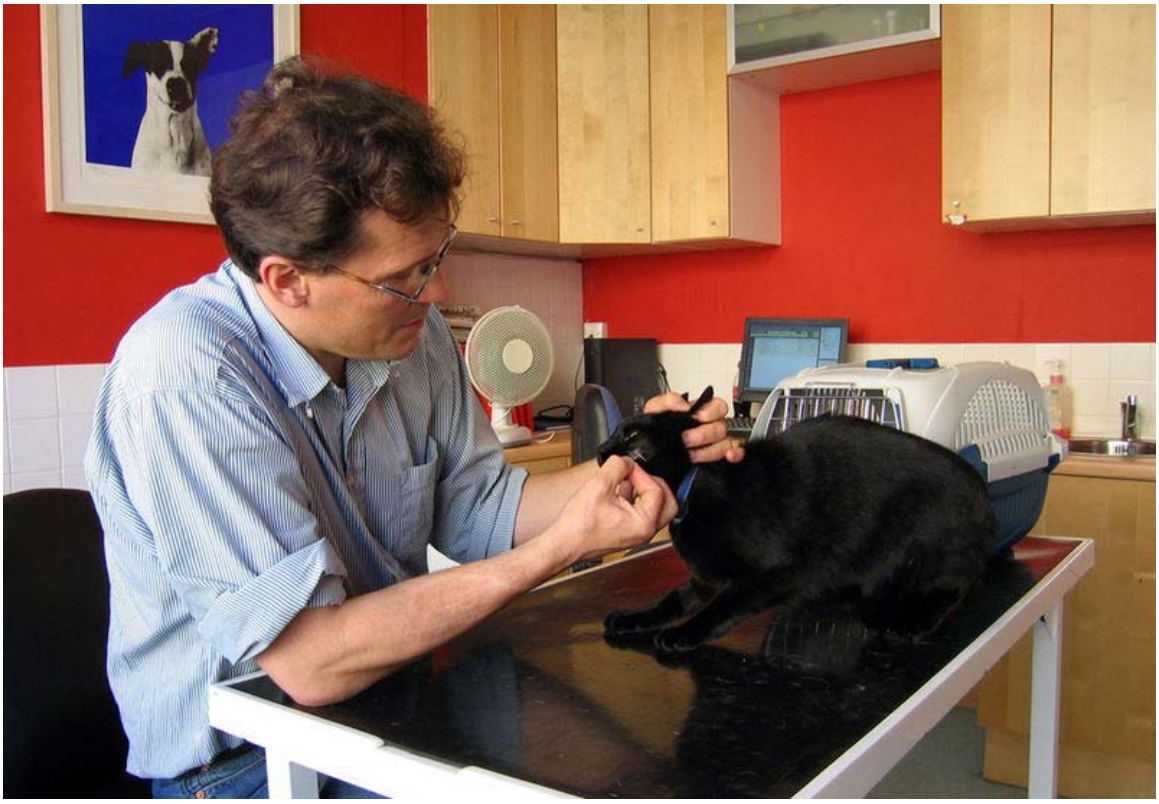
An injured horse being secured to the vertically oriented table

With the table rotated to its horizontal orientation and supported by a drum on one side and folding cot-like legs on the other, a veterinarian operates on a horse.

The Egyptian *Papyrus of Kahun* (1900 BCE) and Vedic literature in ancient India offer one of the first written records of veterinary medicine. One of the edicts of Ashoka reads: "Everywhere King Piyadasi (Asoka) erected two kinds of hospitals, hospitals for people and hospitals for animals. Where there were no healing herbs for people and animals, he ordered that they be bought and planted." The Talmud does state that no mares were exported from Egypt in Roman times without being subjected to a hysterectomy, which tend to prove that successful surgery was implemented in such an early period.

Modern veterinary medicine

Some animal hospitals have segregated waiting rooms for dogs and cats

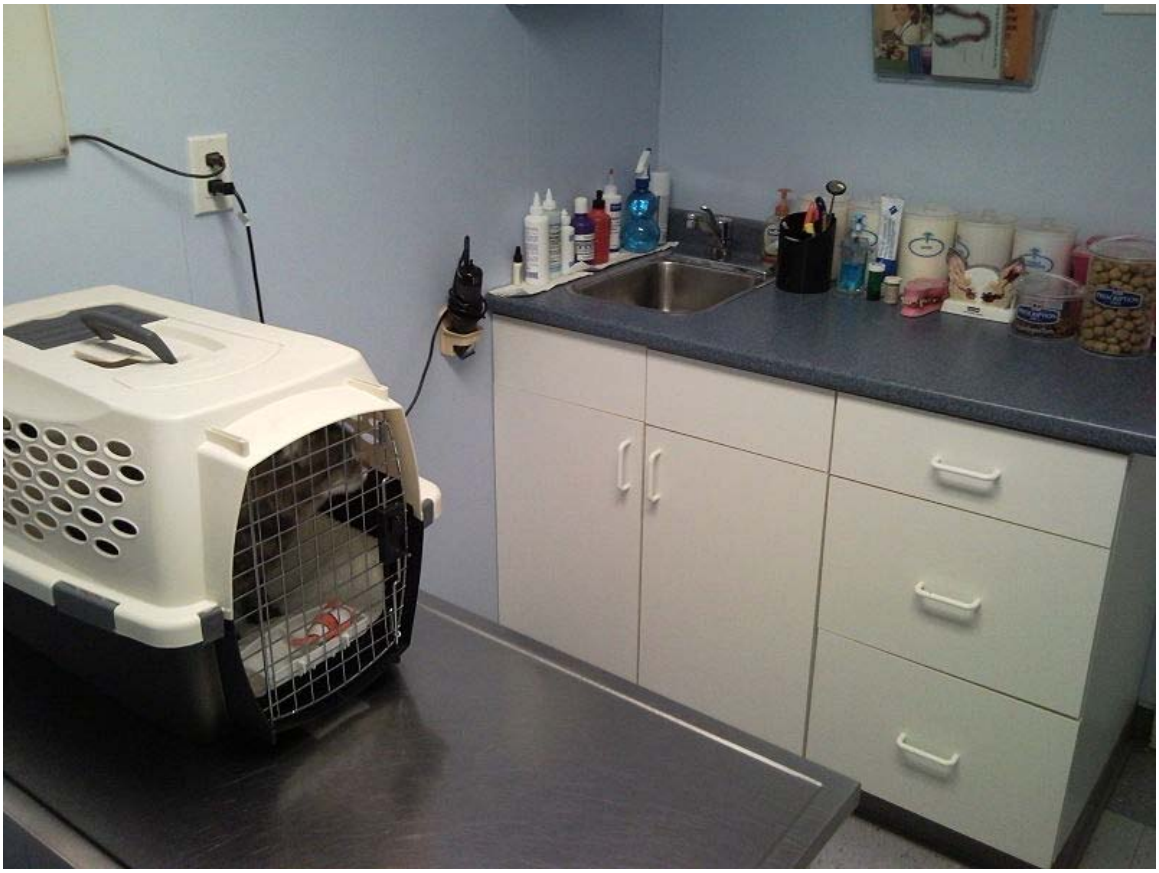


A veterinary surgeon in Cambridge, UK at work with a black cat

Patient, with clouded eyes, on day five of hospital stay as she recovers from second surgery

Same patient after one week of hospital stay and third surgery, eyes have cleared up.

Same patient, same day, in play, yellow bandage protects intravenous insertion sites



Patient awaiting discharge after completing 11 days in the hospital

Patient back home where she is protected against her (and others') better instincts in a cage until the sutures from her multiple surgeries are removed, the cage is provided with kitty litter, food, and water

Modern veterinary medicine is aided by the availability of advanced diagnostic and therapeutic techniques for many species. Today animals may receive advanced medical, dental, and surgical care, including insulin injections, root canals, hip replacements, cataract extractions, and pacemakers.

Veterinary specialization has become more common in recent years. Currently, 20 veterinary specialties are recognized by the American Veterinary Medical Association (AVMA), including anesthesiology, behavior, dermatology, emergency and critical care, internal medicine, cardiology, oncology, ophthalmology, neurology, radiology and surgery. To become a specialist, a veterinarian must complete additional training after

graduation from veterinary school in the form of an internship and residency, and then pass a rigorous examination.

Veterinarians assist in ensuring the quality, quantity, and security of food supplies by working to maintain the health of livestock and inspecting the meat itself. Veterinary scientists occupy important positions in biological, chemical, agricultural and pharmaceutical research.

In many countries, equine veterinary medicine is also a specialized field. Clinical work with horses involves mainly locomotor and orthopedic problems, digestive tract disorders (including equine colic, which is a major cause of death among domesticated horses), and respiratory tract infections and disease.

Zoologic medicine, which encompasses the healthcare of zoo and wild animal populations, is another veterinary specialty that has grown in importance and sophistication in recent years as wildlife conservation has become more urgent.

Today's veterinarian

According to consumer surveys, the veterinarian ranks across the United States as one of the most respected career paths. Veterinarians are encouraged to take an oath in which they swear to use their knowledge and skills for the overall benefit of society through protecting the health needs of every species of animal and also environmental protection, food safety, and public health. Many of today's veterinarians are dedicated to working long difficult hours to live out this oath in their respective practices.

There are many personal attributes that contribute to a successful career in veterinary medicine, the most important being a scientific mind, good communication skills, and management experience. Having a scientific mind consists of having an inquiring mind and a keen sense of observation. A career in veterinary medicine means a lifelong pursuit in scientific learning, so an interest in the biological sciences is a must and a genuine love and understanding of animals is crucial. Good communication skills are vital because veterinarians should be able to meet, talk, and work well with a variety of personalities and characters. Compassion is essential for success in the career field because they will be working directly with their animal client's human owners, who most likely have strong bonds with their pets. Many of the fields within the career require the veterinarians to manage other employees and businesses as a whole. These positions are made more rewarding and simpler if one has a background in basic management or leadership positions .

A study was performed in attempts to discover professional identity and professionals' workplace learning based on a theoretical proposal. Veterinarians were found to approach workplace learning differently according to two key variables: perceived alignment with professional identity and perceived importance to professional practice. Differences were evident when comparing how veterinarians approached learning about the medical aspects of their profession in contrast to practice management that consisted of

nonmedical disciplines that are a definite part of veterinary practice. It was common for these veterinarians to associate their professional identity with scientific, medical, clinical disciplines, but less common for these veterinarians to include the nonmedical disciplines.

This study by Hoskin and Anderson-Gough (2004) helped to explain the effects of disciplinary action on workplace learning. They found that educational systems that produce members of established disciplines tend to be highly specialized. This then resulted in significant influence on the type of content that is transmitted in the process of becoming qualified to practice a professional discipline. Furthermore, Lewis and Klausner (2003) found that veterinary schools in the United States recognize that it is their role to be gatekeepers of the profession. They are beginning to understand the full responsibility for selecting candidates who have the skills to capitalize on their education and build a successful career. It is their responsibility because it is their institution that has a significant amount of influence in the type or personality of the individual that will then graduate with a degree to practice animal medicine. This personality is then directly correlated to whether or not the graduate succeeds in their profession or does not succeed.

Overview

An animal hospital in North Smithfield, Rhode Island

As in the human health field, veterinary medicine (in practice) requires a diverse group of professionals to meet the needs of patients. In the year 2006, veterinarians held about 62,000 jobs.

According to the American Veterinary Medical Association, about three-quarters of veterinarians were employed in either an individual or group practice. The remainder were employees in other settings, including colleges of veterinary medicine, medical schools, research laboratories, animal food companies, and pharmaceutical companies. The Bureau of Labor Statistics reports that around 1,400 civilian veterinarians are employed by the United States federal government, mainly in the Department of Agriculture, Department of Health and Human Services, and Department of Homeland Security. State and local governments also employ veterinarians.

Employment is expected to increase more than average and much faster in comparison to other career options, ensuring job opportunities in the field of veterinary medicine. It has been stated that this expected increase is near 35% over the next decade; it is a direct result of the increase of certain pet populations, such as cats, and the increased amount of pet owners willing to purchase pet insurance, which then increases the amount of treatment that the owner is willing to fund. Additionally, modern veterinary medicine has caught up to human medicine in many areas such as cancer treatment, preventative dental care, hip replacements, transplants, and blood transfusions. These medical advances have encouraged pet owners to take advantage of these new medical possibilities, likewise increasing the need for veterinary care because of the increased demand. One other area of increased demand for veterinarians in seen in the continued support for public health and food and animal safety, CDC national disease control programs, and biomedical research on human health problems.

These job opportunities can be expected because there are only 28 accredited Veterinary medicine schools in the United States and five in Canada, creating stiff competition for admittance into veterinary school. This small number of schools results in a limited number of graduates each year, averaging around 2,700 in the US each academic year.

There continues to be a steady trend in the different fields of veterinary medicine, which doctors go into these respective fields, and what hours they usually take on to work. New graduates continue to be attracted to companion-animal or small animal practice because they prefer to work with pets and live/work in metropolitan areas. Therefore, employment opportunities are good in cities and suburbs, but tend to be better in rural areas because fewer veterinarians compete for work in those areas. Beginning veterinarians may take positions requiring evening or weekends to accommodate the extended hours of practice that many places offer. Then there are some veterinarians that take salaried positions in retail stores offering veterinary services whereas others that are self-employed have to work long and hard to establish a good client base. The number for large animal veterinarians is much less than that of companion or small animal veterinarians. This is directly correlated to the simple fact that most people do not want to live/work in rural or isolated areas. Nevertheless job prospects are great in the large animal practice because of the previously stated tendencies. Finally, veterinarians with training or qualifications in

food safety and security, animal health and welfare, and public health and epidemiology should have the best opportunities for a career within one of the departments of the Federal Government .

Threatening veterinary shortage

A shortage of veterinarians who treat farm animals is stressing the nation's food inspection system. This shortage is becoming so severe that it is prompting the Federal Government to offer bonuses and cover moving expenses to fill hundreds of empty employment opportunities. The shortage is mainly due to veterinarians choosing to live in metropolitan areas and pursue a practice specializing in pets or small animals. The main scarcity is seen in veterinarians who treat farm animals or work as government inspectors. The shortage is most severe in the USA's Farm Belt, which is in the rural areas of the Midwest that is responsible for much of the nations meat production.

The American Veterinary Medical Association reported there are roughly 500 counties that have large populations of food animals, but no veterinarian to treat these animals. The common concern of a lesser salary in the farm animal field was disproven by the statistics showing that starting salaries for private practice veterinarians are generally higher than that of public practice veterinarians, but after about 10 years of practice they roughly even out. The Bureau of Labor Statistics also reports that the number of veterinarians needed will just continue to increase to 22,000 by the year 2016. This would make it one of the fastest growing professions. The nation's 28 veterinary schools provide around 2,700 graduates a year, something that has not changed in three decades. However, something that has changed is the fact that the baby boomer generation, the generation that fills the employment for farm animals mostly, is retiring fast, and therefore hastening the shortage.

Gender distribution

Historically, veterinary medicine used to be a man's world. Nowadays, most students in veterinary school are women, and it was in 2005 that women become the majority. For instance, out of the 77 new doctors from Tufts University, 62 of them are women, 75 percent of 2002 graduates were women, and 81 percent of those from the University of California at Davis were women. According to the Employment Policy Foundation, the number of female veterinarians since 1991 has more than doubled to 24,356, while the number of male veterinarians has fallen 15 percent to 33,461. This trend will continue based on the statistics of the applicant pool and the gender distribution in the various veterinary schools.

Women have also made these increased strides in other professions, such as law and medicine, where the distribution is half and half, but the number of women in veterinary medicine is shocking. Many veterinary students have reported that the reason for this is because veterinary salaries are not as competitive as those of other medical professions. Veterinarians average $70–80,000 a year, whereas physicians can easily average $150,000 a year. This shift in gender distribution can also be attributed to the personality

of this career and the qualities that would result in the most successful practice. Additionally, women are attracted to the flexible scheduling and part-time physicians are not very common, but part-time veterinarians are.

The shift of women becoming the majority in veterinary medicine have some negative effect in areas such as farm animal and food industry veterinarians, causing them to suffer. This is because women tend to not go into these fields and consequently the shortage that is produced has negative effects on the community as a whole .

Earnings

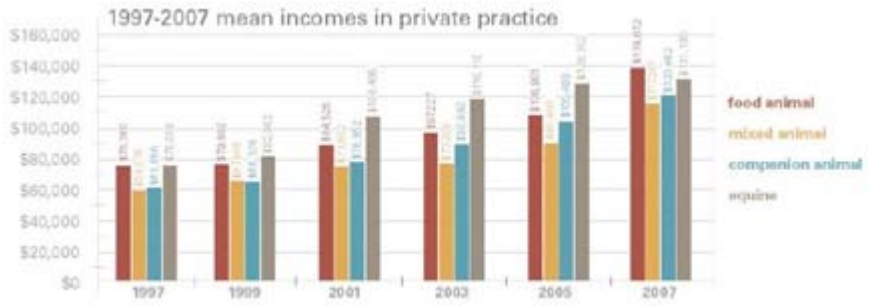
Veterinarians' incomes continued to increase during 2005–2007, but this increase is not expected to continue as much in the years of 2007-2009 . Salaries in the field of veterinary medicine vary depending on the individuals' experience, responsibility, location geographically, and field of employment . In particular at the end of 2007, veterinarians who worked in private practice earned more in comparison to many other areas of public practice, and men still earned more than women .

Furthermore, according to the survey done by the American Veterinary Medical Association, the average starting salaries of new graduates in 2006 depended upon their respective fields of practice. The Bureau of Labor Statistics in the Occupational Outlook Handbook, 2008-2009 Edition recorded the following

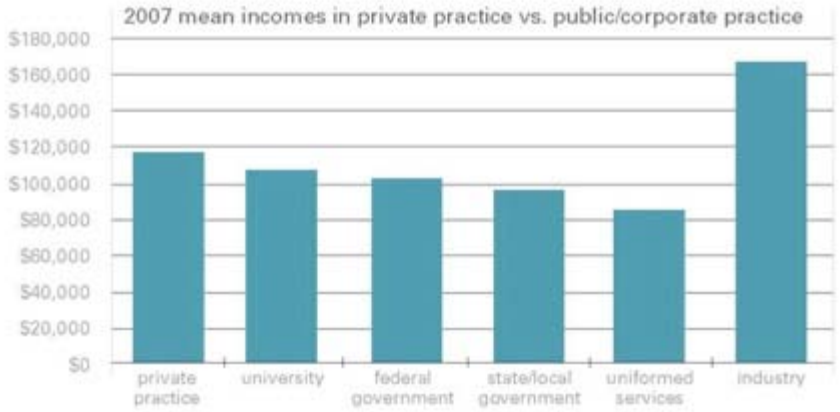
Large animals, exclusively: $61,029
Small animals, predominantly: $57,117
Small animals, exclusively: $56,241
Private clinical practice: $55,031
Large animals, predominately: $53,397
Mixed animals: $52,254
Equine (horses): $40,130

In addition in May 2006 the annual earnings of veterinarians was $71,990. These data range between fields, specialties, experience, and many other factors, but the middle 50 percent noted in the data provided earned $43,530 and $94,880. The lowest 10 percent earned less than $43,530 and the highest 10 percent earned more than $133,150. In particular, the average annual salary for veterinarians in the Federal Government was $84,335.

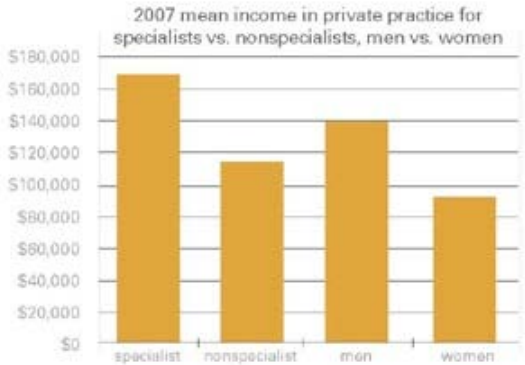
Veterinary incomes are up across the board, but some areas of employment are doing better than others, and the reasons for this were explored.



The average income for private practice rose from $105,510 in 2005 to $115,447 in 2007. These increased values exceed those of public practice including uniformed services and government .



On almost the same scale of income, disparities between specialists and nonspecialists are men and women.



Criticisms

Concerns about the role of veterinary surgeons in helping health threats survive and spread have been raised by several commentators, particularly with respect to pedigree dogs. Koharik Arman (2007) reached the following conclusion for example:

"Veterinarians also bear some responsibility for the welfare situation of purebred dogs. In fact, the veterinary profession has facilitated the evolution of purebred dogs. 'Breeds' that would not normally be sustainable are propagated by the compliance of veterinarians to breeder wishes." A finding that was echoed by Sir Patrick Bateson in his Independent Review of Dog Breeding following the broadcast of the BBC documentary Pedigree Dogs Exposed: "Its only the ready availability of modern veterinary medicine that has permitted some conditions…to become widespread."

Chapter 2

Albinism

Albinism is a congenital disorder characterized by the complete or partial absence of pigment in the skin, hair and eyes due to absence or defect of an enzyme involved in the production of melanin. Albinism results from inheritance of recessive gene alleles and is known to affect all vertebrates, including humans. The most common term used for an organism affected by albinism is "**albino**". Additional clinical adjectives sometimes used to refer to animals are "albinoid" and "albinic".

Albinism is associated with a number of vision defects, such as photophobia, nystagmus and astigmatism. Lack of skin pigmentation makes the organism more susceptible to sunburn and skin cancers.

Classification in humans

There are two main categories of albinism in humans:

- In **oculocutaneous albinism Types 1-4 with different levels with pigmentation** (despite its Latin-derived name meaning "eye-and-skin" albinism), pigment is lacking in the eyes, skin and hair. (The equivalent mutation in non-humans also results in lack of melanin in the fur, scales or feathers.) People with oculocutaneous albinism can have anything from no pigment at all to almost normal levels.
- In **ocular albinism**, only the eyes lack pigment. People who have ocular albinism have generally normal skin and hair color, although it is typically lighter than either parent. Many even have a normal eye appearance. Also, ocular albinism is generally sex-linked, therefore males are more likely to be affected. Males are without another X chromosome to mask recessive alleles on the X they inherit.

Other conditions include albinism as part of their presentation. These include Hermansky-Pudlak syndrome, Chediak-Higashi syndrome, Griscelli syndrome, Waardenburg syndrome, and Tietz syndrome. These conditions are sometimes classified with albinism. Several have sub-types. Some are easily distinguished by appearance, but in most cases genetic testing is the only way to be certain.

Albinism was formerly categorized as tyrosinase-positive or -negative. In cases of tyrosinase-positive albinism, the enzyme tyrosinase is present. The melanocytes (pigment cells) are unable to produce melanin for any one of a variety of reasons that do not directly involve the tyrosinase enzyme. In tyrosinase-negative cases, either the tyrosinase enzyme is not produced or a nonfunctional version is produced. This classification has been rendered obsolete by recent research.

Signs and symptoms

Most albinistic humans appear white or very pale as the melanin pigments responsible for brown, black, and some yellow colorations are not present.

Because individuals with albinism have skin that partially or entirely lacks the dark pigment melanin, which helps protect the skin from the sun's ultraviolet radiation, their skin can burn more easily from overexposure.

The human eye normally produces enough pigment to colour the iris and lend opacity to the eye. However, there are cases in which the eyes of an albinistic person appear red or purple, depending on the amount of pigment present. Lack of pigment in the eyes also results in problems with vision, related and unrelated to photosensitivity.

The albinistic are generally as healthy as the rest of the population, with growth and development occurring as normal, and albinism by itself does not cause mortality, although the lack of pigment increases the risk of skin cancer and other problems.

Visual problems

Development of the optical system is highly dependent on the presence of melanin, and the reduction or absence of this pigment in albinistic individuals may lead to

- Misrouting of the retinogeniculate projections, resulting in abnormal decussation (crossing) of optic nerve fibres
- Photophobia and decreased visual acuity due to light scattering within the eye
- Reduced visual acuity due to foveal hypoplasia and possibly light-induced retinal damage

Eye conditions common in albinism include:

- Nystagmus, irregular rapid movement of the eyes back and forth, or in circular motion.
- Refractive errors such as myopia or hyperopia and especially astigmatism
- Amblyopia, decrease in acuity of one or both eyes due to poor transmission to the brain, often due to other conditions such as strabismus.
- Optic nerve hypoplasia, underdevelopment of the optic nerve

Some of the visual problems associated with albinism arise from a poorly developed retinal pigment epithelium (RPE) due to the lack of melanin. This degenerate RPE causes foveal hypoplasia (a failure in the development of normal foveae), which results in eccentric fixation and lower visual acuity, and often a minor level of strabismus.

The iris is a sphincter formed from pigmented tissue that contracts when the eye is exposed to bright light, to protect the retina by limiting the amount of light passing through the pupil. In low light conditions the iris relaxes to allow more light to enter the eye. In albinistic subjects, the iris does not have enough pigment to block the light, thus the decrease in pupil diameter is only partially successful in reducing the amount of light entering the eye. Additionally, the improper development of the RPE, which in normal eyes absorbs most of the reflected sunlight, further increases glare due to light scattering within the eye. The resulting sensitivity (photophobia) generally leads to discomfort in bright light, but this can be reduced by the use of sunglasses and/or brimmed hats.

Genetics

Most forms of albinism are the result of the biological inheritance of genetically recessive alleles (genes) passed from both parents of an individual, though some rare forms are inherited from only one parent. There are other genetic mutations which are proven to be associated with albinism. All alterations, however, lead to changes in melanin production in the body.

The chance of offspring with albinism resulting from the pairing of an organism with albinism and one without albinism is low. However, because organisms can be carriers of genes for albinism without exhibiting any traits, albinistic offspring can be produced by two non-albinistic parents. Albinism usually occurs with equal frequency in both genders. An exception to this is ocular albinism, which it is passed on to offspring through X-linked inheritance. Thus, ocular albinism occurs more frequently in males as they have a single X and Y chromosome, unlike females, whose genetics are characterized by two X chromosomes.

There are two different forms of albinism; a partial lack of the melanin is known as hypomelanism, or hypomelanosis and the total absence of melanin is known as amelanism or amelanosis.

Diagnosis

Genetic testing can confirm albinism and what variety it is, but offers no medical benefits except in the cases of non-OCA disorders that cause albinism *along with* other medical problems which may be treatable. The *symptoms* of albinism can be treated by various methods detailed below.

Treatment

For the most part, treatment of the eye conditions consists of visual rehabilitation. Surgery is possible on the ocular muscles to decrease nystagmus, strabismus and common refractive errors like astigmatism. Strabismus surgery may improve the appearance of the eyes. Nystagmus-damping surgery can also be performed, to reduce the "shaking" of the eyes back and forth. The effectiveness of all these procedures varies greatly and depends on individual circumstances. More importantly, since surgery will not restore a normal RPE or foveae, surgery will not provide fine binocular vision. In the case of esotropia (the "crossed eyes" form of strabismus), surgery may help vision by expanding the visual field (the area that the eyes can see while looking at one point).

Glasses and other vision aids, large-print materials and CCTV, as well as bright but angled reading lights, can help individuals with albinism, even though their vision cannot be corrected completely. Some people with Albinism do well using bifocals (with a strong reading lens), prescription reading glasses, and/or hand-held devices such as magnifiers or monoculars (a very simple telescope). Contact lenses may be colored to block light transmission through the iris. But in case of nystagmus this is not possible, due to the irritation that is caused by the movement of the eyes. Some use bioptics, glasses which have small telescopes mounted on, in, or behind their regular lenses, so that they can look through either the regular lens or the telescope. Newer designs of bioptics use smaller light-weight lenses. Some US states allow the use of bioptic telescopes for driving motor vehicles.

Although still disputed among the experts, many ophthalmologists recommend the use of spectacles from early childhood onward to allow the eyes the best development possible.

Epidemiology

Albinism affects people from all races and its frequency across the human population is estimated to be approximately 1 in 20,000.

In other animals

Many animals with albinism lack their protective camouflage and are unable to conceal themselves from their predators or prey; the survival rate of animals with albinism in the wild is usually quite low. However the novelty of albino animals has occasionally led to their protection by groups such as the Albino Squirrel Preservation Society.

In partial albinism there can be a single patch or patches of skin that lack melanin. Especially in albinistic birds and reptiles, ruddy and yellow hues or other colors may be present on the entire body or in patches (as is common among pigeons), because of the presence of other pigments unaffected by albinism such as porphyrins, pteridines and psittacins, as well as carotenoid pigments derived from the diet.

In some animals albinism-like conditions may affect other pigments or pigment-production mechanisms:

- "Whiteface" a condition that affect some parrot species is caused by a lack of psittacins.
- Axanthism is a condition common in reptiles and amphibians, in which xanthophore metabolism is affected rather than synthesis of melanin, resulting in reduction or absence of red and yellow pteridine pigments.
- Leucism differs from albinism in that the melanin is, at least, partially absent but the eyes retain their usual color. Some leucistic animals are white or pale because of chromatophore (pigment cell) defects, and do not lack melanin.
- Melanism is the direct opposite of albinism. An unusually high level of melanin pigmentation (and sometimes absence of other types of pigment in species that have more than one) results in an appearance darker than non-melanistic specimens from the same genepool.

Intentionally bred albinistic strains of some animal species are commonly used as model organisms in biomedical study and experimentation, although some researchers have argued that they are not always the best choice. Examples include the BALB/c mouse and Wistar and Sprague Dawley rat strains, while albino rabbits were historically used for Draize toxicity testing. The *yellow* mutation in fruit flies is their version of albinism.

The incidence of albinism can be artificially increased in fish by exposing the eggs to heavy metals (arsenic, cadmium, copper, mercury, selenium, zinc).

The eyes of an albino animal appear red because the colour of the red blood cells in the underlying retinal blood vessels shows through where there is no pigment to obscure it.

Albino Red-necked Wallaby

An albino Wistar rat, a strain commonly used for both biomedical and basic research.

Albino deer

An Albino American Alligator

European Mole (*Talpa europaea* Linnaeus, 1758)

An Albino Kookaburra

Albino rabbit

An Albino Squirrel, (Colombo, Sri Lanka)

Chapter 3

Rabies

Rabies

Dog with rabies virus

ICD-10	A82.
DiseasesDB	11148
eMedicine	med/1374 eerg/493 ped/1974
MeSH	D011818

Rabies is a viral disease that causes acute encephalitis (inflammation of the brain) in warm-blooded animals. It is zoonotic (i.e., transmitted by animals), most commonly by a bite from an infected animal. Rabies is almost invariably fatal if post-exposure

prophylaxis is not administered prior to the onset of severe symptoms. The rabies virus infects the central nervous system, ultimately causing disease in the brain and death. The early symptoms of rabies in people are similar to that of many other illnesses, including fever, headache, and general weakness or discomfort. As the disease progresses, more specific symptoms appear and may include insomnia, anxiety, confusion, slight or partial paralysis, excitation, hallucinations, agitation, hypersalivation (increase in saliva), difficulty swallowing, and hydrophobia (fear of water). Death usually occurs within days of the onset of these symptoms.

The rabies virus travels to the brain by following the peripheral nerves. The incubation period of the disease is usually a few months in humans, depending on the distance the virus must travel to reach the central nervous system. Once the rabies virus reaches the central nervous system and symptoms begin to show, the infection is effectively untreatable and usually fatal within days.

Early-stage symptoms of rabies are malaise, headache and fever, progressing to acute pain, violent movements, uncontrolled excitement, depression, and hydrophobia. Finally, the patient may experience periods of mania and lethargy, eventually leading to coma. The primary cause of death is usually respiratory insufficiency. Worldwide, the vast majority of human rabies cases (approximately 97%) come from dog bites. In the United States, however, animal control and vaccination programs have effectively eliminated domestic dogs as reservoirs of rabies. In several countries, including the United Kingdom, Estonia and Japan, rabies carried by animals that live on the ground has been eradicated entirely. Concerns exist about airborne and mixed-habitat animals including bats. Bats in the U.K. and in some other countries carry European Bat Lyssavirus 1 and European Bat Lyssavirus 2. The symptoms of these viruses are similar to those of rabies and so the viruses are both known as bat rabies. An unvaccinated Scottish bat handler died from an EBLV infection in 2002.

The economic impact is also substantial, as rabies is a significant cause of death of livestock in some countries.

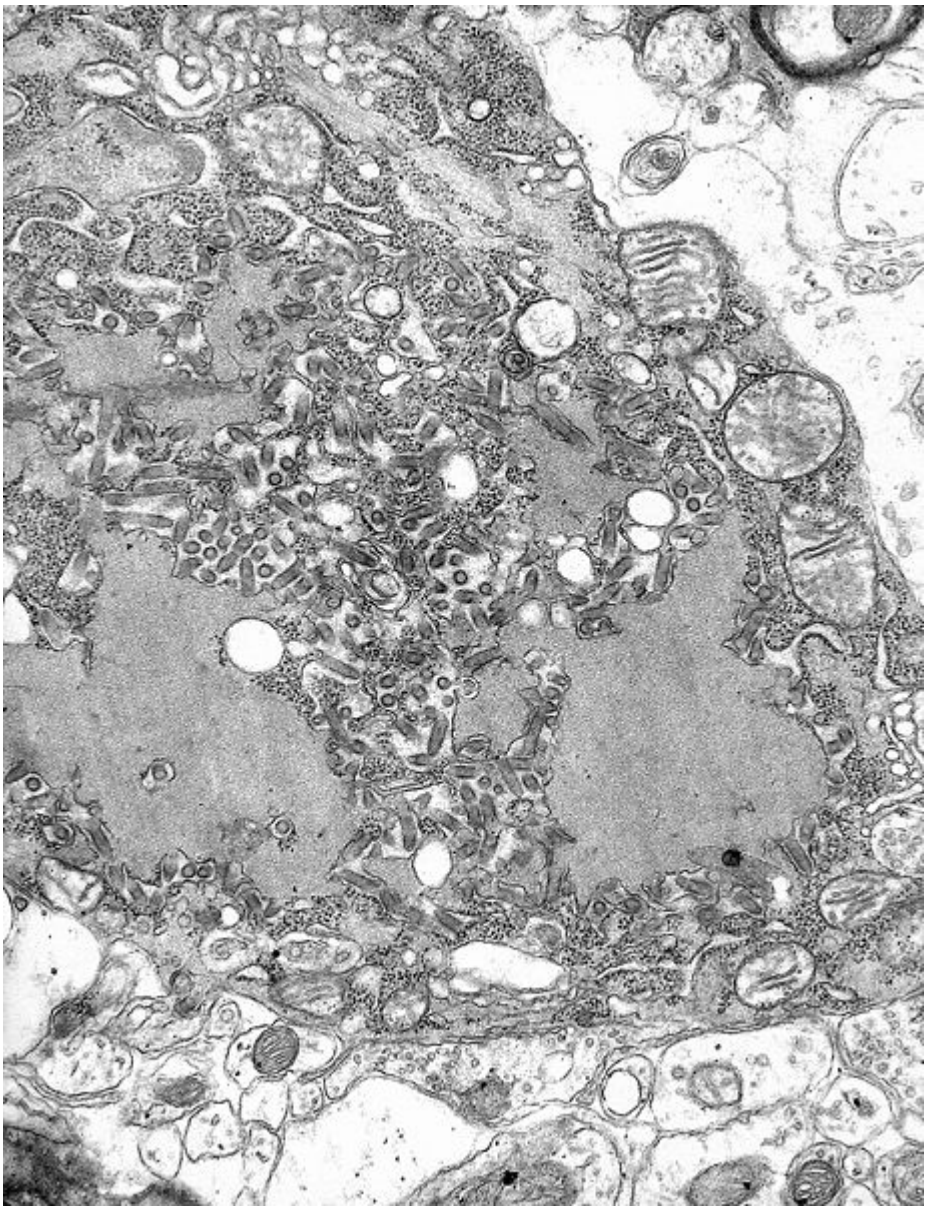
Signs and symptoms

The period between infection and the first flu-like symptoms is normally two to twelve weeks, but can be as long as two years. Soon after, the symptoms expand to slight or partial paralysis, cerebral dysfunction, anxiety, insomnia, confusion, agitation, abnormal behavior, paranoia, terror, hallucinations, progressing to delirium. The production of large quantities of saliva and tears coupled with an inability to speak or swallow are typical during the later stages of the disease; this can result in hydrophobia, in which the patient has difficulty swallowing because the throat and jaw become slowly paralyzed, shows panic when presented with liquids to drink, and cannot quench his or her thirst.

Death almost invariably results two to ten days after first symptoms. In 2005, the first patient was treated with the Milwaukee protocol, and Jeanna Giese became the first person ever recorded to survive rabies without receiving successful post-exposure

prophylaxis. An intention to treat analysis has since found that this protocol has a survival rate of about 8%.

Virology



TEM micrograph with numerous rabies virions (small, dark grey, rodlike particles) and Negri bodies (the larger pathognomonic cellular inclusions of rabies infection).

The rabies virus is the type species of the *Lyssavirus* genus, in the family *Rhabdoviridae*, order *Mononegavirales*. Lyssaviruses have helical symmetry, with a length of about 180 nm and a cross-sectional diameter of about 75 nm. These viruses are enveloped and have a single stranded RNA genome with negative-sense. The genetic information is packaged as a ribonucleoprotein complex in which RNA is tightly bound by the viral

nucleoprotein. The RNA genome of the virus encodes five genes whose order is highly conserved: nucleoprotein (N), phosphoprotein (P), matrix protein (M), glycoprotein (G) and the viral RNA polymerase (L).

From the point of entry, the virus is neurotropic, traveling quickly along the neural pathways into the central nervous system (CNS), and then further into other organs. The salivary glands receive high concentrations of the virus thus allowing further transmission.

Diagnosis

The reference method for diagnosing rabies is by performing PCR or viral culture on brain samples taken after death. The diagnosis can also be reliably made from skin samples taken before death. It is also possible to make the diagnosis from saliva, urine and cerebrospinal fluid samples, but this is not as sensitive. Inclusion bodies called Negri bodies are 100% diagnostic for rabies infection, but are found in only about 80% of cases. If possible, the animal from which the bite was received should also be examined for rabies.

The differential diagnosis in a case of suspected human rabies may initially include any cause of encephalitis, particularly infection with viruses such as herpesviruses, enteroviruses, and arboviruses (e.g., West Nile virus). The most important viruses to rule out are herpes simplex virus type 1, varicella-zoster virus, and (less commonly) enteroviruses, including coxsackieviruses, echoviruses, polioviruses, and human enteroviruses 68 to 71. In addition, consideration should be given to the local epidemiology of encephalitis caused by arboviruses belonging to several taxonomic groups, including eastern and western equine encephalitis viruses, St. Louis encephalitis virus, Powassan virus, the California encephalitis virus serogroup, and La Crosse virus.

New causes of viral encephalitis are also possible, as was evidenced by the recent outbreak in Malaysia of some 300 cases of encephalitis (mortality rate, 40%) caused by Nipah virus, a newly recognized paramyxovirus. Similarly, well-known viruses may be introduced into new locations, as is illustrated by the recent outbreak of encephalitis due to West Nile virus in the eastern United States. Epidemiologic factors (e.g., season, geographic location, and the patient's age, travel history, and possible exposure to animal bites, rodents, and ticks) may help direct the diagnostic workup.

Cheaper rabies diagnosis will be possible for low-income settings: accurate rabies diagnosis can be done at a tenth of the cost of traditional testing using basic light microscopy techniques.

Prevention

All human cases of rabies were fatal until a vaccine was developed in 1885 by Louis Pasteur and Émile Roux. Their original vaccine was harvested from infected rabbits, from which the virus in the nerve tissue was weakened by allowing it to dry for five to ten

days. Similar nerve tissue-derived vaccines are still used in some countries, as they are much cheaper than modern cell culture vaccines. The human diploid cell rabies vaccine was started in 1967; however, a new and less expensive purified chicken embryo cell vaccine and purified vero cell rabies vaccine are now available. A recombinant vaccine called V-RG has been successfully used in Belgium, France, Germany and the United States to prevent outbreaks of rabies in wildlife. Currently pre-exposure immunization has been used in both human and non-human populations, whereas in many jurisdictions domesticated animals are required to be vaccinated.

In the U.S., since the widespread vaccination of domestic dogs and cats and the development of effective human vaccines and immunoglobulin treatments, the number of recorded deaths from rabies has dropped from one hundred or more annually in the early twentieth century, to 1–2 per year, mostly caused by bat bites, which may go unnoticed by the victim and hence untreated.

September 28 is World Rabies Day, which promotes information on, and prevention and elimination of the disease.

Management

Post-exposure prophylaxis

Treatment after exposure, known as post-exposure prophylaxis (PEP), is highly successful in preventing the disease if administered promptly, generally within ten days of infection. Thoroughly washing the wound as soon as possible with soap and water for approximately five minutes is very effective at reducing the number of viral particles. “If available, a virucidal antiseptic such as povidone-iodine, iodine tincture, aqueous iodine solution, or alcohol (ethanol) should be applied after washing. Exposed mucous membranes such as eyes, nose or mouth should be flushed well with water.”

In the United States, the Centers for Disease Control and Prevention (CDC) recommend patients receive one dose of *human rabies immunoglobulin* (HRIG) and four doses of rabies vaccine over a fourteen day period. The immunoglobulin dose should not exceed 20 units per kilogram body weight. HRIG is very expensive and constitutes the vast majority of the cost of post-exposure treatment, ranging as high as several thousand dollars. As much as possible of this dose should be infiltrated around the bites, with the remainder being given by deep intramuscular injection at a site distant from the vaccination site. The first dose of rabies vaccine is given as soon as possible after exposure, with additional doses on days three, seven and fourteen after the first. Patients who have previously received pre-exposure vaccination do not receive the immunoglobulin, only the post-exposure vaccinations on day 0 and 2.

Modern cell-based vaccines are similar to flu shots in terms of pain and side effects. The old nerve-tissue-based vaccinations that require multiple painful injections into the abdomen with a large needle are cheap, but are being phased out and replaced by affordable WHO ID (intradermal) vaccination regimens.

Intramuscular vaccination should be given into the deltoid, not gluteal area which has been associated with vaccination failure due to injection into fat rather than muscle. In infants the lateral thigh is used as for routine childhood vaccinations.

An individual awakening to find a bat in the room, or finding a bat in the room of a previously unattended child or mentally disabled or intoxicated person is regarded as an indication for post-exposure prophylaxis. The recommendation for the precautionary use of post-exposure prophylaxis in occult bat encounters where there is no recognized contact has been questioned in the medical literature based on a cost-benefit analysis. However, recent studies have further confirmed the wisdom of maintaining the current protocol of precautionary administering of PEP in cases where a child or mentally compromised individual has been left alone with a bat, especially in sleep areas (where a bite/or exposure may occur while the victim is asleep and unaware or awake and unaware that a bite occurred). This is illustrated by the September 2000 case of a nine-year old boy from Quebec who died from rabies 3 weeks after being in the presence of a sick bat, even though there was no apparent report of a bite; as shown in the following conclusion made by the doctors involved in the case:

Despite recent criticism (45), the dramatic circumstances surrounding our patient's history, as well as increasingly frequent reports of human rabies contracted in North America, support the current Canadian guidelines which state that RPEP [PEP] is appropriate in cases where a significant contact with a bat cannot be excluded (45). The notion that a bite or an overt break in the skin needs to be seen or felt for rabies to be transmitted by a bat is a myth in many cases.

It is highly recommended that PEP be administered as soon as possible. Begun with little or no delay, PEP is 100% effective against rabies. In the case in which there has been a significant delay in administering PEP, the treatment should be administered regardless of that delay, as it may still be effective. If there has been a delay between exposure and attempts at treatment, such that the possibility exists that the virus has already penetrated the nervous system, the possibility exists that amputation of the affected limb might thwart rabies, if the bite or exposure was on an arm or leg. This treatment should be combined with an intensive PEP regimen.

Blood-brain barrier

Some recent work has shown that during lethal rabies infection, the blood-brain barrier (BBB) does not allow anti-viral immune cells to enter the brain, the primary site of rabies virus replication. This aspect contributes to the pathogenicity of the virus and artificially increasing BBB permeability promotes viral clearance. Opening the BBB during rabies infection has been suggested as a possible novel approach to treating the disease, even though no attempts have yet been made to determine whether or not this treatment could be successful.

Induced coma

In 2005, American teenager Jeanna Giese survived an infection of rabies unvaccinated. She was placed into an induced coma upon onset of symptoms and given ketamine, midazolam, ribavirin, and amantadine. Her doctors administered treatment based on the hypothesis that detrimental effects of rabies were caused by temporary dysfunctions in the brain and could be avoided by inducing a temporary partial halt in brain function that would protect the brain from damage while giving the immune system time to defeat the virus. After thirty-one days of isolation and seventy-six days of hospitalization, Giese was released from the hospital. She survived with almost no permanent sequelae and as of 2009 was starting her third year of university studies.

Giese's treatment regimen became known as the "Milwaukee protocol", which has since undergone revision (the second version omits the use of ribavirin). There were 2 survivors out of 25 patients treated under the first protocol. A further 10 patients have been treated under the revised protocol and there have been a further 2 survivors. The anesthetic drug ketamine has shown the potential for rabies virus inhibition in rats, and is used as part of the Milwaukee protocol.

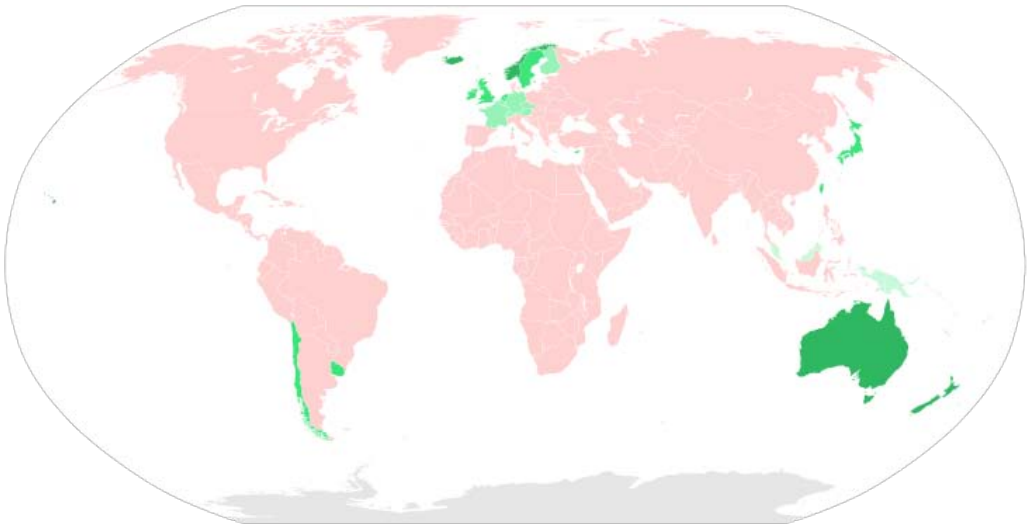
On April 10, 2008 in Cali, Colombia, an eleven year-old boy was reported to survive rabies and the induced coma without noticeable brain damage.

Prognosis

In unvaccinated humans, rabies is almost always fatal after neurological symptoms have developed, but prompt post-exposure vaccination may prevent the virus from progressing. Rabies kills around 55,000 people a year, mostly in Asia and Africa. There are only six known cases of a person surviving symptomatic rabies, and only one known case of survival in which the patient received no rabies-specific treatment either before or after illness onset.

The most current survival data using the Milwaukee protocol is available from the rabies registry.

Epidemiology



Rabies-free countries as of 2010

Transmission

Any warm-blooded animal (including humans) may become infected with the rabies virus and develop symptoms (although birds have only been known to be experimentally infected). Indeed the virus has even been adapted to grow in cells of poikilothermic ("cold-blooded") vertebrates. Most animals can be infected by the virus and can transmit the disease to humans. Infected bats, monkeys, raccoons, foxes, skunks, cattle, wolves, coyotes, dogs, mongoose (normally yellow mongoose) or cats present the greatest risk to humans. Rabies may also spread through exposure to infected domestic farm animals, groundhogs, weasels, bears and other wild carnivores. Small rodents such as squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, and mice and lagomorphs like rabbits and hares are almost never found to be infected with rabies and are not known to transmit rabies to humans.

The virus is usually present in the nerves and saliva of a symptomatic rabid animal. The route of infection is usually, but not always, by a bite. In many cases the infected animal is exceptionally aggressive, may attack without provocation, and exhibits otherwise uncharacteristic behavior.

Transmission between humans is extremely rare. A few cases have been recorded through transplant surgery.

After a typical human infection by bite, the virus enters the peripheral nervous system. It then travels along the nerves towards the central nervous system. During this phase, the virus cannot be easily detected within the host, and vaccination may still confer cell-mediated immunity to prevent symptomatic rabies. When the virus reaches the brain, it rapidly causes encephalitis. This is called the *prodromal* phase, and is the beginning of

the symptoms. Once the patient becomes symptomatic, treatment is almost never effective and mortality is over 99%. Rabies may also inflame the spinal cord producing transverse myelitis.

Prevalence

The rabies virus survives in widespread, varied, rural fauna reservoirs. It is present in the animal populations of almost every country in the world, except in Australia and New Zealand. In some countries like those in western Europe and Oceania, rabies is considered to be prevalent among bat populations only.

In Asia, parts of the Americas and large parts of Africa, dogs remain the principal host. Mandatory vaccination of animals is less effective in rural areas. Especially in developing countries, pets may not be privately kept and their destruction may be unacceptable. Oral vaccines can be safely distributed in baits, and this has successfully reduced rabies in rural areas of France, Ontario, Texas, Florida and elsewhere, like the City of Montréal, Québec, where baits are successfully used on raccoons in the Mont-Royal park area. Vaccination campaigns may be expensive, and cost-benefit analysis suggests that baits may be a cost effective method of control.

There are an estimated 55,000 human deaths annually from rabies worldwide, with about 31,000 in Asia, and 24,000 in Africa. One of the sources of recent flourishing of rabies in East Asia is the pet boom. China introduced the “one-dog policy” in the city of Beijing in November 2006 to control the problem. India has been reported as having the highest rate of human rabies in the world, primarily because of stray dogs. As of 2007, Vietnam had the second-highest rate, followed by Thailand; in these countries too the virus is primarily transmitted through canines (feral dogs and other wild canine species). Recent reports suggest that wild rabid dogs are roaming the streets. Because much cheaper pre-vaccination is not commonly administered in places like Thailand, the expense for lack of preparation with far more costly post-exposure prophylaxis can hit families hard.

Rabies was once rare in the United States outside the Southern states, but as of 2006, raccoons in the mid-Atlantic and northeast United States had been suffering from a rabies epidemic since the 1970s, which was moving westwards into Ohio. In the midwestern United States, skunks are the primary carriers of rabies, comprising 134 of the 237 documented non-human cases in 1996.

History

Etymology

The term is derived from the Latin *rabies*, "madness". This, in turn, may be related to the Sanskrit *rabhas*, "to do violence". The Greeks derived the word "lyssa", from "lud" or "violent"; this root is used in the name of the genus of rabies *lyssavirus*.

Impact

Because of its potentially violent nature, rabies has been known since c.2000 B.C. The first written record of rabies is in the Codex of Eshnunna (ca. 1930 BC), which dictates that the owner of a dog showing symptoms of rabies should take preventive measure against bites. If another person was bitten by a rabid dog and later died, the owner was heavily fined.

Rabies was considered a scourge for its prevalence in the 19th century. Fear of rabies related to methods of transmissions was almost irrational; however, this gave Louis Pasteur ample opportunity to test post-exposure treatments from 1885.

In other animals

Rabies is infectious to mammals. Three stages of rabies are recognized in dogs and other animals. The first stage is a one- to three-day period characterized by behavioral changes and is known as the prodromal stage. The second stage is the excitative stage, which lasts three to four days. It is this stage that is often known as *furious rabies* for the tendency of the affected dog to be hyperreactive to external stimuli and bite at anything near. The third stage is the paralytic stage and is caused by damage to motor neurons. Incoordination is seen owing to rear limb paralysis and drooling and difficulty swallowing is caused by paralysis of facial and throat muscles. Death is usually caused by respiratory arrest.

As recently as 2004, a new symptom of rabies has been observed in foxes. Probably at the beginning of the prodromal stage, foxes, which are extremely cautious by nature, seem to lose this instinct. Foxes will come into settlements, approach people, and generally behave as if tame. How long such "euphoria" lasts is not known. But even in this state such animals are extremely dangerous, as their saliva and secretions still contain the virus, and they remain very unpredictable.

Chapter 4

Equine Exertional Rhabdomyolysis

Equine exertional rhabdomyolysis (**ER**, also known as **tying up**, **azoturia**, or **Monday morning disease**) is a syndrome that damages the muscle tissue in horses. It is usually due to overfeeding a horse carbohydrates and appears to have a genetic link.

Causes and process of ER

Beyond a highly probable hereditary factor, there does not seem to be a single cause that triggers ER in horses. Exercise is seen in every case, but exercise is always accompanied by another factor. It is likely that several factors must act together in order to cause an ER attack.

Other possible factors include:

- The overfeeding of non-structural carbohydrates (grain and pellets, for example)
- Poor conditioning or fitness, sudden increase of workload
- The work of a horse after a period of rest, if the concentrate ration was not reduced
- Electrolyte or mineral imbalances, especially seen with potassium
- A deficiency in selenium or vitamin E
- Imbalance of hormones, including the reproductive hormones in nervous fillies and mares and thyroid hormones in horses with hypothyroidism
- Wet, cold, or windy weather conditions

The more factors that are present, the greater the likelihood that the horse will develop ER. However, the most common cause of ER is an imbalance between the animal's diet and his workload, especially when he has a high-grain diet.

ER occurs when there is an inadequate flow of blood to the muscles of an exercising horse. The muscle cells, lacking in oxygen, begin to function anaerobically to produce the needed ATP. The anaerobic work creates a buildup of waste products, acid, and heat. This subsequently alters the cell by preventing the cell's enzymes from functioning and the myofilaments from efficiently contracting. The cell membranes may then be damaged

if the horse is forced to continue work, which allows muscle enzymes and myoglobin to leak into the bloodstream.

The body builds up a store of glycogen from converted carbohydrates in muscle cells. Glycogen, a fuel used by muscles for energy, is depleted during work and restocked when a horse rests. Oxygen-carrying blood metabolizes glycogen, but the blood can not flow fast enough to metabolise the excess stored glycogen. The glycogen that is not metabolized aerobically (by the oxygenated blood) must then be metabolized anaerobically, which then creates the cell waste products and heat, and ER has begun. A horse on a high-grain diet with little work collects more glycogen in its muscles than it can use efficiently when exercise begins, which is why horses on a high-grain diet are more likely to develop ER.

Proper conditioning can help prevent ER, as it promotes the growth of capillaries in muscles and the number of enzymes used for energy production in muscle cells. However, improvement in these areas can take several weeks. Thus, ER is more common in horses that are only worked sporadically or lightly, and in horses just beginning an exercise regimen.

A common misconception is that ER is caused by the buildup of lactic acid. Lactate is *not* a waste product for a cell, but a fuel, used when the cell's oxygen supply is insufficient. Lactate does not damage a cell, but is rather a byproduct of the true cause of cell damage: inadequate blood supply and altered cell function. Lactate naturally builds up in an exercising horse without harming the muscle cells, and is metabolized within an hour afterward.

The pain is caused by the inadequate blood flow to the muscle tissue, the inflammation from the resulting cell damage, and the release of cell contents. Muscle spasms, caused by the lack of blood to the muscle tissue, are also painful.

Symptoms

A horse developing ER will usually begin showing signs right after the beginning of exercise, although for mild cases, signs may not be seen until after the horse is cooled out. Signs include reluctance to move, stiffness or shortened gait when the animal is forced to move, and muscle spasms or cramps, with hard, painful muscles (especially the hindquarters) when palpated. If an observer is unfamiliar with ER, initial symptoms may appear to be tiredness or perhaps lameness but the condition is far more complex.

Signs of a severe bout of ER may include: reluctance to move, sweating, elevated heart and respiratory rates as a result of the pain, anxious expression, shifting of weight from side to side, standing hunched and tense, passing reddish-brown urine, dehydration, shock, and inability to rise. Usually there is a correlation between how long it takes the signs to be seen and how severe the bout of ER is, with the more severe bouts of ER displaying signs right after work has begun.

If signs of ER are seen, the horse should not be moved. Movement can cause further muscle damage. If the animal is far from the barn, it is best to trailer him back rather than move him.

After a bout of ER, blood levels of CPK and AST rise.

Treatment

Mild or moderate cases

The horse should receive several days of NSAIDs, rest, and grain or pellets should be withheld. To improve blood flow to the muscles and help to with muscle spasms, heat therapy and Equine Massage may be beneficial, as well as hand-walking if the horse is comfortable walking. Turn-out in a pasture or paddock will encourage movement. A horse should be moving normally within 12–36 hours after the attack.

Severe cases

A horse may need fluids, especially if his urine is colored, the horse is receiving NSAIDs, or if he is dehydrated. Fluids will increase the production of urine that will in turn help flush out the excess, and potentially damaging, myoglobin from the kidneys and will reduce NSAID-produced kidney damage. Fluids should be administered until the urine is clear, which usually takes from a few hours to a few days.

Vasodilators, such as acepromazine, can help improve blood flow to the muscles. However, the owner should only give acepromazine if it is prescribed by the horse's veterinarian, as it can lower the animal's blood pressure and can cause collapse in a severely dehydrated horse. The human drug dantrolene is sometimes given to alleviate the muscle spasms and prevent further degeneration of muscle tissue.

Vitamin E is an anti-oxidant, and so may help prevent further cell degeneration in the affected muscles. However, vitamin E products must be used with caution if they also contain selenium.

Bicarbonate will not help offset any lactic acid in the bloodstream, as lactic acid generally only accumulates in the affected muscles.

Except to get a horse to his stall, a horse showing signs of severe ER should not be moved until he is comfortable enough to do so eagerly. This may take several days. After this point, it is important to either hand-walk the horse a few times each day, or provide him with a few hours of turnout in a pasture or paddock.

Returning the animal to work

A horse may be returned to work after it is no longer showing signs of ER, and is no longer on NSAIDs—which can hide signs of another bout of ER. If NSAIDs are needed

to keep the horse comfortable, or if the horse is reluctant to continue work, the animal is not yet ready for a return to his regular training program. Blood tests should reveal that the horse's CPK concentration and AST levels are normal before the horse is returned to work.

To begin bringing the horse back, he should be exercised at the walk and trot for 10–15 minutes at least once every day. This regimen will gradually be increased as the horse becomes more willing. For a moderate or severe bout of ER, it may take 4–6 weeks to return to the regular program. It is important not to push the horse more than he is ready or a relapse may occur. A second bout of ER is usually more severe than the first, not only taking the horse out of training for a longer time, but possibly causing permanent muscle damage.

Grain is gradually reintroduced as exercise resumes, but grain can contribute to the development of ER.

Prognosis

For mild to moderate cases of ER, the prognosis is excellent, with the horse successfully returning to its former level of competition. However, if the vet's recommendations for preventing ER are not followed, ER may likely recur.

Horses who experience a severe case of ER (the muscle degeneration is significant) are less likely to return to their previous level of competition, as fibrosis may have occurred, which would result in loss of muscle function. The prognosis is guarded for these horses.

Prevention

Diet

Reducing any extra energy in a horse's diet is essential to maintaining a horse that has experienced ER. Decreasing carbohydrates and increasing the daily intake of hay or pasture can usually accomplish this. Grain may need to be cut out altogether and replaced by a substitute, such as vegetable oil, to meet the individual energy needs of the horse.

Grain should be reduced or removed from a horse's ration on days when he cannot be worked.

Exercise

Proper conditioning is very important in preventing ER. Beginning with a base of long, slow distance work will ensure that the horse has a foundation before proceeding on to more strenuous work. The horse should always have a 10-minute warm-up at the walk and trot before more strenuous work is begun, and should always have a proper cool down of 10 minutes.

It is best that a horse receive exercise every day, or possibly twice a day, to prevent the recurrence of ER. If possible, avoid breaks in the horse's exercise schedule. Training, riding, driving, longeing, or turnout are all suitable.

Daily pasture turnout is ideal for horses likely to suffer from ER, as it provides exercise and adds roughage to the animal's diet.

Supplements and drugs

As with any supplements and drugs, it is best to confer with a veterinarian as to the recommended dosages. Some drugs are not allowed in competition and may need to be withheld a few days before.

Adding potassium and salt to the diet may be beneficial to horses that suffer from recurrent bouts of ER. Horses in hard training may need a vitamin E supplement, as their requirements are higher than horses in more moderate work. The horse may also be deficient in selenium, and need a feed in supplement. Selenium can be dangerous if overfed, so it is best to have a blood test to confirm that the horse is in need of supplemental selenium.

Thyroid hormone supplementation is often beneficial for horses with low thyroid activity (only do so if the horse has been diagnosed with hypothyroidism).

Other drugs that have been used with success include phenytoin, dantrolene, and dimetyl glycine.

Bicarbonate and NSAIDs are of no use in preventing ER.

Chapter 5

Zoonosis

Zoonosis

DiseasesDB	28555
MeSH	D015047

A **zoonosis** or **zoonose** is any infectious disease that can be transmitted (in some instances, by a vector) from non-human animals, both wild and domestic, to humans or from humans to non-human animals (the latter is sometimes called **reverse zoonosis** or anthroponosis). Of the 1415 pathogens known to affect humans, 61% are zoonotic. The emergence of a pathogen into a new host species is called *disease invasion*.

The emerging interdisciplinary field of conservation medicine, which integrates human and veterinary medicine, and environmental sciences, is largely concerned with zoonoses.

A partial list of agents that can carry infectious organisms that may be zoonotic are listed below. Xenozoonosis is zoonosis transmitted by xenotransplantation (transplantation between species).

- Assassin bugs
- Bats
- Bank voles
- Birds
- Cats
- Cattle
- Chimpanzees
- Dogs
- Fish
- Fleas
- Flies
- Hamsters
- Horses
- Humans
- Hyraxes
- Lice
- Mice
- Monkeys
- Mosquitos
- Opossums
- Pigs
- Rabbits and hares
- Rodents
- Sloths
- Sheep
- Snails
- Ticks
- Wolves

- Geese
- Goats
- Raccoons
- Rats

List of infectious agents

Zoonoses can be listed according to the infectious agent:

- Parasites
  - protozoa, helminths (nematodes, cestodes and trematodes)
- Fungi
- Bacteria
- Viruses
- Pria

Partial list of zoonoses

- Anthrax
- Babesiosis
- Balantidiasis
- Barmah Forest virus
- Bartonellosis
- Bilharzia
- Bolivian hemorrhagic fever
- Brucellosis
- Borrelia (Lyme disease and others)
- Borna virus infection
- Bovine tuberculosis
- Campylobacteriosis
- Cat Scratch Disease
- Chagas disease
- Chlamydophila psittaci
- Cholera
- Cowpox
- Creutzfeldt-Jakob disease (vCJD),

  a transmissible spongiform encephalopathy (TSE) from bovine spongiform encephalopathy (BSE) or "mad cow disease"

- Crimean-Congo
- Escherichia coli O157:H7
- Erysipelothrix rhusiopathiae
- Eastern equine encephalitis virus
- Western equine encephalitis virus
- Venezuelan equine encephalitis virus
- Giardia lamblia
- H1N1 flu
- Hantavirus
- Helminths
- Hendra virus
- Henipavirus
- Korean hemorrhagic fever
- Kyasanur forest disease
- Lábrea fever
- Lassa fever
- Leishmaniasis
- Leptospirosis
- Listeriosis
- Lymphocytic choriomeningitis virus
- Marburg fever
- Mediterranean
- Oropouche fever
- Pasteurellosis
- Plague
- Puumala virus
- Q-Fever
- Psittacosis, or "parrot fever"
- Rabies
- Rift Valley fever
- Ringworms (*Tinea canis*)
- Rotavirus
- Salmonellosis
- Sodoku
- Sparganosis
- Streptococcus suis
- Toxocariasis
- Toxoplasmosis
- Trichinosis
- Tularemia, or "rabbit fever"
- Typhus of Rickettsiae
- Venezuelan hemorrhagic fever
- Visceral larva migrans
- West Nile virus
- Yellow fever

hemorrhagic fever
- Cryptosporidiosis
- Cutaneous larva migrans
- Dengue fever
- Ebola
- Echinococcosis

spotted fever
- Mycobacterium marinum
- Monkey B
- Nipah fever
- Ocular larva migrans
- Omsk hemorrhagic fever
- Ornithosis (psittacosis)
- Orf (animal disease)

- Yersiniosis

Other zoonoses might be:

- Glanders
- SARS (possibly; civet cats may spread the disease, or may catch the disease from humans.)

Historical development of zoonotic diseases

Most of human prehistory was spent as small bands of hunter-gatherers; these bands were rarely larger than 150 individuals, and were not in contact with other bands very often. Because of this, epidemic or pandemic diseases, which depend on a constant influx of humans who have not developed an immune response, tended to burn out after their first run through a population. To survive, a biological pathogen had to be a chronic infection, stay alive in the host for long periods, or have a non-human reservoir in which to live while waiting for new hosts to pass by. In fact, for many 'human' diseases, the human is actually an accidental victim and a dead-end host. (This is the case with rabies, anthrax, tularemia, West Nile virus, and many others). Thus, much of human development has been in relation to zoonotic, not epidemic, diseases.

Many modern diseases, even epidemic diseases, started out as zoonotic diseases. It is hard to be certain which diseases jumped from other animals to humans, but there is good evidence that measles, smallpox, influenza, HIV, and diphtheria came to us this way. The common cold, and tuberculosis may also have started in other species.

In modern days, zoonoses are of practical interest because they are often previously unrecognized diseases or have increased virulence in populations lacking immunity. The West Nile virus appeared in the United States in 1999 in the New York City area, and moved through the country in the summer of 2002, causing much distress. Bubonic plague is a zoonotic disease, as are salmonella, Rocky Mountain spotted fever, and Lyme disease.

The major factor contributing to the appearance of new zoonotic pathogens in human populations is increased contact between humans and wildlife (Daszak *et al.*., 2001). This can be caused either by encroachment of human activity into wilderness areas or by movement of wild animals into areas of human activity due to anthropological or environmental disturbances. An example of this is the outbreak of Nipah virus in peninsular Malaysia in 1999, when intensive pig farming intruded into the natural habitat of fruit bats carrying the virus. Unidentified spillover events caused infection of the pig population which acted as an amplifier host, eventually transmitting the virus to farmers and resulting in 105 human deaths (Field *et al.*., 2001).

Similarly, in recent times avian influenza and West Nile virus have spilled over into human populations probably due to interactions between the carrier host and domestic animals. Highly mobile animals such as bats and birds may present a greater risk of zoonotic transmission than other animals due to the ease with which they can move into areas of human habitation.

Diseases like malaria, schistosomiasis, river blindness, and elephantiasis are *not* zoonotic, even though they may be transmitted by insects or use intermediate hosts vectors, because they depend on the human host for part of their life-cycle.

Archaeological examples of Zoonosis

Partial list of outbreaks of zoonosis associated with fairs and petting zoos

Outbreaks of zoonosis have been traced to human interaction with and exposure to animals at fairs, petting zoos, and in other settings. In 2005, the Centers for Disease Control and Prevention (CDC) issued an updated list of recommendations for preventing zoonosis transmission in public settings. The CDC recommendations, which were developed in conjunction with the National Association of State Public Health Veterinarians, include sections on the educational responsibilities of venue operators, managing public and animal contact, and animal care and management.

In 1988, a person became ill with swine influenza virus (swine flu) and died after visiting the display area of the pig barn at a Wisconsin county fair. Three healthcare personnel treating the case patient also developed flu-like illness with laboratory evidence of swine influenza virus infection. Investigators from the CDC indicated in their final report that the swine flu had been transmitted directly from pig to human host.

In 1994, seven cases of *E. coli* O157:H7 infection were traced to a farm in Leicestershire, United Kingdom. An epidemiological investigation into the outbreak revealed that the strain of *E. coli* O157:H7 isolated from nine animals on the farm was indistinguishable from the strain isolated from human samples. Investigators concluded that the most likely cause of this outbreak was direct human contact with animals.

In 1995, 43 children who had visited a rural farm in Wales became ill with Cryptosporidiosis. *Cryptosporidium* was isolated from seven of the ill children. An epidemiological investigation indicated that the source of the children's illness was contact with calves at the farm.

Also in 1995, at least 13 children became ill with *Cryptosporidiosis* after visiting a farm in Dublin, Ireland. In a case-control study, researchers compared the activities of the 13 ill children, or cases, to the activities of 52 out of 55 people who had visited the farm – the controls. The study revealed that illness was significantly associated with playing in the sand in a picnic area beside a stream where animals had access.

In 1997, an *E. coli* O157:H7 outbreak was identified among one child who lived on an open farm and two children who visited the farm during school parties. Two of the three children developed hemolytic-uremic syndrome (HUS). Isolates collected from the three children and from samples taken at the farm were indistinguishable, demonstrating evidence of the link between the farm and the children's illness.

In 1999, what is believed to be the largest outbreak of waterborne *E. coli* O157:H7 illness in United States history occurred at the Washington County, New York fair. The New York State Department of Health identified 781 individuals who were suspected of being infected with either *E. coli* O157:H7 or *Campylobacter jejuni*. An investigation into the outbreak revealed that consumption of beverages purchased from vendors supplied with water drawn from an unchlorinated fairgrounds well was associated with illness. In all, 127 outbreak victims were confirmed ill with *E. coli* O157:H7 infections; 71 were hospitalized, 14 developed HUS, and two died.

In 2000, 51 people became ill with confirmed or suspected *E. coli* O157:H7 infections after visiting a dairy farm in Pennsylvania. Eight children developed HUS. A case-control study among visitors to the dairy was conducted jointly by the CDC, Pennsylvania Department of Health, and the Montgomery County Health Department. The study's authors concluded that *E. coli* was transmitted to visitors as a result of contamination on animal hides and in the environment.

Also in 2000, 43 visitors to the Medina County fair in Ohio were confirmed ill with *E. coli* O157:H7 infections. An investigation into the outbreak suggested that the water system from which food vendors were supplied was the source of the *E. coli* outbreak. Several months later, five children became ill with *E. coli* infections after attending a "Carnival of Horrors" event held at the Medina County fairgrounds. PFGE analysis of the strains of *E. coli* isolated from members of both outbreaks revealed an indistinguishable pattern, and investigators from the Medina County Health Department and the CDC determined that the Medina County Fairgrounds water distribution system was the source of both *E. coli* outbreaks.

In 2001, an *E. coli* O157:H7 outbreak was traced to exposure in the Cow Palace at the Lorain County Fair in Ohio. CDC investigators identified 23 cases of *E. coli* infection associated with attendance at the Lorain County Fair, with additional secondary cases

likely. Two people developed HUS. An environmental and site investigation revealed *E. coli* contamination on doorways, rails, bleachers, and sawdust. Investigators concluded that the Lorain County Fair was the source of the outbreak.

Wyandot County, Ohio, also reported an *E. coli* O157:H7 outbreak in 2001. Ninety-two *E. coli* infections were reported to the Wyandot County Health Department and the CDC, with 27 cases confirmed using laboratory analysis. Two cases developed HUS. Contact with infected cattle was believed to be the source of the outbreak; however, a specific cause was never identified.

In 2002, seven people became ill with *E. coli* O157:H7 infections after visiting a large agricultural fair in Ontario, Canada. Outbreak investigators conducted a case-control study, which indicated that goats and sheep from a petting zoo were the source of the *E. coli* among fair visitors. Other indications were that the fencing and environment surrounding the petting zoo could have been a source of transmission.

What is believed to be the largest *E. coli* O157:H7 outbreak in Oregon history occurred among attendees at the Lane County fair in 2002. An Oregon Department of Human Services – Health Services investigation led to the belief that the *E. coli* outbreak originated from exposure in the sheep and goat barn. In all, 79 people were confirmed ill with *E. coli* infections as part of the outbreak; 22 were hospitalized, and 12 suffered HUS.

In 2003, fair visitors and animal exhibitors at the Fort Bend County Fair in Texas became ill with *E. coli* O157:H7 infections. An outbreak investigation led to the determination that 25 people had become ill with *E. coli* infections after attending the Fort Bend County Fair; seven people were laboratory-confirmed with *E. coli*, and 5 developed HUS or TTP (Thrombotic thrombocytopenic purpura). Investigators isolated a strain of *E. coli* indistinguishable from the outbreak strain from four animal husbandry sites, and found high levels of *E. coli* contamination in both rodeo and animal exhibit areas.

In 2004, a large *E. coli* O157:H7 outbreak occurred among visitors at the 2004 North Carolina State Fair. During its investigation into the outbreak, the North Carolina Department of Health and Human Services (NCDHHS) received over 180 reports of illness, and documented 33 culture-confirmed cases of *E. coli* O157:H7 associated with attendance at the fair, with 15 children developing HUS. In its final investigation report, NCDHHS concluded that the North Carolina State Fair *E. coli* outbreak had originated at a petting zoo exhibit. The conclusion was supported by a case-control study, environmental sampling, and laboratory analysis of samples collected from the fair and members of the outbreak.

In 2005, a petting zoo that exhibited at two Florida fairs and a festival was traced as the source of an *E. coli* O157:H7 outbreak. Sixty-three people who had visited either the Florida State Fair, the Central Florida Fair, or the Florida Strawberry Festival reported illness to investigators for the Florida Department of Health, including 20 who were

culture-confirmed and 7 with HUS. A case-control study revealed that illness was associated with exposure to a petting zoo exhibit present at all three events.

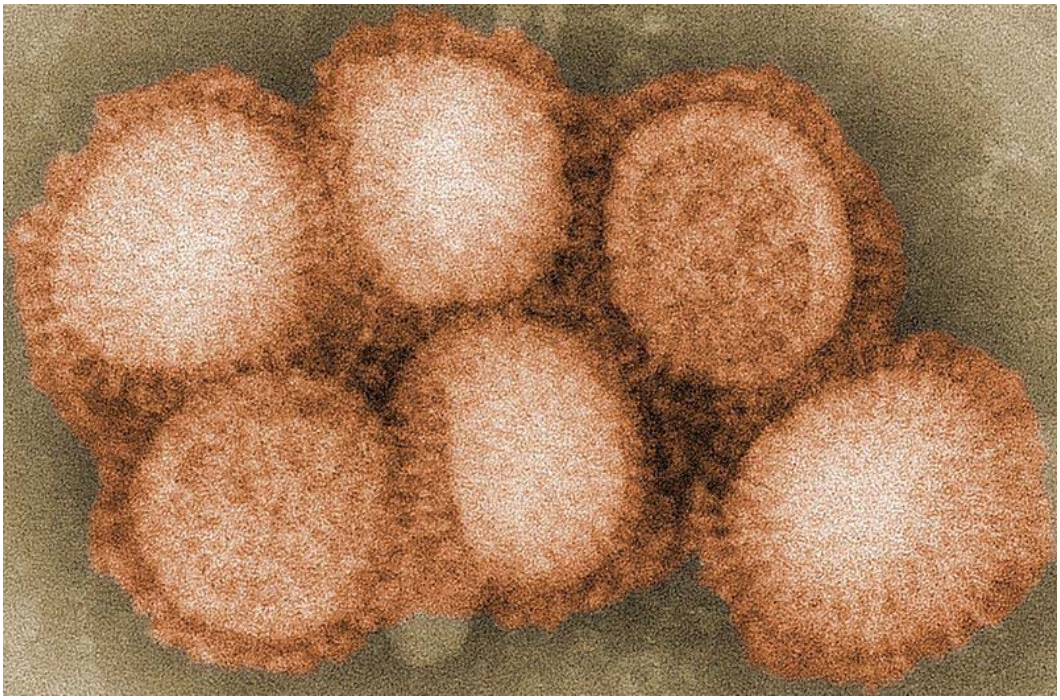
Contribution of zoonotic pathogens to foodborne illness

The most significant zoonotic pathogens that cause the foodborne diseases are those of *Escherichia coli* O157:H7, *Campylobacter*, *Caliciviridae*, and *Salmonella*.

In 2006, a conference held in Berlin was focusing on the issue of zoonotic pathogen effects on food safety, urging governments to intervene the problem, and the public to be vigilant towards the risks of catching food-borne diseases via the route of farm-to-dining table.

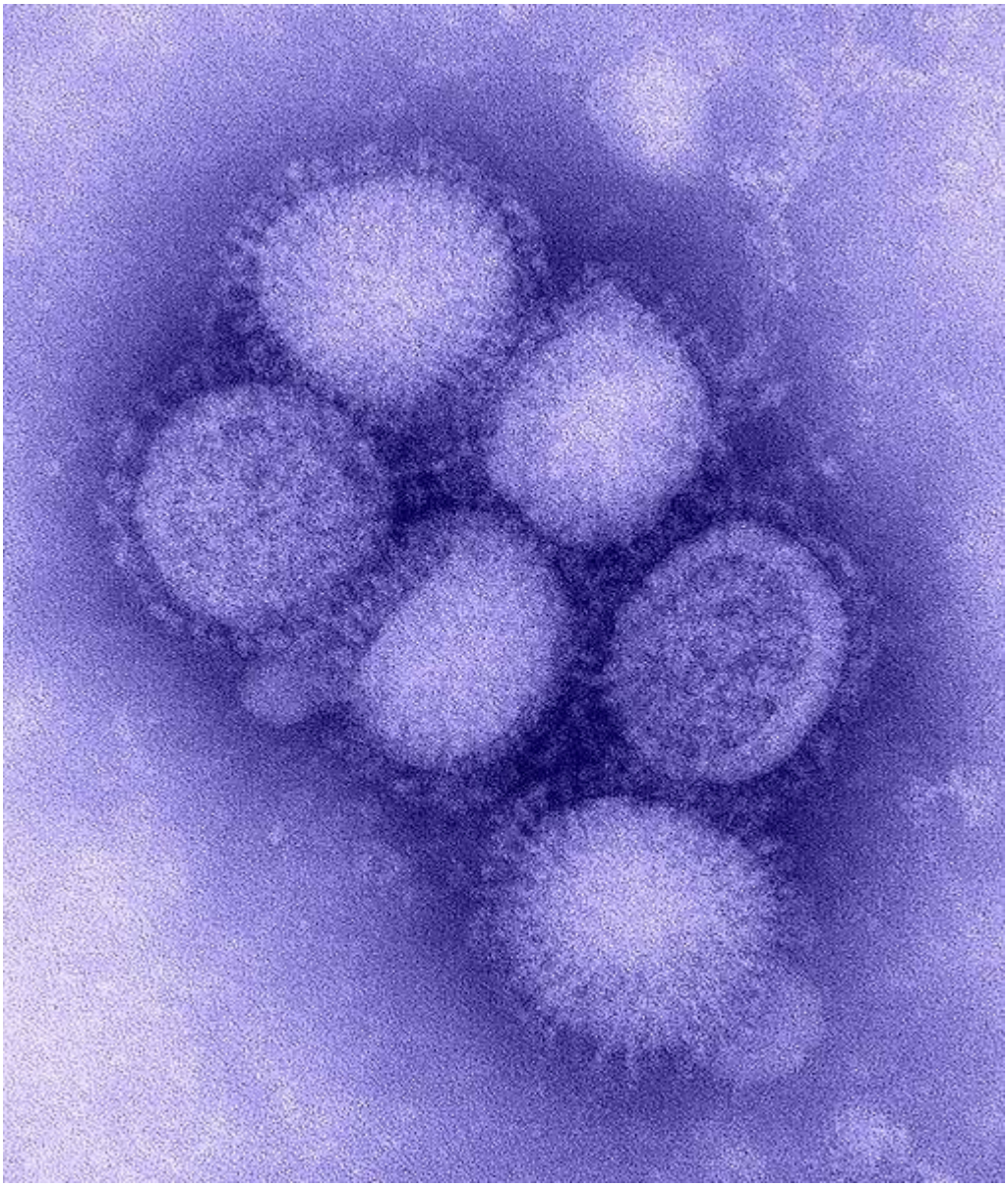
Chapter 6

Swine Influenza



Swine influenza, also called **pig influenza**, **swine flu**, **hog flu** and **pig flu**, is an infection by any one of several types of swine influenza virus. **Swine influenza virus** (**SIV**) or **S-OIV** (**swine-origin influenza virus**) is any strain of the influenza family of viruses that is endemic in pigs. As of 2009, the known SIV strains include influenza C and the subtypes of influenza A known as **H1N1**, H1N2, H3N1, H3N2, and H2N3.

Swine influenza virus is common throughout pig populations worldwide. Transmission of the virus from pigs to humans is not common and does not always lead to human influenza, often resulting only in the production of antibodies in the blood. If transmission does cause human influenza, it is called zoonotic swine flu. People with regular exposure to pigs are at increased risk of swine flu infection. The meat of an infected animal poses no risk of infection when properly cooked.



Electron microscope image of the reassorted H1N1 influenza virus photographed at the CDC Influenza Laboratory. The viruses are 80–120 nanometres in diameter.

During the mid-20th century, identification of influenza subtypes became possible, allowing accurate diagnosis of transmission to humans. Since then, only 50 such transmissions have been confirmed. These strains of swine flu rarely pass from human to human. Symptoms of zoonotic swine flu in humans are similar to those of influenza and of influenza-like illness in general, namely chills, fever, sore throat, muscle pains, severe headache, coughing, weakness and general discomfort.

In August 2010 the World Health Organization declared the swine flu pandemic officially over.

Classification

Of the three genera of influenza viruses that cause human flu, two also cause influenza in pigs, with influenza A being common in pigs and influenza C being rare. Influenza B has not been reported in pigs. Within influenza A and influenza C, the strains found in pigs and humans are largely distinct, although because of reassortment there have been transfers of genes among strains crossing swine, avian, and human species boundaries.

Influenza C

Influenza C viruses infect both humans and pigs, but do not infect birds. Transmission between pigs and humans have occurred in the past. For example, influenza C caused small outbreaks of a mild form of influenza amongst children in Japan and California. Because of its limited host range and the lack of genetic diversity in influenza C, this form of influenza does not cause pandemics in humans.

Influenza A

Swine influenza is known to be caused by influenza A subtypes H1N1, H1N2, H2N3, H3N1, and H3N2. In pigs, three influenza A virus subtypes (H1N1, H1N2, and H3N2) are the most common strains worldwide. In the United States, the H1N1 subtype was exclusively prevalent among swine populations before 1998; however, since late August 1998, H3N2 subtypes have been isolated from pigs. As of 2004, H3N2 virus isolates in US swine and turkey stocks were triple reassortants, containing genes from human (HA, NA, and PB1), swine (NS, NP, and M), and avian (PB2 and PA) lineages.

Surveillance

Although there is no formal national surveillance system in the United States to determine what viruses are circulating in pigs, there is an informal surveillance network in the United States that is part of a world surveillance network.

Veterinary medical pathologist, Tracey McNamara, set up a national disease surveillance system in zoos because the zoos do active disease surveillance and many of the exotic animals housed there have broad susceptibilities. Many species fall below the radar of any federal agencies (including dogs, cats, pet prairie dogs, zoo animals, and urban wildlife), even though they may be important in the early detection of human disease outbreaks.

History

Swine influenza was first proposed to be a disease related to human influenza during the 1918 flu pandemic, when pigs became sick at the same time as humans. The first identification of an influenza virus as a cause of disease in pigs occurred about ten years later, in 1930. For the following 60 years, swine influenza strains were almost exclusively H1N1. Then, between 1997 and 2002, new strains of three different subtypes and five

different genotypes emerged as causes of influenza among pigs in North America. In 1997–1998, H3N2 strains emerged. These strains, which include genes derived by reassortment from human, swine and avian viruses, have become a major cause of swine influenza in North America. Reassortment between H1N1 and H3N2 produced H1N2. In 1999 in Canada, a strain of H4N6 crossed the species barrier from birds to pigs, but was contained on a single farm.

The H1N1 form of swine flu is one of the descendants of the strain that caused the 1918 flu pandemic. As well as persisting in pigs, the descendants of the 1918 virus have also circulated in humans through the 20th century, contributing to the normal seasonal epidemics of influenza. However, direct transmission from pigs to humans is rare, with only 12 recorded cases in the U.S. since 2005. Nevertheless, the retention of influenza strains in pigs after these strains have disappeared from the human population might make pigs a reservoir where influenza viruses could persist, later emerging to reinfect humans once human immunity to these strains has waned.

Swine flu has been reported numerous times as a zoonosis in humans, usually with limited distribution, rarely with a widespread distribution. Outbreaks in swine are common and cause significant economic losses in industry, primarily by causing stunting and extended time to market. For example, this disease costs the British meat industry about £65 million every year.

1918 pandemic in humans

The 1918 flu pandemic in humans was associated with H1N1 and influenza appearing in pigs; this may reflect a zoonosis either from swine to humans, or from humans to swine. Although it is not certain in which direction the virus was transferred, some evidence suggests that, in this case, pigs caught the disease from humans. For instance, swine influenza was only noted as a new disease of pigs in 1918, after the first large outbreaks of influenza amongst people. Although a recent phylogenetic analysis of more recent strains of influenza in humans, birds, and swine suggests that the 1918 outbreak in humans followed a reassortment event within a mammal, the exact origin of the 1918 strain remains elusive. It is estimated that anywhere from 50 to 100 million people were killed worldwide.

1976 U.S. outbreak

On February 5, 1976, in the United States an army recruit at Fort Dix said he felt tired and weak. He died the next day and four of his fellow soldiers were later hospitalized. Two weeks after his death, health officials announced that the cause of death was a new strain of swine flu. The strain, a variant of H1N1, is known as A/New Jersey/1976 (H1N1). It was detected only from January 19 to February 9 and did not spread beyond Fort Dix.

This new strain appeared to be closely related to the strain involved in the 1918 flu pandemic. Moreover, the ensuing increased surveillance uncovered another strain in

circulation in the U.S.: A/Victoria/75 (H3N2) spread simultaneously, also caused illness, and persisted until March. Alarmed public-health officials decided action must be taken to head off another major pandemic, and urged President Gerald Ford that every person in the U.S. be vaccinated for the disease.

The vaccination program was plagued by delays and public relations problems. On October 1, 1976, immunizations began and three senior citizens died soon after receiving their injections. This resulted in a media outcry that linked these deaths to the immunizations, despite the lack of any proof that the vaccine was the cause. According to science writer Patrick Di Justo, however, by the time the truth was known—that the deaths were not proven to be related to the vaccine—it was too late. "The government had long feared mass panic about swine flu—now they feared mass panic about the swine flu vaccinations." This became a strong setback to the program.

There were reports of Guillain-Barré syndrome, a paralyzing neuromuscular disorder, affecting some people who had received swine flu immunizations. Although if a link exists is still not clear, this syndrome may be a rare side-effect of influenza vaccines. As a result, Di Justo writes that "the public refused to trust a government-operated health program that killed old people and crippled young people." In total, 48,161,019 Americans, or just over 22% of the population, had been immunized by the time the National Influenza Immunization Program (NIIP) was effectively halted on December 16, 1976.

Overall, there were 1098 cases of Guillain-Barré Syndrome (GBS) recorded nationwide by CDC surveillance, 532 of which occurred after vaccination and 543 before vaccination. There are about one to two cases of GBS per 100,000 people every year, whether or not people have been vaccinated. The vaccination program seems to have increased this normal risk of developing GBS by about to one extra case per 100,000 vaccinations.

The CDC states that most studies on modern influenza vaccines have seen no link with GBS, Although one review gives an incidence of about one case per million vaccinations, a large study in China, reported in the NEJM covering close to 100 million doses of H1N1 flu vaccine found only eleven cases of Guillain-Barre syndrome, which is lower than the normal rate of the disease in China; "The risk-benefit ratio, which is what vaccines and everything in medicine is about, is overwhelmingly in favor of vaccination."

1988 zoonosis

In September 1988, a swine flu virus killed one woman and infected others. 32-year old Barbara Ann Wieners was eight months pregnant when she and her husband, Ed, became ill after visiting the hog barn at a county fair in Walworth County, Wisconsin. Barbara died eight days later, after developing pneumonia. The only pathogen identified was an H1N1 strain of swine influenza virus. Doctors were able to induce labor and deliver a healthy daughter before she died. Her husband recovered from his symptoms.

Influenza-like illness (ILI) was reportedly widespread among the pigs exhibited at the fair. Of the 25 swine exhibitors aged 9 to 19 at the fair, 19 tested positive for antibodies to SIV, but no serious illnesses were seen. The virus was able to spread between people, since 1-3 health care personnel who had cared for the pregnant woman developed mild influenza-like illnesses, and antibody tests suggested that they had been infected with swine flu. However, there was no community outbreak.

1998 US outbreak in swine

In 1998, swine flu was found in pigs in four U.S. states. Within a year, it had spread through pig populations across the United States. Scientists found that this virus had originated in pigs as a recombinant form of flu strains from birds and humans. This outbreak confirmed that pigs can serve as a crucible where novel influenza viruses emerge as a result of the reassortment of genes from different strains. Genetic components of these 1998 triple-hybrid stains would later form six out of the eight viral gene segments in the 2009 flu outbreak.

2007 Philippine outbreak in swine

On August 20, 2007, the Department of Agriculture officers investigated the outbreak (epizootic) of swine flu in Nueva Ecija and Central Luzon, Philippines. The mortality rate is less than 10% for swine flu, unless there are complications like hog cholera. On July 27, 2007, the Philippine National Meat Inspection Service (NMIS) raised a hog cholera "*red alert*" warning over Metro Manila and 5 regions of Luzon after the disease spread to backyard pig farms in Bulacan and Pampanga, even if these tested negative for the swine flu virus.

2010 Northern Ireland outbreak in swine

Since November 2010, there have been 14 reported deaths as a result of swine flu in Northern Ireland. The majority of the victims were reported to have pre-existing health conditions which had lowered the patients' immune system. This closely corresponds to the 19 patients that had died in the year prior due to swine flu where 18 of the 19 were determined to have lowered immune systems. Because of this, many mothers whom have just given birth are strongly encouraged to get a flu shot because their immune systems are vulnerable. Also, studies have shown that people between the ages of 15 and 44 have the highest rate of infection. Although most people now recover, having any conditions that lower ones immune system increases the risk of having the flu become potentially lethal. In Northern Ireland now, approximately 56% of all people under 65 that are entitled to the vaccine have gotten the shot and the outbreak is said to be under control.

Transmission

Transmission between pigs

Influenza is quite common in pigs, with about half of breeding pigs having been exposed to the virus in the US. Antibodies to the virus are also common in pigs in other countries.

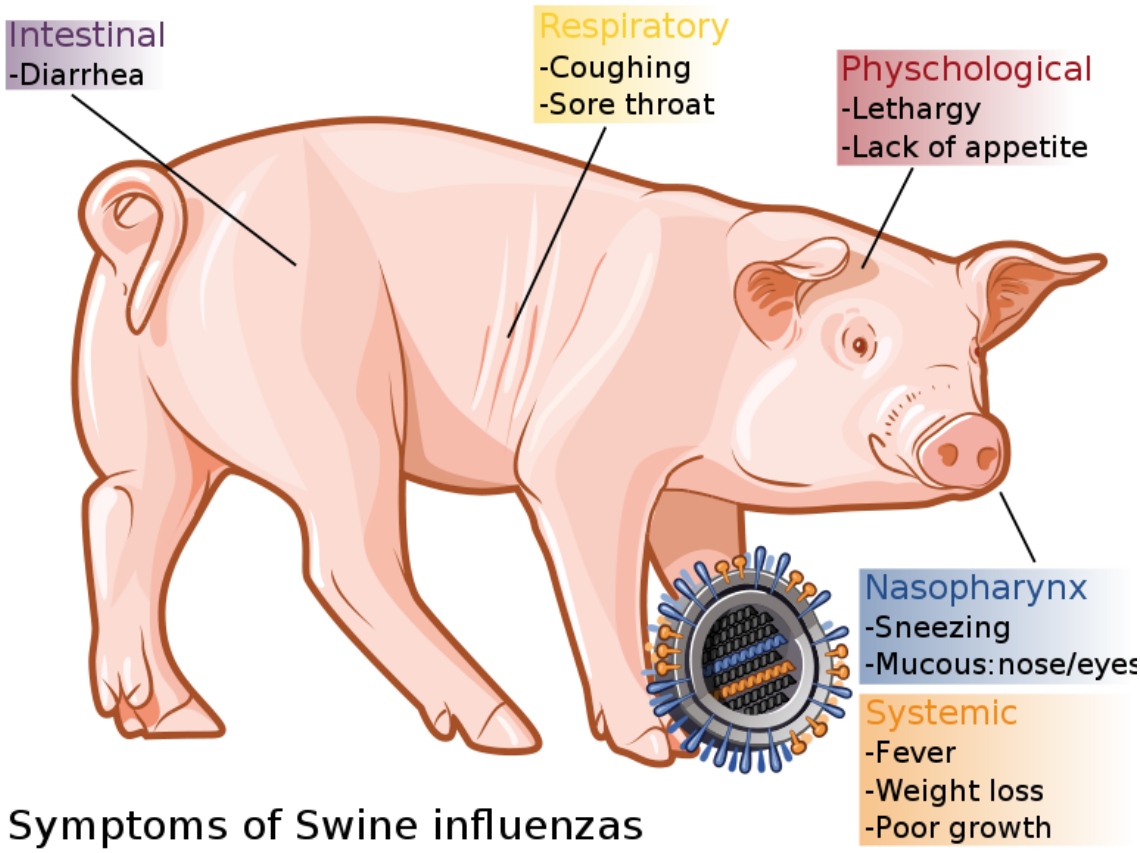
The main route of transmission is through direct contact between infected and uninfected animals. These close contacts are particularly common during animal transport. Intensive farming may also increase the risk of transmission, as the pigs are raised in very close proximity to each other. The direct transfer of the virus probably occurs either by pigs touching noses, or through dried mucus. Airborne transmission through the aerosols produced by pigs coughing or sneezing are also an important means of infection. The virus usually spreads quickly through a herd, infecting all the pigs within just a few days. Transmission may also occur through wild animals, such as wild boar, which can spread the disease between farms.

Transmission to humans

People who work with poultry and swine, especially people with intense exposures, are at increased risk of zoonotic infection with influenza virus endemic in these animals, and constitute a population of human hosts in which zoonosis and reassortment can co-occur. Vaccination of these workers against influenza and surveillance for new influenza strains among this population may therefore be an important public health measure. Transmission of influenza from swine to humans who work with swine was documented in a small surveillance study performed in 2004 at the University of Iowa. This study among others forms the basis of a recommendation that people whose jobs involve handling poultry and swine be the focus of increased public health surveillance. Other professions at particular risk of infection are veterinarians and meat processing workers, although the risk of infection for both of these groups is lower than that of farm workers.

Interaction with avian H5N1 in pigs

Pigs are unusual as they can be infected with influenza strains that usually infect three different species: pigs, birds and humans. This makes pigs a host where influenza viruses might exchange genes, producing new and dangerous strains. Avian influenza virus H3N2 is endemic in pigs in China and has been detected in pigs in Vietnam, increasing fears of the emergence of new variant strains. H3N2 evolved from H2N2 by antigenic shift. In August 2004, researchers in China found H5N1 in pigs.



Main symptoms of swine flu in swine.

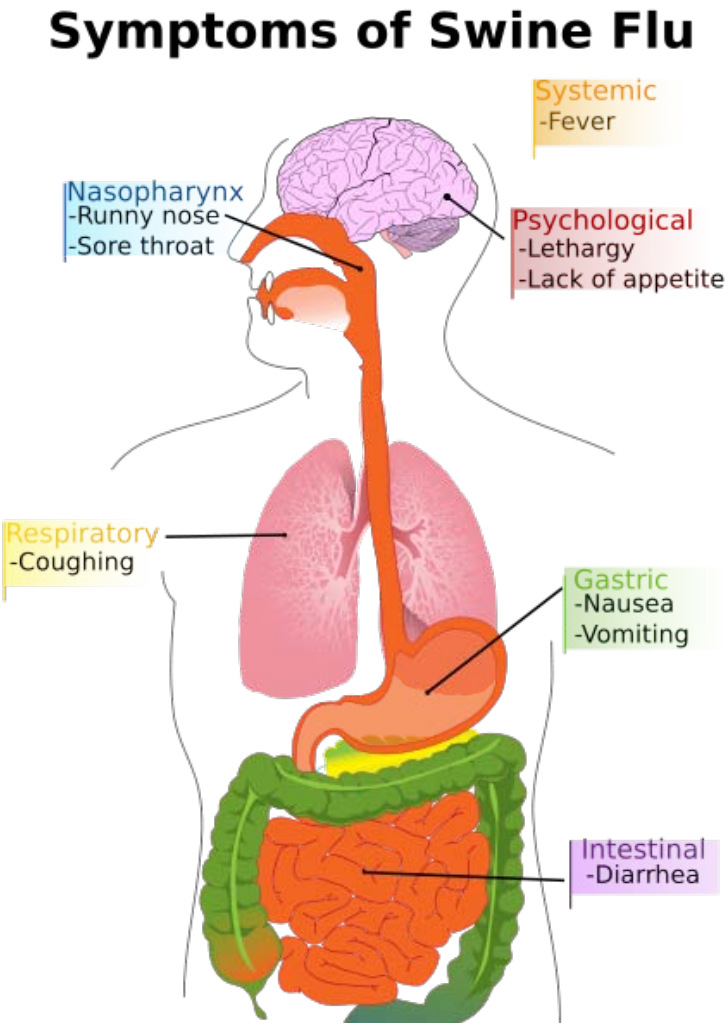
These H5N1 infections may be quite common: in a survey of 10 apparently healthy pigs housed near poultry farms in West Java, where avian flu had broken out, five of the pig samples contained the H5N1 virus. The Indonesian government has since found similar results in the same region. Additional tests of 150 pigs outside the area were negative.

Signs and symptoms

In swine

In pigs influenza infection produces fever, lethargy, sneezing, coughing, difficulty breathing and decreased appetite. In some cases the infection can cause abortion. Although mortality is usually low (around 1–4%), the virus can produce weight loss and poor growth, causing economic loss to farmers. Infected pigs can lose up to 12 pounds of body weight over a 3 to 4 week period.

In humans



Main symptoms of swine flu in humans

Direct transmission of a swine flu virus from pigs to humans is occasionally possible (called zoonotic swine flu). In all, 50 cases are known to have occurred since the first report in medical literature in 1958, which have resulted in a total of six deaths. Of these six people, one was pregnant, one had leukemia, one had Hodgkin disease and two were known to be previously healthy. Despite these apparently low numbers of infections, the true rate of infection may be higher, since most cases only cause a very mild disease, and will probably never be reported or diagnosed.

According to the Centers for Disease Control and Prevention (CDC), in humans the symptoms of the 2009 "swine flu" H1N1 virus are similar to those of influenza and of influenza-like illness in general. Symptoms include fever, cough, sore throat, body aches, headache, chills and fatigue. The 2009 outbreak has shown an increased percentage of patients reporting diarrhea and vomiting. The 2009 H1N1 virus is not zoonotic swine flu, as it is not transmitted from pigs to humans, but from person to person.

Because these symptoms are not specific to swine flu, a differential diagnosis of *probable* swine flu requires not only symptoms but also a high likelihood of swine flu due to the person's recent history. For example, during the 2009 swine flu outbreak in the United States, CDC advised physicians to "consider swine influenza infection in the differential diagnosis of patients with acute febrile respiratory illness who have either been in contact with persons with confirmed swine flu, or who were in one of the five U.S. states that have reported swine flu cases or in Mexico during the 7 days preceding their illness onset." A diagnosis of *confirmed* swine flu requires laboratory testing of a respiratory sample (a simple nose and throat swab).

The most common cause of death is respiratory failure. Other causes of death are pneumonia (leading to sepsis), high fever (leading to neurological problems), dehydration (from excessive vomiting and diarrhea), electrolyte imbalance and kidney failure. Fatalities are more likely in young children and the elderly.

Diagnosis

The CDC recommends real time RT-PCR as the method of choice for diagnosing H1N1. This method allows a specific diagnosis of novel influenza (H1N1) as opposed to seasonal influenza. Near-patient point of care tests are in development.

Prevention

Prevention of swine influenza has three components: prevention in swine, prevention of transmission to humans, and prevention of its spread among humans.

In swine

Methods of preventing the spread of influenza among swine include facility management, herd management, and vaccination (ATCvet code: QI09AA03). Because much of the illness and death associated with swine flu involves secondary infection by other pathogens, control strategies that rely on vaccination may be insufficient.

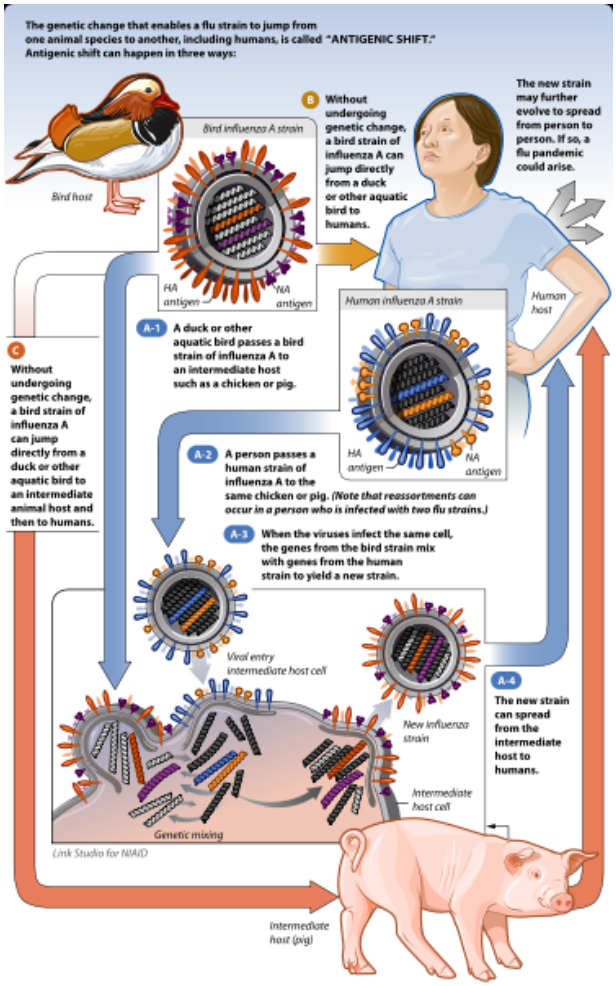
Control of swine influenza by vaccination has become more difficult in recent decades, as the evolution of the virus has resulted in inconsistent responses to traditional vaccines. Standard commercial swine flu vaccines are effective in controlling the infection when the virus strains match enough to have significant cross-protection, and custom (autogenous) vaccines made from the specific viruses isolated are created and used in the more difficult cases. Present vaccination strategies for SIV control and prevention in swine farms typically include the use of one of several bivalent SIV vaccines commercially available in the United States. Of the 97 recent H3N2 isolates examined, only 41 isolates had strong serologic cross-reactions with antiserum to three commercial SIV vaccines. Since the protective ability of influenza vaccines depends primarily on the closeness of the match between the vaccine virus and the epidemic virus, the presence of nonreactive H3N2 SIV variants suggests that current commercial vaccines might not effectively protect pigs from infection with a majority of H3N2 viruses. The United

States Department of Agriculture researchers say that while pig vaccination keeps pigs from getting sick, it does not block infection or shedding of the virus.

Facility management includes using disinfectants and ambient temperature to control virus in the environment. The virus is unlikely to survive outside living cells for more than two weeks, except in cold (but above freezing) conditions, and it is readily inactivated by disinfectants. Herd management includes not adding pigs carrying influenza to herds that have not been exposed to the virus. The virus survives in healthy carrier pigs for up to 3 months and can be recovered from them between outbreaks. Carrier pigs are usually responsible for the introduction of SIV into previously uninfected herds and countries, so new animals should be quarantined. After an outbreak, as immunity in exposed pigs wanes, new outbreaks of the same strain can occur.

In humans

Prevention of pig to human transmission



Swine can be infected by both avian and human influenza strains of influenza, and therefore are hosts where the antigenic shifts can occur that create new influenza strains.

The transmission from swine to human is believed to occur mainly in swine farms where farmers are in close contact with live pigs. Although strains of swine influenza are usually not able to infect humans this may occasionally happen, so farmers and veterinarians are encouraged to use a face mask when dealing with infected animals. The use of vaccines on swine to prevent their infection is a major method of limiting swine to human transmission. Risk factors that may contribute to swine-to-human transmission include smoking and, especially, not wearing gloves when working with sick animals—thereby increasing the likelihood of subsequent hand-to-eye, hand-to-nose or hand-to-mouth transmission.

Prevention of human to human transmission

Influenza spreads between humans when infected people cough or sneeze, then other people breathe in the virus or touch something with the virus on it and then touch their own face. "Avoid touching your eyes, nose or mouth. Germs spread this way." Swine flu cannot be spread by pork products, since the virus is not transmitted through food. The swine flu in humans is most contagious during the first five days of the illness although some people, most commonly children, can remain contagious for up to ten days. Diagnosis can be made by sending a specimen, collected during the first five days for analysis.

Recommendations to prevent spread of the virus among humans include using standard infection control against influenza. This includes frequent washing of hands with soap and water or with alcohol-based hand sanitizers, especially after being out in public. Chance of transmission is also reduced by disinfecting household surfaces, which can be done effectively with a diluted chlorine bleach solution.

Experts agree that hand-washing can help prevent viral infections, including ordinary influenza and the swine flu virus. Also not touching your eyes, nose or mouth with your hands helps to prevent the flu. Influenza can spread in coughs or sneezes, but an increasing body of evidence shows small droplets containing the virus can linger on tabletops, telephones and other surfaces and be transferred via the fingers to the eyes, nose or mouth. Alcohol-based gel or foam hand sanitizers work well to destroy viruses and bacteria. Anyone with flu-like symptoms such as a sudden fever, cough or muscle aches should stay away from work or public transportation and should contact a doctor for advice.

Social distancing is another tactic. It means staying away from other people who might be infected and can include avoiding large gatherings, spreading out a little at work, or perhaps staying home and lying low if an infection is spreading in a community. Public health and other responsible authorities have action plans which may request or require social distancing actions depending on the severity of the outbreak.

Vaccination

Vaccines are available for different kinds of swine flu. The U.S. Food and Drug Administration (FDA) approved the new swine flu vaccine for use in the United States on September 15, 2009. Studies by the National Institutes of Health (NIH), show that a single dose creates enough antibodies to protect against the virus within about 10 days.

Treatment

In swine

As swine influenza is rarely fatal to pigs, little treatment beyond rest and supportive care is required. Instead veterinary efforts are focused on preventing the spread of the virus throughout the farm, or to other farms. Vaccination and animal management techniques are most important in these efforts. Antibiotics are also used to treat this disease, which although they have no effect against the influenza virus, do help prevent bacterial pneumonia and other secondary infections in influenza-weakened herds.

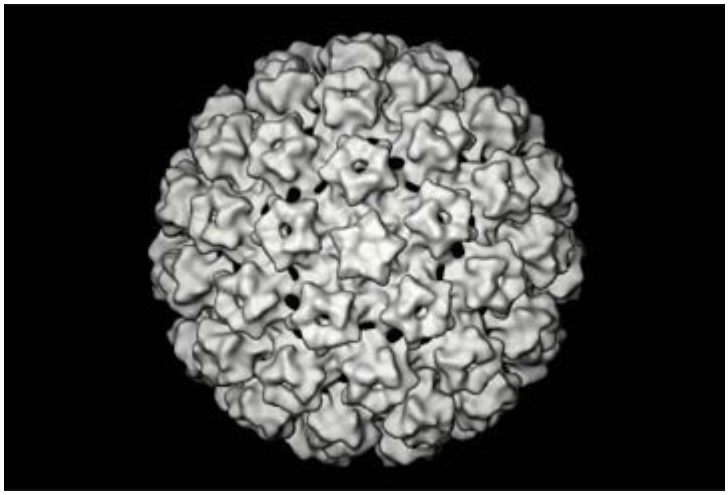
In humans

If a person becomes sick with swine flu, antiviral drugs can make the illness milder and make the patient feel better faster. They may also prevent serious flu complications. For treatment, antiviral drugs work best if started soon after getting sick (within 2 days of symptoms). Beside antivirals, supportive care at home or in hospital, focuses on controlling fevers, relieving pain and maintaining fluid balance, as well as identifying and treating any secondary infections or other medical problems. The U.S. Centers for Disease Control and Prevention recommends the use of Tamiflu (oseltamivir) or Relenza (zanamivir) for the treatment and/or prevention of infection with swine influenza viruses; however, the majority of people infected with the virus make a full recovery without requiring medical attention or antiviral drugs. The virus isolates in the 2009 outbreak have been found resistant to amantadine and rimantadine.

In the U.S., on April 27, 2009, the FDA issued Emergency Use Authorizations to make available Relenza and Tamiflu antiviral drugs to treat the swine influenza virus in cases for which they are currently unapproved. The agency issued these EUAs to allow treatment of patients younger than the current approval allows and to allow the widespread distribution of the drugs, including by non-licensed volunteers.

Chapter 7

Bovine Papillomavirus

Bovine papillomavirus

Bovine papillomavirus type 1 (3D reconstruction)

Virus classification	
Group:	Group I (dsDNA)
Order:	*Unranked*
Family:	*Papillomaviridae*
Genus:	*Deltapapillomavirus, Epsilonpapillomavirus, Xipapillomavirus*
Species:	*BPV-1* *BPV-2* *BPV-3* *BPV-4* *BPV-5* *BPV-6*

Bovine papillomavirus (BPV) is a group of DNA viruses of the family *Papillomaviridae* that are common in cattle. Infection causes warts (papillomas and fibropapillomas) of the skin and alimentary tract, and more rarely cancers of the alimentary tract and urinary

bladder. They are also thought to cause the skin tumour equine sarcoid in horses and donkeys.

BPV provides an excellent model for studying papillomavirus molecular biology, and also allows the dissection of the processes by which this group of viruses cause cancer.

Structure and genetic organisation

Like other papillomaviruses, BPVs are small non-enveloped viruses with an icosahedral capsid around 50–60 nm in diameter. The capsid is formed of the L1 and L2 structural proteins, with the L1 C-terminus exposed.

All BPVs have a circular double-stranded DNA genome of 7.3–8.0 kb. The genetic organisation of those BPVs which have been sequenced is broadly similar to other papillomaviruses. The open reading frames (ORFs) are all located on one strand, and are divided into early and late regions. The early region encodes nonstructural proteins E1 to E7. There are three viral oncoproteins, E5, E6 and E7; BPVs of the *Xipapillomavirus* group lack E6. The late region encodes structural proteins L1 and L2. There is also a non-coding long control region (LCR).

Types

Six types of BPV have been characterised, BPV-1 to BPV-6, which are divided into three broad subgroups.

- ***Deltapapillomavirus*** or **fibropapillomaviruses** (formerly known as subgroup A), including types 1 and 2, have a genome of around 7.9 kb. Similar papillomaviruses of ungulates (e.g. deer papillomavirus, European elk papillomavirus, ovine papillomavirus 1,2) are also found in this group. Like all members of the papillomavirus class, these viruses infect only keratinocytes (epithelial cells); however, unlike other papillomaviruses, they cause proliferation of both keratinocytes and fibroblasts, causing benign fibropapillomas involving both the epithelium and the underlying dermis. The specificity of the types differs:
    - BPV-1 infects paragenital areas, including penis, teats and udders
    - BPV-2 infects skin, alimentary canal and urinary bladder

- ***Xipapillomavirus*** or **epitheliotropic BPVs** (formerly known as subgroup B), including types 3, 4 and 6, have a smaller genome of around 7.3 kb and are unique among papillomaviruses in lacking the E6 oncoprotein. They infect keratinocytes (epithelial cells), causing pure papillomas involving only the epithelium. The specificity of the types differs:
    - BPV-3 infects skin
    - BPV-4 infects the upper alimentary tract
    - BPV-6 infects teats and udders

- ***Epsilonpapillomavirus*** has the single member BPV-5, with features intermediate between the other two groups. BPV-5 infects teats and udders, and can cause both pure papillomas and fibropapillomas.

A further thirteen putative BPVs have recently been identified; the novel viruses have yet to be assigned to subgroups.

Pathology

BPV is highly prevalent, with around 50% of cattle being estimated to bear lesions in the UK. Cutaneous warts are most common in younger animals (under 2 years) and usually spontaneously regress due to the animal's immune response without significant scarring. The duration of infection is very variable (from one month to over a year) and recurrence is possible.

Warts caused by the *Xipapillomavirus* group have a cauliflower-like appearance and can attain the size of a fist; most common on the head, neck and shoulders, they may also occur in other locations. Cutaneous fibropapillomas caused by *Deltapapillomavirus* group have a nodular appearance. Although unsightly, most skin warts rarely cause problems except in show animals. However, large warts may bleed, potentially leading to secondary infections, and florid warts of the teat can cause mastitis and interfere with suckling and milking. Fibropapillomas can be troublesome when present in the genital area, causing pain and sometimes loss of reproductive functions as well as interfering with calving. Chronically immunosuppressed animals may develop extensive papillomatosis in the upper gastrointestinal tract, which can cause difficulties with eating and breathing.

Multiple small facial warts

Warts contain large amounts of infectious virus which is relatively stable. Transmission between animals is common via, for example, fence posts or halters. Warts on the teats of lactating cows are readily transmitted to calves via abrasions. Contaminated tattooing or tagging equipment is another common source of infection.

Association with cancer

Cattle

BPV-4 causes squamous cell carcinomas of the alimentary tract, and BPV-1/2 causes carcinomas and haemangioendotheliomas of the urinary bladder, in both cases in animals that have fed on bracken (*Pteridium aquilinum*). Such cancers are common in locations where grazing land is infested with bracken, such as the western Scottish Highlands, southern Italy and the Nasampolai Valley in Kenya. Bracken contains several immunosuppressants and mutagens, including quercetin and ptaquiloside. Consumption of large quantities by cattle leads to an acute poisoning syndrome with symptoms of bone

marrow depletion, while at lower levels of long-term consumption it acts as a cancer cofactor. Carcinogenesis is a multistep process; tumours also contain activated Ras, as well as mutation or downregulation of the tumour suppressor genes *p53* in alimentary tract cancers and *fragile histidine tetrads* (*FHIT*) in urinary bladder cancers. Viral particles are not produced in either alimentary tract or urinary bladder tumours.

Cows feeding on bracken are at risk of developing BPV-associated cancers

These bracken-associated tumours might form a model for some types of human oesophageal cancer. Human papillomavirus DNA has been detected in around 18% of squamous cell carcinomas of the oesophagus, and there is an association between exposure to or consumption of bracken (which is used as a foodstuff and herbal remedy in South America, China, Japan, Korea and other countries) and risk of developing oesophageal cancer.

Other mammals

BPV-1 and BPV-2 can also induce sarcomas and fibrosarcomas in other mammals, including equids (equine sarcoid) and, experimentally, rabbits, hamsters and mice (and reviewed in). Viral particles are not produced during infection of other species and, unlike in tumours associated with human papillomavirus, the viral DNA is not integrated into the host genome.

Equine sarcoid, a naturally occurring skin tumour affecting horses, donkeys and mules, is associated with strains of BPV-1/2 which may be equine specific. The lesions can occur anywhere on the body, often multiply, with the limbs, thorax–abdomen, head and

paragenital areas being particularly commonly affected. The method of transmission is currently unclear; the involvement of face flies (*Musca autumnalis*) has been suggested, and transmission via contaminated tack is likely. The disease forms the only known example of natural cross-species infection by a papillomavirus. The involvement of BPV leads to hope that vaccination or antiviral therapy might be possible in the future for this common tumour.

Treatment, prophylaxis & prevention

Treatment is not usually required, as most warts eventually regress spontaneously. Surgical removal is possible but may lead to recurrence. Disinfection with formaldehyde of stalls, fence posts and other environmental virus reservoirs can prevent transmission.

Vaccination

Vaccines against BPV types 1, 2 and 4 have been developed by M. Saveria Campo and others.

- **Prophylactic vaccination** (*i.e.*, vaccination of wart-free animals to prevent infection) with whole virus (e.g. formalin-killed wart tissue suspension), virus-like particles (L1 or L1+L2), L1 protein or (for BPV-4) L2 protein confers long-lasting protection against challenge with the same BPV type, but is generally ineffective against existing warts. Protection appears to be mediated via type-specific neutralising antibodies. Vaccination of calves as early as 4–6 weeks might be necessary to prevent infection.

- **Therapeutic vaccination** (*i.e.*, vaccination of animals with existing warts) with BPV-4 E7 or BPV-2 L2 induces early regression of warts. Wart rejection involves a cell-mediated immune response, with infiltration of the site by large numbers of lymphocytes and macrophages.

These vaccine systems have served as models for the successful development of prophylactic vaccines against the human papillomavirus types associated with cervical and anal cancers. Both Gardasil (a quadrivalent prophylactic HPV vaccine licensed in 2006) and Cervarix (a bivalent prophylactic vaccine license in the EU in 2007 and USA in 2009) contain virus-like particles assembled from L1 protein, an approach successful against BPV, and both vaccines induce sustained immunity. Various therapeutic HPV vaccines based on E6, E7 and L2 are currently in early-stage clinical trials.

Chapter 8

Diabetes in Cats

Diabetes mellitus strikes 1 in 400 cats, though recent veterinary studies note that it is becoming more common lately in cats. Symptoms in cats are similar to those in humans. Diabetes in cats occurs less frequently than in dogs. 80-95% of diabetic cats experience something similar to type-2 diabetes. but are generally severely insulin-dependent by the time symptoms are diagnosed. The condition is definitely treatable, and need not shorten the animal's life span or life quality. In type-2 cats, prompt effective treatment can even lead to diabetic remission, in which the cat no longer needs injected insulin. Untreated, the condition leads to increasingly weak legs in cats, and eventually malnutrition, ketoacidosis and/or dehydration, and death.

Symptoms

Cats will generally show a gradual onset of the disease over a few weeks, and it may escape notice for a while. The condition is unusual in cats less than seven years old. The first obvious symptoms are a sudden weight loss (occasionally gain), accompanied by excessive drinking and urination; for example, cats can appear to develop an obsession with water and lurk around faucets or water bowls. Appetite is suddenly either ravenous (up to three-times normal) or absent. In cats the back legs may become weak and the gait may become stilted or wobbly (peripheral neuropathy). A quick test at this point can be done using urine keto/glucose strips (the same as used on the Atkins diet) with the animal. If the keto/glucose strips show glucose in the urine, diabetes is indicated. If a strip shows ketones in the urine, the animal should be brought to an emergency clinic right away. Testing can also be performed with a home glucose meter by obtaining a blood sample with a lancet via an ear prick or paw prick.

Owners should watch for noticeable thinning of the skin and apparent fragility: these are also serious and indicate that the animal is metabolizing (breaking down) its own body fat and muscle to survive. Lethargy or limpness, and acetone-smelling breath are acute symptoms indicating likely ketoacidosis and/or dehydration and demand emergency care within hours.

Treatment

Diabetes can be treated but is life-threatening if left alone. Early diagnosis and treatment by a qualified veterinarian can help, not only in preventing nerve damage, but in some cases, in cats, can even lead to remission. Cats usually seem to do best with long-lasting insulins and low carbohydrate diets.

Diet

Diet is a critical component of treatment, and is in many cases effective on its own. For example, a recent mini-study showed that many diabetic cats stopped needing insulin after changing to a low carbohydrate diet. The rationale is that a low carbohydrate diet reduces the amount of insulin needed and keeps the variation in blood sugar low and easier to predict. Also, fats and proteins are turned into blood glucose much more slowly and evenly than carbohydrates, reducing blood-sugar highs right after mealtimes.

Latest veterinary good practice is to recommend a low carbohydrate diet for cats rather than the formerly-recommended high-fiber diet.

It's now becoming clear that lower carbohydrate diets will significantly lower insulin requirements for diabetic cats. Carbohydrate levels are highest in dry cat foods (even the expensive "prescription" types) so cats are best off usually with a low carbohydrate healthy canned diet. Some prescription canned foods made for diabetic cats are effective, but some ordinary ones work just as well. Between 3 and 9% calories from carbohydrates seems to be optimal. You can use the cat food calculator to determine the carbohydrates and calories in any cat food, though numbers in American brands are given as minimums and maximums rather than actual estimates, and may be very inaccurate. If in the US, try these commercial food contents lists which are kept up-to-date with actual manufacturer's as-fed content amounts. Numbers in most other countries are "as fed" rather than "guaranteed" values. "As fed" values are generally more accurate and you can use them directly in the calculator.

Pills

Oral medications like Glipizide that stimulate the pancreas promoting insulin release, (or in some cases, reduce glucose production) are less and less used in cats, and these drugs may be completely ineffective if the pancreas is not working. Worse, these drugs have been shown in some studies to damage the pancreas further, reducing the chances of remission for cats. They have also been shown to cause liver damage. Many owners are reluctant to switch from pills to insulin injections, but the fear is unjustified; the difference in cost and convenience is minor, (most cats are easier to inject than to pill) and injections are more effective in almost all cases.

Insulin injections

Humans with Type-1 diabetes are often treated with a "basal plus bolus" method, where a long-acting insulin is injected once or twice daily to provide a "basal" insulin level, then shorter-acting insulin is used just before mealtimes.

For cats, a "basal" method is usually employed instead—a single slow-acting dose, twice daily, along with a very low carbohydrate diet, attempts to keep the blood sugar within a recommended range for the entire day. In this case it's important for the pet to avoid large meals or high-carbohydrate food, since they can seriously affect the blood sugar. (Meals may also be timed to coincide with peak insulin activity.) Once-daily doses are not recommended, since insulin usually metabolizes faster in cats than in humans or dogs or humans; an insulin brand that lasts 24 hours in people may only be effective for about 12 in a cat.

Cats and dogs may be treated with animal insulins (pork-based is most similar to a dog's natural insulin, beef-based for a cat), or with human synthetic insulins. The best choice of insulin brand and type varies between pets and may require some experimentation. One of the popular human synthetic insulins, Humulin N /Novolin N/ NPH, is reasonable for dogs, but is usually a poor choice for cats, since cats' metabolize (most) insulin about twice as fast. The Lente and Ultralente versions were therefore very popular for feline use until summer 2005, when Eli Lilly and Novo Nordisk both discontinued them.

Until the early 1990s, the most recommended type for pets was beef/pork-derived PZI, but that type was phased out over the 1990s and is now difficult to find in many countries. There are sources in the US and UK, and many vets are now starting to recommend them again for pets, but they have been discontinued by most manufacturers as of 2007-2008. A new synthetic PZI analogue called ProZinc is now available.

Caninsulin, known in the USA as Vetsulin, made by Intervet is a brand of pork-based insulin, which is approved for dogs and cats, and is available both through veterinarians and pharmacies with a veterinarian's prescription, depending on the country. Note, though, that Vetsulin was recently recalled due to inconsistent strength and is no longer used in the USA. According to the manufacturer's website, this insulin's action profile is long and flat in dogs, but in cats very similar to that of NPH insulins, and lowers blood sugar quickly, but for only about 6–8 hours.

Two new ultra-slow time-release synthetic human insulins became available in 2004 and 2005 for improving basal stability, generically known as Insulin Detemir (Levemir) and Insulin Glargine (Lantus). Studies at the University of Queensland, Brisbane, Australia have had remarkable results with Insulin Glargine in cats. Followup research Roomp and Rand, Vet Intern Med 2008, 22 (3):791 shows that Levemir can be used with a similar protocol and that either insulin, on this protocol, leads in most uncomplicated feline cases to remission, with the most success being in cats who start on these protocols as soon as possible after diagnosis.

Neuropathy

The weak legs syndrome found in many diabetic cats is a form of neuropathy, in particular caused by damage to the myelin sheath of the peripheral nerves caused by glucose toxicity and cell starvation, which are caused by chronic hyperglycemia. (There are other conditions that can cause weak legs too, consult your vet before assuming neuropathy.) Most common in cats, the back legs become weaker until the cat displays "Plantigrade stance", standing on its hocks instead of on its toes as usual. The cat may also have trouble walking and jumping, and may need to sit down after a few steps. Some recommend a specific form of vitamin B_{12} called methylcobalamin to heal the nerve damage. Neuropathy sometimes heals on its own within 6–10 weeks once blood sugar is regulated, but anecdotal evidence points to a faster and more complete recovery with methylcobalamin supplements.

Dosage and regulation

Cats may in some cases have their mealtimes strictly scheduled and planned to match with injection times, especially when on insulins with a pronounced peak action like Caninsulin/Vetsulin or Humulin N. In other cases where the pet free-feeds and normally eats little bits all day or night, it may be best to remain on this schedule and try to use a very slow-acting insulin to keep a constant level of blood glucose. Consult your veterinarian. Note that some veterinarians still use the outdated recommendation of using Humulin "N" or NPH insulin for cats. This insulin is too fast-acting for most cats (though fine for dogs and humans). Cats metabolize insulin about twice as fast as humans, and the often-effective slower-acting Lente and Ultralente (Humulin L and Humulin U) insulins are being discontinued (as of 2005), so most cats are now using either the veterinary PZI insulins, or the new full-day analogs glargine (Lantus) and detemir (Levemir).

The goal at first is to "regulate" the pet's blood glucose, which may take a few weeks. This process is basically the same as in type-1 diabetic humans. The goal is to keep the blood glucose values in a comfortable range for the pet during the whole day, or most of it.

The most successful documented method is **Tight Regulation with Lantus or Levemir**.

- Typical obstacles to regulation:
    - Chronic overdose masked by Somogyi: A dose that is too high can easily cause a Somogyi rebound, which can look like a need for more insulin. This condition can continue for days or weeks, and it's very hard on the cat's metabolism.
    - High-carb cat food: Many commercial foods (especially "Light" foods) are extremely high in cereals and therefore carbohydrates. The extra carbohydrates will keep the cat's blood sugar high, and if you're free-feeding may also make the blood sugar curve over the course of the day

volatile and unpredictable. In general, canned foods are lower in carbohydrates than dry ones, and canned "kitten" foods lower still. Recent studies show that cats' diabetes can be better regulated and even sometimes cured with a low carbohydrate diet. If switching to a lower-carbohydrate food, do it gradually and lower the insulin dosage appropriately, using home blood-glucose testing several times daily to be sure your insulin dose is being adjusted properly. If your cat is on a special diet for pancreatitis, chronic renal failure, or any other condition, consult your vet for the appropriate diet for that condition plus diabetes.

- Wrong insulin: Different brands and types of insulin have idiosyncratic effects on different cats. If you've settled on a dose that seems to keep the pet's blood sugar within range at peak effectiveness, but the sugar readings remain dangerously high at shot times, the insulin may not be lasting long enough for your pet, or may not be the best choice. Testing blood sugar more frequently (for example, at 6, 8, 10 and 12 hours after shot) will determine if the insulin is lasting long enough. Switching to a slower-acting or a better-tolerated insulin for that pet, and lowering the dose initially to be safe, may be the next step.

Blood sugar guidelines

Absolute numbers vary between pets, and with meter calibrations. Glucometers made for humans are generally accurate using feline blood except when reading lower ranges of blood glucose (<80 mg/dl—4.44 mmol/L). It is at this point where the size difference in human vs animal red blood cells can create inaccurate readings. The numbers below are **as shown on a typical home glucometer** while hometesting blood glucose, not necessarily the more accurate numbers a vet would see (though many vets use meters similar to those used in hometesting). For general guidelines only, the levels to watch are approximately:

mmol/L	mg/dL(US)	
<2.2	**<40**	**Readings below this level are usually considered hypoglycemic when giving insulin, even if you see no symptoms of it. Treat immediately**
2.2-7.5	**40-130**	Non-diabetic range (usually unsafe to aim for when on insulin, unless your control is very good). These numbers, when not giving insulin, are very good news.
3.38-6.88	**61-124**	This is an average non-diabetic cat's level, but leaves little margin of safety for a diabetic on insulin. Don't aim for this range, but don't panic if you see it, either. If the number is not falling, it's healthy.
5	**90**	A commonly cited minimum safe value for the lowest blood sugar of the day

5.5-10	**100-180**	Commonly used target range for diabetics, for as much of the time as possible.
7.8	**140**	According to the American Association of Clinical Endocrinologists (AACE), threshold above which organ and pancreatic damage may begin.
<13.33-15	**240-270**	**Feline Renal threshold for glucose, when excess glucose from the kidneys spills into the urine and roughly when the cat begins to show diabetic symptoms.**
16.7	**300**	Approximate maximum safe value for the highest blood sugar of the day, in cats, to avoid neuropathy and complications. Some cats can go on long-term at this level or higher, but there will be side effects eventually. Check for ketones.
>20	**>360**	**Check for ketones frequently, be sure you are giving insulin. Cats are much more resilient than dogs or humans at these high levels; nevertheless, the blood sugar should be lowered. The cat can feel any of numerous ill effects both short and long-term.**

Detecting and avoiding chronic Somogyi rebound

Oddly, too much insulin may result in increase of blood glucose (BG). This "Somogyi effect" is often noted by pet owners who monitor their pets' blood glucose at home.

The reason: anytime the glucose level drops too far or too fast, the animal's organism may defensively dump glucose (converted from glycogen in the liver), as well as hormones epinephrine and cortisol, into the bloodstream. (If these are insufficient, hypoglycemia ensues!) The glycogen raises the blood glucose, the other two may make the pet insulin-resistant for a time. This phenomenon was first documented by a Dr. Michael Somogyi.

Even when raising the insulin dose slowly and carefully, it's possible to pass the correct dose and go on to an overdose. (A typical case is increasing bidaily dosage from 1 unit to 2, passing a correct dose of 1.5 units.) This may produce a rebound—a swift jump in blood glucose up from a dangerously low reading, to beyond the previous pre-shot level. Always consult your veterinarian when changing your pet's insulin, and consider smaller dose changes.

Hypoglycemic episode

An acute hypoglycemic episode (very low blood sugar) can happen even if you are careful, since pets' insulin requirements sometimes change without warning. The symptoms are depression/lethargy, confusion/dizziness, loss of excretory/bladder control, vomiting, and then loss of consciousness and/or seizures. As soon as possible, administer honey or corn syrup by rubbing it on the gums (even if unconscious, but not if in

seizures), and rush it to the vet. Carry more honey or corn syrup with you on the way and keep rubbing it on the gums, where it can be absorbed—it could save the pet's life. Every minute without blood sugar causes brain damage. (Some recommend administering syrup anally if the animal is in seizures!)

If the pet has hypoglycemia according to the blood glucose meter (<2.2 mmol/L or 40 mg/dL), but no symptoms, give treats or food if possible. If they won't eat, try putting food in their mouth. If that doesn't work, administer some honey followed by food or cat treats, and continue to do so until the blood glucose is rising, and the latest insulin shot's peak action is past.

Sometimes a mild hypoglycemic episode will go unnoticed, or leave evidence such as an "accident" where kitty fails to make it to the litterbox. In these cases the blood sugar will probably appear paradoxically *high* upon the next test hours later, since the pet's body will react to the low blood sugar by stimulating the liver to release stored glycogen. This condition is known as ***Somogyi rebound***. Consult your vet.

Diabetic Ketoacidosis

Too little insulin over time can cause tissue starvation (as glucose can't reach the brain or body). In combination with dehydration, fasting, infection, or other body stresses, this can turn over a few hours into Diabetic Ketoacidosis, a medical emergency with a high fatality rate, that cannot be treated at home. Many undiagnosed diabetic cats first come to the vet in this state, since they haven't been receiving insulin. Symptoms include lethargy, acetone or fruity smell on breath, shortness of breath, high blood sugar, huge thirst drive. Emergency care includes lots of water, some insulin, and an immediate trip to the all-night emergency vet.

Remission

It is in most cases possible to induce remission (a temporary or permanent freedom from insulin-dependence) in type-2 diabetic cats. This appears to be unique to cats. There is growing agreement among experts that a combination of low carbohydrate healthy diet, long-lasting insulin, and well-chosen dosage plans can in many cases partially heal a damaged pancreas and allow the cat's blood sugar to be controlled entirely by diet thereafter. (A low carbohydrate diet is usually required for the remainder of the cat's life.)

Remission is a realistic goal for treatment of type-2 diabetic cats (that is, 80-95% of all diabetic cats) who are properly regulated quickly. Chances of success are highest in the first few months after initial diagnosis, due to ongoing damage from glucose toxicity caused by hyperglycemia. This limited time window is a good reason to start with low carbohydrate diet and very slow-acting insulins, the most successful known combination, right away.

An explanation can be pieced together from recent studies in which diabetes in cats is perpetuated, if not always caused, by a combination of glucose toxicity and amyloidosis,

such that the insulin-producing islets of the pancreas become clogged with amyloid deposits. Cats may present with type-2 (insulin-resistant) diabetes, at least at first, but hyperglycemia and amyloidosis, left untreated, will damage the pancreas over time and progress to insulin-dependent diabetes.

If blood sugar is well-controlled over a period of 2–3 months, glucose toxicity and amyloidosis cease to attack newly generated tissue, and many cats are able to regenerate some of the damaged pancreatic beta cells and slowly resume insulin production.

Note that Glipizide and similar oral diabetic medicines designed for type-2 diabetic humans have been shown to increase amyloid production and amyloidosis, and therefore likely reduce likelihood of remission.

Chapter 9

Rabbit Haemorrhagic Disease

Rabbit haemorrhagic disease	
Scientific classification	
Kingdom:	Viruses
Family:	Caliciviridae
Genus:	***Lagovirus***
Species:	***Rabbit haemorrhagic disease virus***

Rabbit haemorrhagic disease (**RHD**), also known as **rabbit calicivirus disease** (**RCD**) or **viral haemorrhagic disease** (**VHD**), is a highly infectious and often fatal disease that affects wild and domestic rabbits of the species *Oryctolagus cuniculus*. The infectious agent responsible for the disease is rabbit haemorrhagic disease virus (RHDV), or rabbit calicivirus (RCV), genus *Lagovirus* of the family Caliciviridae. The virus infects only rabbits, and has been used in some countries to control rabbit populations.

Two vaccines are available against VHD in the UK. One is named Cylap and made by Fort Dodge Animal Health. The other is Lapinject and is made by CEVA Animal Health Ltd. Both last 12 months and contain inactivated strains of VHD.

History

RHD first appeared in 1984 in the People's Republic of China. Scientists cannot pinpoint its exact origins; however, it is believed the disease emerged from a virulent calicivirus spreading asymptomatically in European rabbit populations, particularly in the German Democratic Republic. The Chinese outbreak was spread by the angora rabbit, which had originated in Europe. Fourteen million domesticated rabbits died within nine months in the outbreak.

In 1984 the virus that caused the disease was identified. The virus spread westward and reached Europe in 1988. The virus has since appeared in Mexico, Cuba, Australia, New Zealand and the USA. In 1992, the United Kingdom reported its first case of RHD in domestic show rabbits. By the late 1990s, RHD stretched to forty countries and had become endemic in wild rabbit populations in Europe, Australia, New Zealand and Cuba. In Europe there was a rapid increase in research into RHD, due to the importance of the commercial breeding of rabbits for meat and fur production.

The first reported case in the United States was in Iowa on March 9, 2000. The affected population included Palominos and California Whites. By April 6, 25 of the 27 affected rabbits had died as a result of RHD. In order to contain the disease, the remaining two rabbits were euthanized. No new introductions of rabbits were placed on the farm for two years after the discovery of RHD and August 1999 was the last time rabbits left and/or returned to the farm. The United States experienced other outbreaks of RHD in 2001 (Utah, Illinois, New York) and 2005 (Indiana).

World geographic distribution

Asia

Within a few months of RHD being reported in China in 1984, the disease was widely seen in many commercial rabbitries and had reached the Republic of Korea. RHD has also been reported in India and the Middle East.

Americas (North and South)

Since 1993, RHD has been endemic in Cuba; it is also believed to be thriving in Bolivia. From 1988 to 1992 Mexico dealt with an RHD endemic in domestic rabbits.

After outbreaks of RHD in 2000, 2001, and 2005 in domesticated rabbits, the United States has eradicated RHD from its rabbit populations. The native species, cottontails *(Sylvilagus floridanus)*, black-tailed jackrabbits *(Lepus californicus)* and volcano rabbits *(Romerolagus diazzi)* seem not to be susceptible to the virus.

Australia

In 1991 the virus that caused RHD was imported to Australia under strict quarantine conditions to research the safety and usefulness of the virus if it was used as a biological control agent against Australia and New Zealand's rabbit pest problem. Testing of the virus was undertaken on Wardang Island in Spencer Gulf off the coast of the Yorke Peninsula, South Australia. In 1995 the virus escaped quarantine and subsequently killed 10 million rabbits within 8 weeks of its release. The disease is currently epidemic throughout Australia and New Zealand. When Calicivirus was first released it wiped out 90% of the rabbits in Australia.

New Zealand

In July 1997, after considering over 800 public submissions, the New Zealand Ministry of Health decided not to allow RHDV to be imported into New Zealand to control rabbit populations. This was backed up in an early August review of the decision by the Director-General of Agriculture. By late August, it was confirmed that RHDV had been deliberately introduced to the Cromwell area of the South Island.

An unsuccessful attempt was made by New Zealand officials to control the spread of the disease. It was, however, being intentionally spread, and several farmers (notably in the Mackenzie Basin area) admitted to processing rabbits that had died from the disease in kitchen blenders for further spreading.

Had the disease been introduced at a better time, there would have been a more effective control of the population. Unfortunately, it was released after breeding had commenced for the season, and rabbits under 2 weeks old at the time of the introduction were immune to the disease. These young rabbits were therefore able to continue to grow and breed back up. Ten years on, rabbit populations (in the Mackenzie Basin in particular) are beginning to reach near plague proportions once again though thay have not yet returned to pre RCD levels.

Europe

RHD is endemic throughout most of Europe. Italy's first case of RHD was recorded in 1986 and Spain's in 1988. France, Belgium (June) and Scandinavia followed in 1990. Within a few years of RHD's first appearance in Europe it had caused the largest mortality in domestic and wild rabbits in Germany, Austria, Spain and Italy. Spain was the worst affected by RHD.

When the United Kingdom's first case of RHD in 1992 was discovered, the disease was transmitted into the wild by domesticated pet rabbits. Sources vary in the number of confirmed cases of RHD; there were 9 known outbreaks in 1994, 32 cases but some sources believe there were as many as 512 cases of RHD in 1995, and around 30 RHD cases in 1996 throughout Scotland, England and Wales.

Transmission

Transmission of RHD occurs by direct contact with an infected animal and fomites. Rabbits acquire RHD through oral, nasal or conjunctival pathways. Urine, faeces and respiratory secretions may also shed the virus. The virus may also be carried by the wind. Carriers of the virus may remain infectious for up to a month depending on climate conditions; however, the virus has been known to persist for as little as 2 days and as long as 215 days. An infected carcass or hairs from an infected animal may also transmit RHD. Fomites such as clothing, contaminated food, cages, bedding, feeders and water will also harbour the virus. Even though the virus cannot reproduce in other mammals, predators and scavengers such as foxes, ferrets and some birds can excrete the virus

through their faeces after ingesting an infected rabbit carcass. Flies, rabbit fleas, and mosquitoes can also spread the virus between rabbits.

Climate appears to play a crucial role in the transmission of RHD. In normal conditions, most outbreaks of RHD occur in winter or spring. High temperatures in late spring and summer will considerably reduce the spreadof the virus. RHD will also be more prevalent in dry and semi-dry areas than in areas that are relatively cool and humid.

Signs

RHD primarily infects only adult rabbits. In fact, research has shown that rabbits younger than 8 weeks of age are resistant to the virus. The incubation period for the RHD virus is between 1 to 3 days, with death following 1 to 2 days after the infection. There is a wide range of RHD symptoms. Most rabbits will show no signs of external symptoms of RHD.

Symptomatic cases of RHD will display fever, squeals, and often coma leading to death within 12 to 36 hours. In less severe cases, rabbits may display uneasiness, excitement, anorexia, swollen eyelids, paralysis, ocular haemorrhages, and paddling. Convulsions may be seen as well. A fatal bloody discharge from the nose as been exhibited along with blood-stained cage floors, though these symptoms may have occurred after death. Rabbits who have recovered from the less severe symptoms usually develop severe jaundice with weight loss and lethargy. Diarrhoea, constipation and abdominal cramping are then exhibited right before death a few weeks later.

Research conducted at the CSIRO Australian Animal Health Laboratory claimed that rabbits did not show any outward signs of pain or suffering as a result of having RHD. The researchers further claimed that most infected rabbits became quieter and died quietly with no pain or suffering. However, the research did not in anyway prove or disprove that the rabbits did not experience severe and prolonged pain, only, that based on the subjective observation of their researchers, that the rabbits CSIRO infected grew quieter before death. It should be noted that CSIRO was actively researching RHD to use as an agent to help exterminate selected rabbit populations in Australia.

RHD causes rapid development of blood clot formation in major organs such as the heart, lungs and kidneys. The clots block blood vessels causing heart and respiratory failure. An infected rabbit that has died from RHD will often have its legs straight out and head over its neck as if trying to catch its breath.

Diagnosis

RHD may be indicated when several animals in the herd die after experiencing a fever and lethargy. Differential diagnosis includes pasteurellosis, myxomatosis, poisoning, heat exhaustion, and *E. coli* or *Clostridium perfringens* type E enterotoxemia.

Rabbits that die from RHD are usually in good outward state. However, the most frequent post-mortem lesions are hepatic necrosis and splenomegaly. The liver of RHD rabbits

may have a fine reticular pattern of necrosis outline each lobule and maybe yellow, gray or pale in colour. The liver is also usually friable and swollen. The spleen will be black in colour and also swollen with rounded edges, while the kidneys are dark brown in colour. Haemorrhages will also be seen many other organs and tissues. The trachea may present a foamy, bloody mucous. Enteritis of the small intestine and swollen meninges may also occur.

Laboratory tests such as reverse transcription polymerase chain reaction (RT-PCR), Western blotting, negative-staining immunoelectron microscopy and ELISAs may be performed on samples from the liver, blood, spleen or other organs.

Morbidity, mortality, and immunity

RHD is extremely hard to locate in the wild since about 75% of rabbits with RHD will die in their burrows underground. Due to this difficulty, the morbidity and mortality estimates for RHD have a broad range. The morbidity rate ranges from 30% to 100% and the mortality rate from 40% to 100%; however, the typical mortality rate is usually around 90%.{

In the wild, outbreaks in rabbits vary depending on the season, breeding cycles and geographical location. Some areas will see a high morbidity and mortality among its rabbit populations followed by calmer periods.

Maternal antibodies such as immunoglobulin G (IgG), which are readily transmitted to the young across the placenta, may explain why very young rabbits are resistant to RHD. Some scientists also believe that the immature immune system of a young rabbit cannot produce the number of chemicals needed to initiate clotting in order to kill. Rabbits may develop immunity against other strains of the RHD virus, while others may endure persistent infections. The immunity does not survive through the next generation, leaving open the possibility of further outbreaks in the population.

A population of rabbits on the San Juan Islands, Washington, USA appears to be immune to RHD. The cottontail rabbit and jackrabbit are not affected by the disease; nor are humans or other mammals.

Control

Countries that are uninfected by RHD may place restrictions on importation from endemic countries. According to the Merck/Merial Manual For Pet Health, Home Edition, 2007, RHD is a reportable disease in the United States. If a diagnosis is made by a veterinarian, a notification to the "appropriate government authorities" must be made.

Because of the highly infectious nature of the disease, strict quarantine is necessary when outbreaks occur. Depopulation, disinfection, surveillance and quarantines are the only way to properly and effectively eradicate the disease. Good disinfectants include 10% sodium hydroxide, 1-2% formalin, 2% One-Stroke Environ, and 10% household bleach.

The RHD virus is resistant to ethers or chloroform. Deceased rabbits must be removed immediately and discarded in a safe manner. Surviving rabbits should be quarantined or humanely euthanized. Test rabbits may be used to monitor the virus on vaccinated farms.

An effective, safe "killed" vaccine (ATCvet code: QI08AA01) has been developed in various countries, particularly Spain, and is believed to give protection for 5 to 15 months; however, vaccine manufacturers recommend a booster be administered annually. The vaccine is also recommended in areas where the disease is widespread and eradication efforts have been made.

Use as biological control agent

The European rabbit is the second most serious pest in New Zealand. Rabbits compete with livestock for grazing pasture, kill trees, shrubs, and have contributed to the extinction of some native plants. Consequently, rabbits contribute to soil erosion by eliminating the protective vegetation and disturb the soil by burrowing.

The estimated combined cost of control and production losses in New Zealand as a result of rabbits is about $23 million annually. This figure is only a small portion of the damage caused by rabbits.

Control measures used against rabbits in New Zealand include poisoning, shooting, ripping, blasting, releasing predators, and fencing.

After the safety of RHD was confirmed by laboratory research, RHD was approved for release as a biological control agent in New Zealand. RHD is safe because it infects rabbits, but not other animals or humans. It is also safe to eat the meat of infected RHD rabbits. Virus mutation is not a concern; many years of research show no evidence that the virus has changed to affect any other species other than European rabbit.

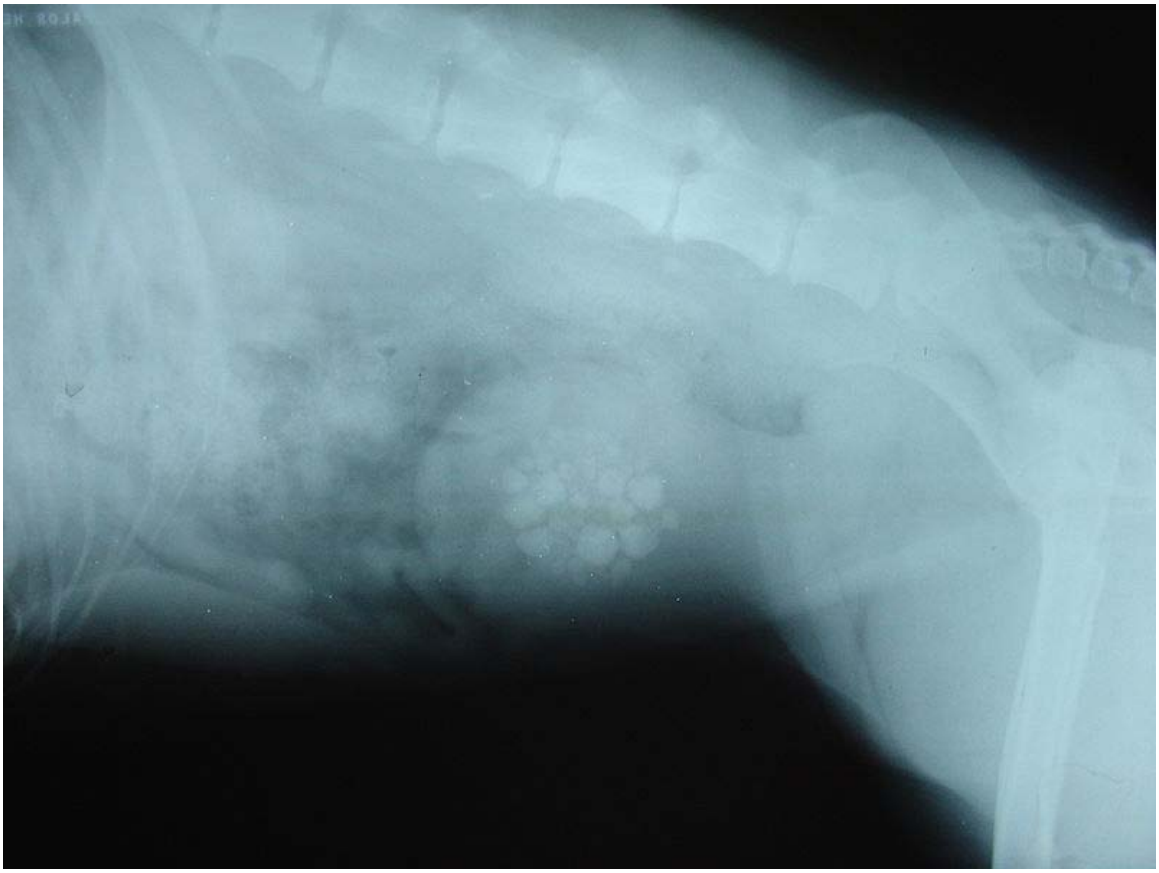
However, it is possible that widespread rabbit deaths might cause predators to prey upon other food sources, such as endangered or rare native species. With proper vaccination plans, the safety of domesticated rabbits should not be a concern.

Chapter 10

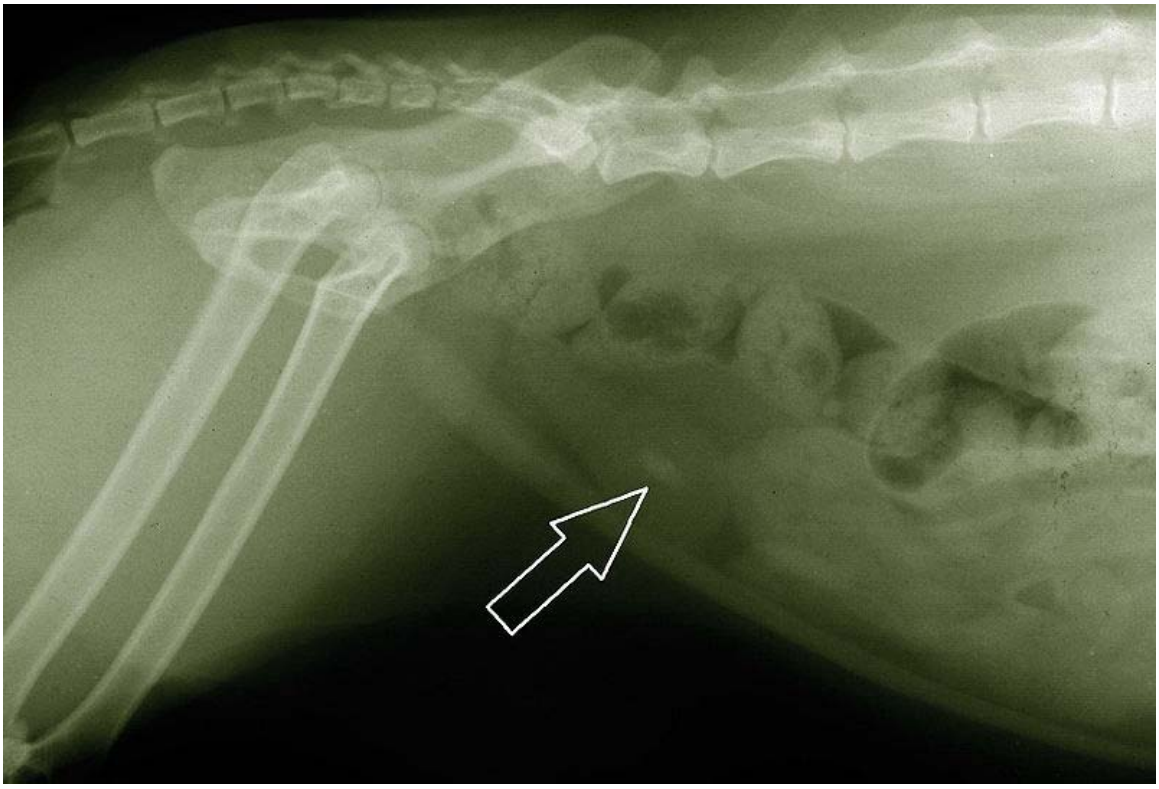
Bladder Stone (Animal)

Bladder stones or **uroliths** are a common occurrence in animals, especially in domestic animals such as dogs and cats. Occurrence in other species, including tortoises, have been reported as well. The stones form in the urinary bladder in varying size and numbers secondary to infection, dietary influences, and genetics. Stones can form in any part of the urinary tract in dogs and cats, but unlike in humans, stones of the kidney are less common and do not often cause significant disease, although they can contribute to pyelonephritis and chronic renal failure. Types of stones include struvite, calcium oxalate, urate, cystine, calcium phosphate, and silicate. Struvite and calcium oxalate stones are by far the most common.

Particularly large (510 gram) canine bladder stone



X-ray of bladder stones in a dog



X-ray of a Struvite bladder stone in a cat

Formation of bladder stones

Oversaturation of urine with crystals is by the far the biggest factor in stone formation. This oversaturation can be caused by increased excretion of crystals by the kidneys, water reabsorption by the renal tubules resulting in concentration of the urine, and changes in urine pH that influence crystallization. Other contributing factors include diet, frequency of urination, genetics, current medications, and the presence of a urinary tract infection.

The stones form around a nidus, which can consist of white blood cells, bacteria, and organic matrix mixed with crystals, or crystals alone. The nidus makes up about two to ten percent of the mass of the stone. It is possible for the nidus to be made of a different type of crystal than the rest of the stone, also known as epitaxial growth.

Symptoms

Bladder stones may cause blood in the urine (hematuria) (giving the appearance that the animal is urinating blood) but sometimes there may be no signs at all. Painful urination or straining to urinate are other signs. Urinary tract infections are commonly associated with bladder stones. Smaller stones may become lodged in the urethra, especially in male animals, causing urinary tract obstruction and the inability to urinate. This condition causes acute renal failure, hyperkalemia, septicemia, and death within a few days.

Diagnosis

When symptoms indicate bladder stones, the first step is usually to take an x-ray. Most types of stones will appear readily in an x-ray, urate and occasionally cystine stones being the most common exceptions. Stones smaller than three millimeters may not be visible. Ultrasonography is also useful for identifying bladder stones. Crystals identified in a urinalysis may help identify the stones, but analysis of the stones is necessary for identification of the complete chemical composition.

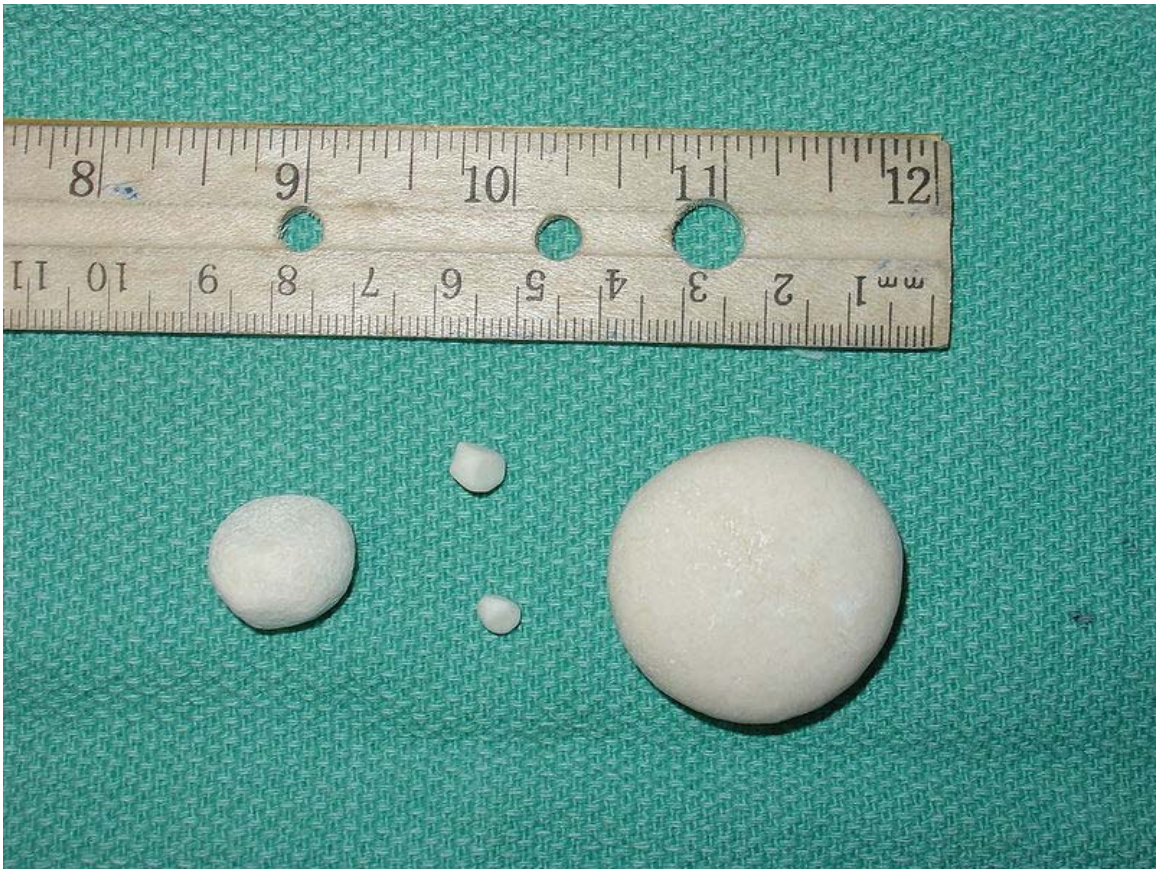
Treatment

Reasons for treatment of bladder stones include recurring symptoms and risk of urinary tract obstruction. Some stones can be dissolved using dietary modifications and/or medications. Small stones in female dogs may possibly be removed by urohydropropulsion, a nonsurgical procedure. Urohydropropulsion is performed under sedation by filling the bladder with saline through a catheter, holding the dog vertically, and squeezing the bladder to expel the stones through the urethra. Bladder stones can be removed surgically by a cystotomy, opening of the bladder. Stones lodged in the urethra can often be flushed into the bladder and removed, but sometimes a urethrotomy is necessary. In male dogs with recurrent urinary tract obstruction a scrotal urethrostomy creates a permanent opening in the urethra proximal to the area where most stones lodge, behind the os penis. In male cats, stones lodge where the urethra narrows in the penis. Recurrent cases can be treated surgically with a perineal urethrostomy, which removes the penis and creates a new opening for the urethra.

To prevent recurrence of stones, special diets can be used for each type of stone. Increasing water consumption by the animal dilutes the urine, which prevents oversaturation of the urine with crystals.

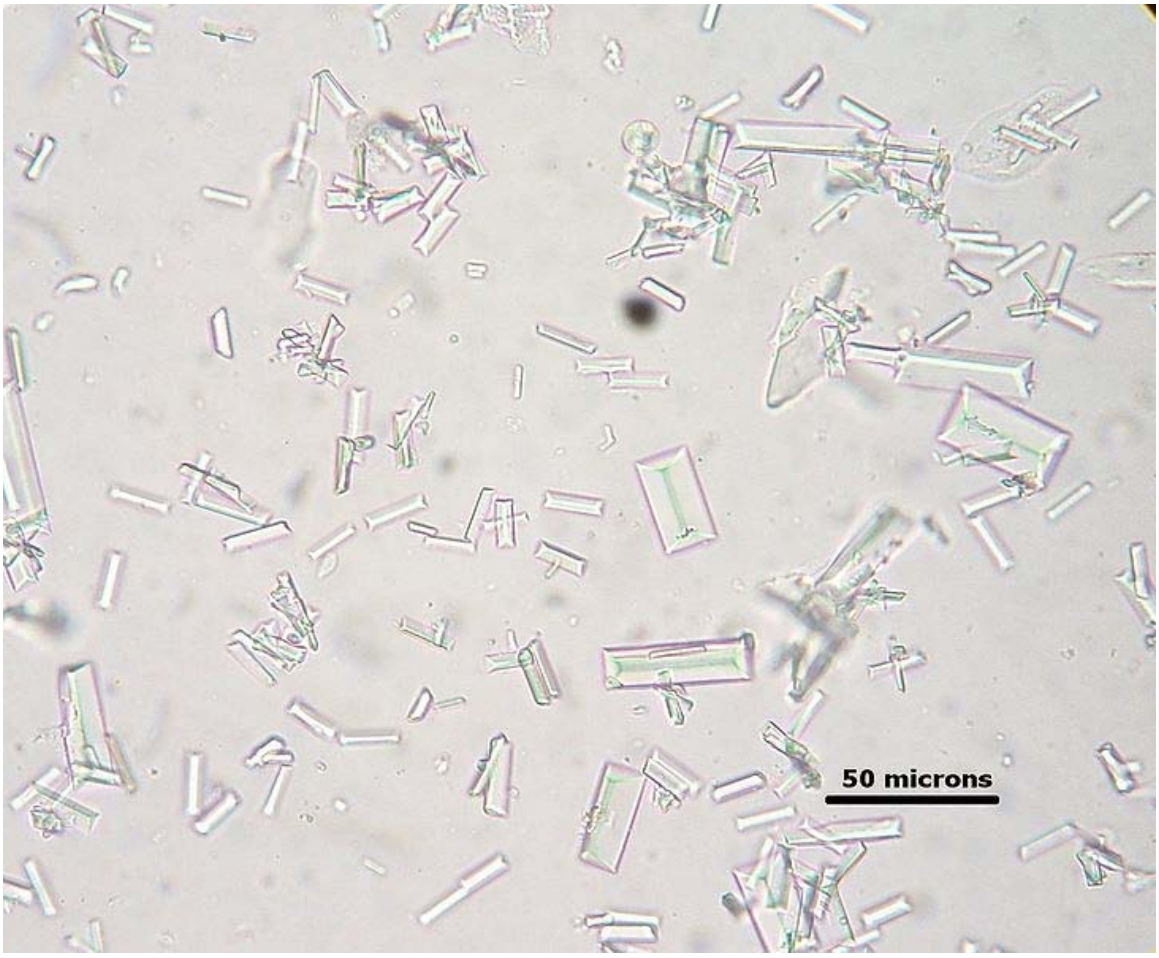
Types of bladder stones

Struvite stones



Struvite stones

Struvite stones are also known as magnesium ammonium phosphate stones due to their chemical composition - $MgNH_4PO_4 \cdot 6H_20$. Often there is a small amount of calcium phosphate present. They form at a neutral to alkaline pH of the urine. Bacterial infections contribute to their formation by increasing the pH of the urine through the urease enzyme in dogs. More than 90 percent of dogs with struvite stones have an associated urease-producing bacterial infection in the urinary tract, but in cats struvite stones usually form in sterile urine. The appearance of the stones vary from large solitary stones to multiple smaller stones. They can assume the shape of the bladder or urethra.

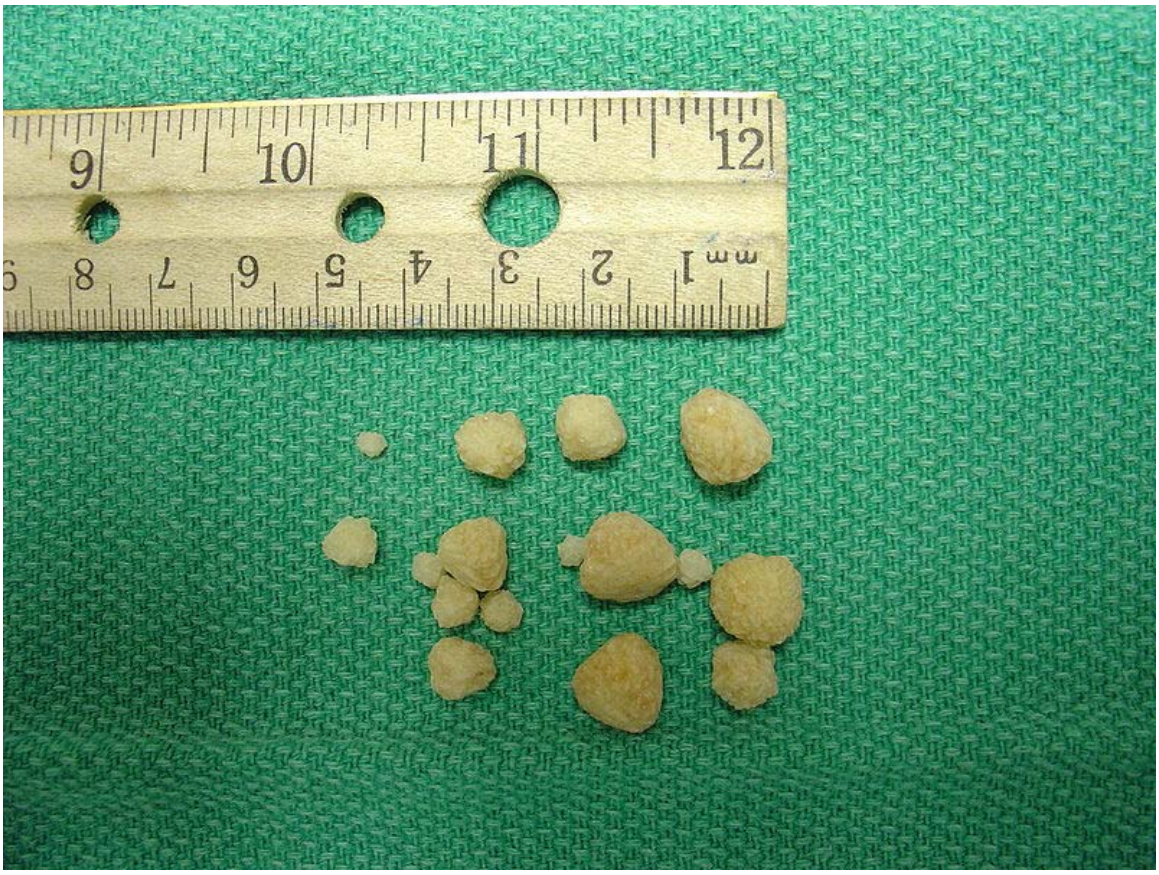


Struvite crystals

Dissolution of the struvite stones depends on acidification of the urine through diet or urinary acidifiers. Special diets for dissolution also have reduced protein, phosphorus, and magnesium, as well as increased salt to increase water consumption and dilute the urine. The diet needs to be fed exclusively, but it can only be fed for a few months total due to potential side effects. Contraindications to this diet include heart failure, liver failure, kidney failure, pancreatitis, hypertension (high blood pressure), and hypoalbuminemia (low serum albumin). Prevention of struvite stones is with a similar diet with milder restrictions.

Certain dog breeds are predisposed to struvite stones, including Miniature Schnauzers, Bichon Frises, and Cocker Spaniels. They are the most commonly reported bladder stone in female dogs and in ferrets (pregnant ferrets may be especially predisposed).

Calcium oxalate stones



Calcium oxalate stones

Calcium oxalate stones form in an acidic to neutral urine. Two types naturally occur, calcium oxalate monohydrate, or whewellite ($CaC_2O_4 \cdot H_2O$), and calcium oxalate dihydrate, or weddellite ($CaC_2O_4 \cdot 2H_2O$). Their appearance can be rough, smooth, spiculated, or jackstone. Calcium oxalate stones form more readily in animals with hypercalcaemia, which can caused by Addison's disease or certain types of cancer. Hypercalcaemia results in hypercalciuria, which can also be caused by Cushing's syndrome or hyperparathyroidism.

There is no recommended diet to dissolve calcium oxalate stones. For prevention a diet low in protein and oxalates and high in magnesium, phosphorus, and calcium is recommended. Increased dietary magnesium and phosphorus decreases the amount of calcium in the urine, and increased dietary calcium reduces absorption of oxalates from the intestines. Potassium citrate has been recommended as a preventative for calcium oxalate stone formation because it forms a soluble complex with oxalates and promotes the formation of alkaline urine.

Dog breeds possible prone to calcium oxalate stones include Miniature Schnauzers, Lhasa Apsos, Yorkshire Terriers, Miniature Poodles, Shih Tzus, and Bichon Frises. They are the most common stone in male dogs. Calcium oxalate stones are also common in

domestic rabbits. Rabbits are prone to hypercalciuria due to intestinal absorption of calcium not being dependent on vitamin D and a high fractional urinary excretion of calcium. The urine will appear thick and creamy or sometimes sandy. Small stones and sand can be removed using urohydropropulsion. Prevention is through reducing calcium intake by feeding more hay and less commercial rabbit pellets, and by not using mineral supplements.

Frequency of struvite and calcium oxalate stones in cats

The Minnesota Urolith Center at the University of Minnesota College of Veterinary Medicine has done detailed analysis of uroliths from animals since 1981 and has noted changing trends in feline uroliths. In 1981, struvite stones were the most common type in cats, making up 78 percent of submitted samples, with only 2 percent comprising calcium oxalate stones. In the mid 1980s there was a substantial increase in the number of calcium oxalate samples, and between 1994 and 2002, 55 percent of feline stones were calcium oxalate and 33 percent were struvite. This may have been caused by the use of dissolution diets for struvite stones in cats and modification of other diets to prevent struvite crystal formation. These modifications predisposed to calcium oxalate crystal formation. However in 2004, struvite stones once again surpassed calcium oxalate stones 44.9 percent to 44.3 percent, and in 2006, 50 percent of stones were struvite and 39 percent were calcium oxalate. This may have been due to the increased use of diets designed to prevent calcium oxalate crystal formation, which because of increased magnesium in the diet and decreased acidity of the urine help promote struvite crystal formation.

Urethral plugs in cats are usually composed of struvite crystals and organic matter.

Urate stones

Urate stones

Urate ($C_5H_4N_4O_3$) stones, usually ammonium urate ($NH_4 \cdot C_5H_4N_4O_3$) or sodium urate monohydrate ($Na \cdot C_5H_4N_4O_3 \times H_2O$), form in an acidic to neutral urine. They are usually small, yellow-brown, smooth stones. Urate stones form due to an increased excretion of uric acid in the urine. Dalmatians (especially males) and to a lesser extent Bulldogs are genetically predisposed to the formation of urate stones because of an altered metabolism of purines. Dalmatians have a decreased rate of urate hepatic transport, leading to only about 30 to 40 percent conversion of urate to allantoin, compared with greater than 90 percent conversion in other breeds. Dogs with portosystemic shunts or endstage liver disease also have increased uric acid excretion in the urine due to reduced conversion of uric acid to allantoin and ammonia to urea. Urate stones make up about six percent of all stones in the cat.

Urate stones can be dissolved using a diet with reduced purines that alkalinizes and dilutes the urine. Allopurinol is used in dogs with altered purine metabolism to prevent the formation of uric acid. Feeding a diet high in purines while simultaneously administering allopurinol can result in the formation of xanthine ($C_5H_4N_4O_2$) stones.

Cystine stones

Cystine ($(SCH_2CHNH_2COOH)_2$) stones form in an acidic to neutral urine. They are usually smooth and round. They are caused by increased urine excretion of cystine (a relatively insoluble amino acid) in dogs with a defect in renal tubule reabsorption of cystine. Dietary reduction of protein and alkalinization of the urine may help prevent formation. Medications such as D-penicillamine and 2-MPG contain thiol, which forms a soluble complex with cystine in the urine. Dog breeds possibly predisposed to formation of cystine stones include Bulldogs, Dachshunds, Basset Hounds, Chihuahuas, Yorkshire Terriers, Irish Terriers, and Newfoundlands. In Newfoundlands, cystinuria is inherited as an autosomal recessive trait, but in the other breeds it is a sex linked trait and found primarily in male dogs.

Calcium phosphate stones

Calcium phosphate, also known as hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$), stones form in neutral to alkaline urine. They are usually smooth and round. Calcium phosphate is usually a component of struvite or calcium oxalate stones and is infrequently a pure stone. They form more readily with hypercalcaemia. Dog breeds possibly predisposed to calcium phosphate stone formation include Yorkshire Terriers, Miniature Schnauzers, and Cocker Spaniels.

Silicate stones

Silicate (SiO_2) stones form in acidic to neutral urine. They are usually jackstone in appearance. There is possibly an increased incidence associated with dogs on diets that have a large amount of corn gluten or soybean hulls. Dog breeds possibly predisposed include German Shepherd Dogs, Golden Retrievers , Labrador Retrievers, and Miniature Schnauzers.

Chapter 11

Ethnoveterinary Medicine

Ethnoveterinary medicine (EVM) considers that traditional practices of veterinary medicine are legitimate and seeks to validate them (Köhler-Rollefson and Bräunig, 1998). Many non-Western traditions of veterinary medicine exist, such as acupuncture and herbal medicine in China, Tibetan veterinary medicine, Ayurveda in India, etc. These traditions have written records that go back thousands of years, for example the Jewish sources in the Old Testament and Talmud and the Sri Lankan 400-year-old palm-leaf frond records of veterinary treatments (Hadani and Shimshony, 1994). Since colonial times scientists had always taken note of indigenous knowledge of animal health and diagnostic skills before implementing their Western-technology projects.

What Is EVM?

In the 1980s the term "Veterinary Anthropology" was coined for a particular approach to animal health care, which was researched through "using the basic repertoire of anthropology's research skills and techniques, including observation, interview and participation" (Köhler-Rollefson and Bräunig, 1998). Ethnoveterinary medicine or ethnoveterinary research was defined by McCorkle in 1995 as:

The holistic, interdisciplinary study of local knowledge and its associated skills, practices, beliefs, practitioners, and social structures pertaining to the healthcare and healthful husbandry of food, work, and other income-producing animals, always with an eye to practical development applications within livestock production and livelihood systems, and with the ultimate goal of increasing human well-being via increased benefits from stockraising.

Stock owners continue to utilize EVM until better alternatives in terms of efficacy, low cost, availability and ease of administration, are found. By far the most-studied element of EVM is veterinary ethnopharmacopoeia, especially botanicals.

Justification of studies

According to Tabuti et al. (2003) and others, systematic studies on EVM can be justified for three main reasons:

1. they can generate useful information needed to develop livestock healing practices and methods that are suited to the local environment,
2. EVM could be a key veterinary resource and could add useful new drugs to the pharmacopoeia, and
3. EVM can contribute to biodiversity conservation.

Organizations

Developing world institutes involved in EVM include Mexico's Universidad Nacional Autónoma de Chiapas, Ethiopia's Addis Ababa University, the School of Veterinary Medicine of the University of the West Indies, and Rwanda's University Centre for Research on Traditional Pharmacology and Medicine.

The Heifer Project International works in Cameroon with herders and healers experienced in EVM. The League for Pastoral People (L.P.P.) has worked with camel pastoralists in Rajasthan, India and has produced a field manual on camel diseases. Recent research on EVM in the developed world has come from Italy (Pieroni, 2004), British Columbia, Canada (Lans et al., 2006) and the Netherlands (van Asseldonk).

Women and EVM

The importance of gender is being increasingly recognised in EVM. One of the first studies to document gender was conducted by Diana Davis who noted a difference in knowledge of EVM of Afghan Pashtun nomads that paralleled the gender-based division in the society. Davis found that women know more about healthcare for newborns and very sick animals that are taken care of near the home. Since women prepare the carcass for consumption they know twice as many types of internal parasites as men. Women also help with dystocias and the manual removal of ectoparasites.

Another study is that of the Tzotzil Maya shepherdesses who developed their own breed of sheep and have their own husbandry and healthcare system based on their traditions (Perezgrovas, 1996).

In research conducted in Trinidad it was noted that male farmers were using the reproductive knowledge of their female relatives to assist in the health care of their ruminants. Female farmers were using the same plants for their animals that they used for themselves (Lans, 2004).

ANTHRA, an organization of women veterinary scientists, has been documenting and validating EVM since 1996 in different parts of the states of Andhra Pradesh and Maharashtra in India (Ghotge, 2002). ANTHRA chose to study EVM because women

farmers performed 50 – 90% of all daily activities related to livestock care but were denied aspects of the local EVM because knowledge was traditionally passed from father to son.

Women are not trained as traditional Dinka healers (atet) in Sudan (VSF/Switzerland, 1998). However female-headed households are increasing in Sudan due to war, and women are thus more visible as livestock rearers.

Validation

Herbal remedies used for hundreds of years by stockraisers can be put to commercial use, but scientists are demanding that traditional knowledge should be validated, to verify the safety and efficacy of the treatments.

IT Kenya has a project in the Samburu District that is investigating effective EVM treatments. Vetaid is collaborating with the Animal Disease Research Institute of Dar-es-Salaam in Tanzania while the Christian Veterinary Mission is investigating EVM in Karamojo, Uganda. Other organizations in the field are ANTHRA and SEVA in India, ITDG and KEPADA in Kenya and World Concern in Uganda (Mathias, 2004). Studies on EVM have been commissioned by UNICEF.

- Gauthuma (2004) tested the efficacy of Myrsine africana, Albizia anthelmintica and Hildebrandtia sepalosa against mixed natural helminthosis in sheep (Haemonchus spp, Trichostrogylus spp and Oesophagostomum spp) in the Samburu district of Kenya. Healers were included in the study and the extracts were prepared following traditional methods including mortar and pestle. Albizia anthelmintica and Hilderbrantia sepalosa treatments showed significant improvement over controls from day 4 after treatment to day 12. On day 12 the three plant remedies showed 100% efficacy while albendazole had an efficacy of 63%.
- Six of the 17 plant extracts used by the Hausa and other tribes of Northern Nigeria for symptoms probably indicative of viral illness were found to have antiviral activity (Kudi and Myint, 1999). The extracts of Eugenia uniflora, Acacia artaxacantha, Terminalia ivorensis, T. superba and Alchornea cordifolia showed trypanocidal activity (Adewunmi, 2001).
- The International Centre of Insect Physiology and Ecology in Kenya is evaluating 40 natural products used against ticks by the Bukusu people in Bungoma District (Wanzala, in process).
- The Onderstepoort Veterinary Institute in South Africa is screening the plants used in EVM for biological activity (Van der Merwe, 2002). Other work in South Africa has been conducted by Masika (2002).
- The anthelmintic efficacies of Teminalia glaucescens (48.4%), Solanum aculeastrum (34.4%), Khaya anthoteca (55.8%) and Vernonia amygdalina (52.4%) were tested by Nfi (2001). Nfi and colleagues had previously tested the insecticidal activity of Nicotiana tabacum and Tephrosia vogelli and plan to test the acaricidal potential of Euphorbia kamerunica and Psorospermum guianensis.

- MacDonald (2004) found that that the traditional form of usage of Chenopodium ambrosioides infusions as a vermifuge is safer than the use of the herb's essential oil.
- Iqbal (2004) compared the in vitro and in vivo anthelmintic activity of *Artemisia brevifolia* with levamisole. In vitro studies revealed anthelmintic effects of crude aqueous (CAE) and methanol extracts (CME) of Artemisia brevifolia (whole plant) on live Haemonchus contortus as evident from their paralysis and/or mortality at 6 h post exposure. For in vivo studies, the whole plant of Artemisia brevifolia was administered as crude powder (CP), CAE and CME at graded doses (1, 2 and 3 g kg(-1) body weight (b.w.) to sheep naturally infected with mixed species of gastrointestinal nematodes. Maximum reduction (67.2%) in eggs per gram (EPG) of faeces was recorded on day 14 post treatment in sheep treated with Artemisia brevifolia CAE at 3 g kg(-1) b.w. Levamisole produced a 99.2% reduction in EPG. However, increase in EPG reduction was noted with an increase in the dose of Artemisia brevifolia administered as CP, CAE and CME.
- Githiori (2004) tested seven plant preparations of Hagenia abyssinica, Olea europaea var. africana, Annona squamosa, Ananas comosus, Dodonea angustifolia, Hildebrandtia sepalosa and Azadirachta indica in 151 lambs infected with 5000 or 3000 L3 Haemonchus contortus in 3 experiments and all were found to be ineffective.

Chinese medicine is also being investigated; treatments historically used for large animals are now being tested on pets. Nagle (2001) conducted a randomized, double-blind, placebo-controlled trial of P07P, a product derived from a traditional Chinese herbal remedy. There was a positive result, but it was not statistically significant.

Blanco (1999) found that traditional veterinary knowledge of Galicia, northwest Spain was still thriving due to distrust of the veterinary service. However the traditional system of holding IK as oral knowledge of certain elders is not necessarily a panacea. Stories of loss are recorded in Giday (2003) and in (Ayisi, 1994).

The PRELUDE database on traditional veterinary medicine has over 5000 plant-based prescriptions for livestock disorders with each plant listed by family.

Environment, epidemiology and health

EVM in future may be increasingly linked to discussions and research on ecosystem health. EVM is now increasingly integrated into "participatory epidemiology" which seeks to improve epidemiological surveillance in remote areas and encourage community participation in disease control (Mathias, 2004). EVM is also studied to provide solutions to diseases in which antigen variation has made vaccination unrealistic and drug resistant strains to Western medicines have become prevalent (Atawodi, 2002).

Chapter 12

Veterinary Chiropractic

Veterinary chiropractic also known as Animal chiropractic, is an emerging subspecialization for Doctors of Veterinary Medicine (DVMs) and Doctors of Chiropractic (DCs) to provide spinal manipulation, manual therapy and other holistic and conservative techniques for animals. In concert with conventional veterinary care, the complementary use of veterinary chiropractic is primarily used for common neuromusculoskeletal conditions. This reflects the use of complementary and alternative veterinary medicine which continues to grow within the veterinary community. Proposed benefits of animal chiropractic include, enhanced performance, function and quality of life. Currently there are uneven regulation and licensing standards across North America with certain juridictions, such as Ontario, who have regulated the practice of veterinary chiropractic.

Scope of practice

Traditionally, all animal care fell under the exclusive juridiction of veterinarians. With the emergence of veterinary chiropractic, both doctors of chiropractic (DCs) and veterinary medicine (DVMs) can take additional training to become certified in veterinary chiropractic. The minimum standard for practice appears to be a minimum of 210 hours according to the Animal Chiropractic Accreditation Commission although, in Australia, a 3 year Masters Degree in Chiropractic Science (Animal Chiropractic) is offered to licensed doctors of chiropractic, veterinary and osteopathic medicine. Where regulated, typical restricted acts include diagnosis and spinal manipulation. In some locations, a veterinarian must supervise the treatment provided by the veterinary chiropractor Veterinary chiropractors typically treat working horses, racing greyhounds, and pets; and recently have been used more extensively to treat ongoing and chronic pain caused by conditions of the neck and back. Those that specialize in horses are referred to as "Equine Chiropractors." There has been discussion over who should perform animal chiropractic. Veterinarians, chiropractors, or both.

Clinical practice

Before working on an animal, a veterinary chiropractor procures a detailed case history of the animal including prior diagnoses, therapies, and X-ray or laboratory analyses. The American Veterinary Medical Association guidelines recommend that a veterinarian should examine an animal and establish a preliminary diagnosis before any alternative treatment, like chiropractic, is initiated. Before performing a chiropractic adjustment, the doctor examines the animal's gait, posture, and the vertebrae and extremities. In addition to spinal manipulation, other adjustive procedures can be performed to the extremity joints and cranial sutures. Veterinary chiropractors also make neurological evaluations.

History and present status

Chiropractic treatment of large animals dates back to the early 1900s and is common in dogs and popular in horses. Animal chiropractic was formalized in 1989 by Sharon Willoughby, with a 100 hour post-graduate course. Several complementary and alternative veterinary medicine (CAVM) presentations were given in the 2007 annual convention of the American Veterinary Medical Association, including chiropractic care and acupuncture. As of 2008, chiropractors and veterinarians are trained side by side in the Masters Degree in Chiropractic Sciences (Animal Chiropractic) in Australia and the North American AVCA certified programmes, including at schools of chiropractic such as the Canadian Memorial Chiropractic College and Parker College of Chiropractic.

The profession of animal chiropractic is controversial. The policy of the American Chiropractic Association, the largest professional association of chiropractors, is that the term *veterinary chiropractic* is a misnomer and should not be used to refer to applying manipulative techniques to animals, and that the term does not describe what is commonly defined by the ACA and most U.S. states as *chiropractic*. This policy has been in effect since 1994. Consequently, more than simple manipulative techniques constitutes veterinary chiropractic. The AVCA program may address this issue by including applied neurology, biomechanics, philosophy, rehabilitation and other subjects that traditionally fall under a chiropractic curriculum.

Education, certification and regulation

Pre-requisites

North American applicants must have graduated from an accredited veterinary school or CCE-accredited chiropractic school and hold current licenses from their respective provinces or states. In Australia a first professional degree in chiropractic, osteopathic or veterinary medicine is required for admission into the Masters of Chiropractic Science program.

Education

Most veterinary chiropractic programs are a minimum of 210 hours of additional training following the completion of veterinary or chiropractic school, and subsequent licensure. Practitioners will be able to complete an appropriate history, physical examination, communicate a diagnosis and plan of management, and provide care where indicated within their respective scopes of practice. Though there is variation, common topics covered in veterinary chiropractic programs can include:

- Anatomy
- Basic and advanced Neurology
- Rehabilitation Therapy
- Complementary and alternative medicine modalities
- Philosophy
- Basic and advanced chiropractic manipulative techniques

Currently, the Animal Chiropractic Accreditation Commission (ACAC) is the defacto accrediting body for veterinary chiropractic. All accredited programs must meet ACAC's minimum requirement of 210 hours. A passing grade of 75 in both the written, theoretical and the clinical competency examination is required for certification. Continuing education requirements of the ACAC are 30 credits every 3 years for recertification. Though few U.S. veterinary schools offer educational or research programs in complementary and alternative veterinary medicine (CAMV), in a survey, 61% of faculty believe that chiropractic should be included in their school's curriculum.

Efficacy

A study found chiropractic manipulation increased pain thresholds. Another study found that there were significant changes in thoracolumbar and pelvic kinematics after chiropractic manipulation in horses, and gave the opinion that "Some of the changes are likely to be beneficial, but clinical trials with increased numbers of horses and longer follow-up are needed." There is currently insufficient published evidence of efficacy to make specific recommendations about the use of chiropractic intervention for chronic pain in dogs and cats, as well as a lack of research into efficacy of chiropractic care on horses.

Safety

Veterinary chiropractic methods can potentially cause injury through the use of inappropriate technique or excessive force. In addition, there is some degree of risk associated with even skilled manipulation in animals as the potential for injury exists with any technique used. Ramey *et al.* (2000) states that there is an increased risk in the presence of structural disease, such equine cervical vertebral malformation or canine intervertebral disk disease. A reply by Taylor and Romano (2000) states that veterinary chiropractic has been shown clinically to be safe and effective for those conditions.

Chapter 13

Zoophilia and Health

Equines can transmit Brucellosis, Leptospirosis, and Q fever.

This chapter describes the health implications of sexual contact with animals. Areas of possible concern are infection, physical injury and allergic reaction.

Zoonoses acquired via sexual contact

Infections that are transmitted from animals to humans are called zoonoses. A few of zoonoses may be transferred through casual contact, but others are much more readily transferred by activities that expose humans to the semen, vaginal fluids, urine, saliva, feces and blood of animals. This means that sexual activity with animals is sometimes a high risk activity. It is advisable for practitioners of bestiality to assess their relative risk, since risk varies for each species involved, for each disease mentioned below (and others not mentioned), and for each region in the world. Some of the more common zoonoses are listed at the National Agricultural Safety Database (NASD) and the Centers for Disease Control (CDC).

List of zoonoses

Animal diseases that can be transmitted sexually to humans
(Note: not all diseases listed)

Zoonosis	Mode of transmission	Carrier species	Regions	Risky sex acts	Fact sheet
Brucellosis	semen, vaginal fluids, urine	D,E,R,P	A,AF,Er,SA,NAr	A,B,P,S,V	eMedicine
Leptospirosis (Weil's disease)	semen and urine	D,E,P,R,Z	W	A,P,S	eMedicine
	Complicated to treat; easily misdiagnosed; requires urgent hospitalization at specialist center				
Q fever	semen, vaginal fluids, urine	C,D,E,P,R	W	A,B,F,M,P,S,V	eMedicine
	Treated w/ antibiotics, sometimes long-term; vaccine available in Australia & E. Europe				
Rabies	saliva	C,D,E	W (not AUS)	B,M	CDC
	Lethal if untreated; vaccine available (people & animals); post-exposure prophylaxis if exposed				
Flea tapeworm	saliva	C,D	W	M	CDC
	Readily treated w/ anti-parasitics				
Echinococcosis (Hydatid disease)	fecal-oral	C,D,Z	W	F,M	CDC
	Surgical removal of tapeworm cyst; possibly fatal if untreated.				
Campylobacter	fecal-oral	B,C,D,P,R,Z	W	F,M	eMedicine
	Readily treated w/ specific antibiotics				
Cryptosporidium	fecal-oral	B,C,D,Z	W	F,M	eMedicine
	Protozoal infection, usually causes a self-limited diarrhea				
Cysticercosis (Pork tapeworm)	fecal-oral	P	W	F	CDC
	Readily treated w/ anti-parasitics; (rarely) may require surgery (eye, brain)				
Giardia	fecal-oral	C,D,R,Z	W	F,M	CDC
	Diarrhea; readily treated w/ anti-protozoal drugs				
Salmonella	fecal-oral	B,C,D,E,P,R,Z	W	F,M	CDC
	Self-limited diarrhea, complete recovery usual, rarely causes reactive arthritis				
Toxocariasis (Dog roundworm)	fecal-oral	C,D	W	F,M	CDC
	Treated w/ anti-parasitics; usually benign but may be dangerous (eye)				

High Prevalence[†]Region code

Code	Region
A/Ar	Asia, all/regions
AF/AFr	Africa, all/regions
AUS	Australia
E/Er	Europe, all/regions
NA/NAr	North America, all/regions
SA/SAr	South America, all/regions
W	Worldwide

Sexual activity[††] code

Code	Activity
A	Human-receptive anal
B	Involves contact with animal blood
F	Involves contact with animal feces
M	Involves mouth-to-mouth contact
P	Penetration, genital-to-genital
S	Involves contact with animal semen
V	Involves contact with animal vaginal fluids

Species code

Code	Species
B	Birds (incl. Poultry)
C	Cats
D	Dogs
E	Equines (Horses, donkeys etc.)
P	Pigs
R	Ruminants (goats, sheep etc.)
Z	Other

† May occur in other areas but at a lower rate. †† Not a definitive list. Not medical advice.

Brucellosis

Brucellosis in humans is a potentially life-threatening multisystem disease that can be extremely difficult to treat. There are several varieties of Brucellosis, all caused by bacteria of the genus *Brucella*, the most notable being *B. abortus* and *B. melitensis* which affect larger species of domestic animals, and *B. canis* which infects dogs and other canids. All are widely spread around the world. The most severe infections are thought to be associated with *B. melitensis* which primarily infects goats, sheep, and camels in the Mediterranean, Asia, Latin America, parts of Africa and some southern European countries. Humans can catch *B. canis* through contact with the body fluids of infected dogs, especially semen, urine and vaginal fluids. Dogs can be infected with Brucellosis without showing any signs or symptoms, and infection can only be diagnosed with specific blood tests. The typical symptoms of the type of brucellosis contracted from dogs are: fever that comes and goes, loss of appetite, fatigue, weakness, malaise, sore joints, low back pain, spine pain, headache, depression, abdominal pain, constipation, diarrhoea, vomiting, weakness, dizziness, unsteadiness of gait, and urinary retention. Heart and lung complications can occur. Infected people exhibit only some of these symptoms. Other forms of brucellosis can be more severe.

With approximately 500,000 zoonotic infections a year worldwide (source:CDC), brucellosis places a large burden on humanity. Brucellosis has been reduced to rare disease status in North America (excluding Mexico) and northern Europe through vaccination and eradication programs, but it remains rife throughout the rest of the world. In most countries up to 10% of dogs carry this bacterium, and even up to 42.7% in some provinces of China, representing a major threat to the health of veterinarians and people who handle the blood or semen of infected animals. In the USA, there are only about 100 cases of human brucellosis diagnosed per year, although some sources consider it underdiagnosed and underreported. Most other countries have much higher rates, with high risk areas including the Mediterranean Basin (Portugal, Spain, Southern France, Italy, Greece, Turkey, North Africa), South and Central America (including Mexico), Eastern Europe, Asia, Africa, the Caribbean, and the Middle East.

Emerging zoonoses

New diseases that can jump from animals to humans are called *emerging zoonoses*. The emerging zoonosis situation changes constantly, in an upward trend. An example from the equine species is the rare Hendra virus, originally passed from flying foxes to horses. The implications for zoophilic sexual contact of each emerging disease should be carefully assessed by practitioners.

Zoophilia and human sexually transmitted diseases

HIV / AIDS

HIV (the "AIDS" virus) was originally a zoonosis acquired from primates (notably monkeys) in Africa, probably via hunting and eating but possibly via animal bite. It only lives in primates (humans, apes and monkeys) and is not believed to survive long in other species or away from the human body and fluids. The myth that sex with an animal can cure AIDS is false.

Other STDs

Human sexually transmitted diseases ("STDs") are not carried or transmitted by animals.

However, many human pathogens can survive in animal fluids for a limited time, and therefore STDs may theoretically be transmitted by an animal that has multiple consecutive human sexual partners in a short enough time frame to allow pathogen survival.

Bites and other physical injury

Humans may be at substantial physical risk and seriously harmed by sexual activity with animals. Larger animals may have the strength and defensive attributes (e.g. teeth, hooves, horns, claws) to injure a human, either in rejecting physical or sexual contact, or during sexual arousal.

Bites

Many animals bite as part of sexual excitement and foreplay. Animals carry numerous bacteria in their mouths capable of causing disease after a bite. The most common risk after an animal bite is simple infection (infection risk approximately 15-20% in the USA, may be higher elsewhere), and for dogs and other large animals injury from the force of the bite. Bacterial bite infections are usually fully curable, although dog bites may cause *Pasteurella* and *Capnocytophaga canimorsus* infections, which may have severe consequences.

Other injury

The sexual organs of other species may not safely conform to the human anatomy. For example, the penis of a sexually aroused dog has a broad bulb at the base which can cause injury if forcibly pulled from a woman's vagina, and equines can thrust suddenly and "flare" inside a human orifice.

In 2005, Kenneth Pinyan, a resident of Enumclaw, WA state died from internal injury after being anally penetrated by a stallion. In 2002, a 62-year-old farmer in Bulgaria was treated for a torn rectum after sex with a boar (male pig). In 1976, a 46-year-old French farmer underwent surgery for peritonitis after sex with a boar.

Allergic reactions

Sensitization and allergic reactions to animal saliva and semen may occur, ranging from mild irritation to anaphylaxis. Although dried skin flakes, known as *dander*, are the most commonly cited allergen, dog saliva is a more potent allergen than dander. An estimated 10% of people are allergic to animals in general, rising to 20-30% amongst asthma sufferers; the percentage of people allergic to animal *secretions* in particular is currently unknown. Repeated exposure to secretions after sensitization has already occurred may subsequently provoke an anaphylactic reaction, which can be life-threatening, and should be avoided.

Chapter 14

Veterinarian

A **veterinarian** (American English) or a **veterinary surgeon** (British English), often shortened to **vet**, is a doctor who treats animals and a practitioner of veterinary medicine. The word comes from the Latin *veterinae* meaning "working animals". "Veterinarian" was first used in print by Thomas Browne in 1646. Many careers are open to those with veterinary degrees (**Doctor of Veterinary Medicine**), D.V.M., VMD (**Veterinaria Medicina Doctoris**), MVB (**Medicina Veterinaria Baccalaureate**), BVS (**Bachelor of Veterinary Surgery**), BVMS (**Bachelor of Veterinary Medicine and Surgery**), BVetMed (**Bachelor of Veterinary Medicine**) or B.V.Sc. & A. H. (**Bachelor of Veterinary Science and Animal Husbandry**). Those working in clinical settings often practice medicine in a limited field such as "companion animal" or pet medicine, which includes small animals such as dog, cat, and pocket pets, production medicine or livestock medicine. Production medicine includes specialties in dairy cattle, beef cattle, swine, sheep, and poultry, equine medicine (e.g., sport, race track, show, rodeo), laboratory animal medicine, reptile medicine, or ratite medicine. Veterinarians may choose to specialize in medical disciplines such as surgery, dermatology or internal medicine, after post-graduate training and certification.

Some veterinarians pursue post-graduate training and enter research careers and have contributed to advances in many human and veterinary medical fields, including pharmacology and epidemiology. Research veterinarians were the first to isolate oncoviruses, *Salmonella* species, *Brucella* species, and various other pathogenic agents. Veterinarians were in the forefront in the effort to suppress malaria and yellow fever in the United States. Veterinarians identified the botulism disease-causing agent, produced an anticoagulant used to treat human heart disease, and developed surgical techniques for humans, such as hip-joint replacement, limb and organ transplants.

Like physicians and animal therapists, veterinarians must make ethical decisions about their patients' care. For example, there is ongoing debate within the profession over the ethics of performing declawing of cats and docking or cropping tails and ears, spaying or neutering dogs, as well as "debarking" dogs, the housing of sows in gestation crates and the housing of egg laying poultry hens in battery cages.

A veterinarian gives an injection to a goldfish

Education and regulation

Veterinarians must first obtain a degree in Veterinary Medicine and a license to practice. The competition for admission into veterinary school is stiff. Individuals who are interested in pursuing a career in veterinary medicine must graduate with either a Doctor of Veterinary Medicine (D.V.M. or V.M.D.) degree from North America; the equivalent degree for veterinarians who graduate in the U.K. or other commonwealth country is a Bachelor of Veterinary Science/Surgery/Medicine (BVS, BVSc, BVetMed or BVMS) degree and the equivalent for veterinary graduates in Ireland is a Medicina Veterinaria Baccalaureate (MVB) degree. The title and degree name "Doctor" in the US is considered around the world as an honorary one, as the DVM degree does not result in a thesis, publication, or other academic doctorate qualification as in a PhD. There was a time in the US where the name of a veterinary degree was also a Bachelor's, but the degree name and academic system was modified to match the honorary title of the profession. This dynamic is still in place in the UK and Australia, where vets are called veterinary surgeons.

In the United States, there are currently only 28 veterinary schools that meet the accreditation standards set by the Council on Education of the American Veterinary Medical Association (AVMA): Auburn University, Colorado State University, Cornell

University, Iowa State University, Kansas State University, Louisiana State University, Michigan State University, Mississippi State University, North Carolina State University, Ohio State University, Oklahoma State University, Oregon State University, Purdue University, Texas A&M University, Tufts University, Tuskegee University, University of California, Davis, University of Florida, University of Georgia, University of Illinois at Urbana-Champaign, University of Minnesota, University of Missouri, University of Pennsylvania, University of Tennessee, University of Wisconsin-Madison, Virginia-Maryland Regional College of Veterinary Medicine, Washington State University, and Western University of Health Sciences

In Canada, there are currently five veterinary schools that meet the accreditation standards set by the Council on Education of the American Veterinary Medical Association (AVMA) and Canadian Veterinary Medical Association (CVMA) : Ontario Veterinary College (OVC), University of Saskatchewan, Université de Montréal, Atlantic Veterinary College, and UCVM (University of Calgary Veterinary Medicine) - University of Calgary.

There are a number of other countries that also have AVMA-accredited institutions such as Australia, Ireland, New Zealand, the United Kingdom and Netherlands.

In West Indies, 3 Veterinary Schools - (RUSVM-Ross University School of Veterinary Medicine) Ross University, St. George's University School of Veterinary Medicine - St. George's University and St. Matthew's University (SMU) are listed by the American Veterinary Medical Association and Canadian Veterinary Medical Association and its graduates qualify for entrance into the Educational Commission for Foreign Veterinary Graduates (ECFVG) or the Program for the Assessment of Veterinary Education Equivalence (PAVE) certification programs.

An alternative to becoming a licensed veterinarian is becoming a veterinary technician. Veterinary technicians are, essentially, veterinary nurses, and are graduates of two or four year college-level programs and are legally qualified to assist veterinarians in many medical procedures. Veterinary assistants are not licensed by most states, but can be well-trained through programs offered in a variety of technical schools.

The prerequisites for admission to veterinary programs vary from school to school with many programs not requiring a bachelor's degree for entrance. Instead they require a number of credit hours that range from 45 to 90 semester hours at the undergraduate level. However, most students admitted have completed an undergraduate program and earned a bachelor's degree. So despite the fact that a bachelor's degree is not required, applicants without a degree are at a disadvantage.

Preveterinary courses should emphasize the sciences. Veterinary schools typically require applicants to have taken classes in organic and inorganic chemistry, physics, biochemistry, general biology, animal biology, animal nutrition, genetics, vertebrate embryology, cellular biology, microbiology, zoology, and systemic physiology. Additionally, some programs require calculus. On the other hand, some require only

statistics, college algebra and trigonometry, or precalculus. Most veterinary schools also require some courses in English or literature, other humanities, and the social sciences as a basic background education. Furthermore, courses in general business management and career development are more and more becoming a standard part of the curriculum.

In addition to satisfying pre-veterinary course requirements, applicants must submit test scores from standardized tests such as the Graduate Record Examination (GRE), the Veterinary College Admission Test (VCAT), or the Medical College Admission Test (MCAT). The decision as to which test should be taken depends solely on the requirement of the college to which the applicant is applying. As of 2007, 22 schools require the GRE, 4 require the VCAT, and 2 accept the MCAT.

Admission to veterinary school is highly competitive, with the number of qualified applicants admitted varying from year to year. This is due in large part to the fact that the number of accredited veterinary colleges has remained largely the same since 1983, but the number of applicants has risen significantly. As a result, only about 1 in 3 applicants were accepted into veterinary school in 2005.

Approximately 80% of admitted students are female. In the early history of veterinary medicine of the USA, most veterinarians were males. However, in the 1990s this ratio reached parity, and now it has been reversed. Most veterinary schools require their applicants to submit applications through the Veterinary Medical College Application Service (VMCAS).

New graduates with a DVM/VMD/BVS/BVSc degree cannot begin to practice veterinary medicine until they have received their license. To be licensed in the United States for example, one must receive a passing grade on a national board examination, the North America Veterinary Licensing Exam. This exam must be completed over the course of eight hours, and consists of 360 multiple-choice questions. This exam covers all aspects of veterinary medicine, as well as visual material designed to test diagnostic skills. Unlike physicians of whom an academic internship is generally required (and 85% eventually board certify in one of a large number of specialties and subspecialties) veterinarians can enter practice after graduation and licensure. The percentage electing further study has increased from 36.8% to 39.9% in 2008. About 25% of those or about 9% of graduates were accepted into traditional academic internships. (2008 -696 graduates accepted a position in advanced study, 89.2% (621) accepted an internship (private practice, 74.5%; academic, 25.3%; and other internship, 0.2%). An additional 6.0% (42) accepted a residency). Approximately 9% of veterinarians eventually board certify in one of 20 specialties..

Interns receive a small salary, but often find that their internship experience leads to better paying opportunities later. Veterinarians who then wish to pursue board certification in medical or surgical specialties, such as internal medicine, oncology, surgery, dermatology, cardiology, neurology, ophthalmology , must complete a 3- to 4-year residency program that provides intensive training. Other specialties, such as epidemiology or toxicology, require a PhD training.

When the application committee decides who gains admittance and who does not, many schools place heavy emphasis and consideration on a candidate's veterinary and animal experience. Formal experience is a particular advantage to the applicant. Formal experience consists of work with veterinarians or scientists in clinics, agribusiness, research, or some area of health science. Less formal experience is also helpful for the applicant to have, and this includes working with animals on a farm or ranch or at a stable or animal shelter and basic overall animal exposure.

Admittance Comparison

Veterinary school requires extensive preparation, and the likelihood of acceptance is not in favor of the applicant. Nationwide in 2007, approximately 5,750 applicants competed for the 2,650 seats in the 28 accredited veterinary schools in the United States. This statistic results in nationwide acceptance rate of 46 percent

WICHE: Veterinary School Financial Alternative

The Professional Student Exchange Program (PSEP) is one of three exchange programs of the Western Interstate Commission for Higher Education (WICHE). Western states, in particular, can place their residents who are pursuing professional, graduate, and undergraduate programs, which are not available to them in their own state, at a financial disadvantage. These exchange programs are designed to give students in these disadvantageous situations another financial option and place them on a more fair and even status. This is done so by providing the outbound students and their families the option to save money through reduced tuition arrangements.

The Western Interstate Commission for Higher Education, based in Boulder, Colorado, works with 15 states to expand educational access and excellence for all of the citizens in the West region. The states that participate in WICHE include: Alaska, Arizona, California, Colorado, Hawaii, Idaho, Montana, Nevada, New Mexico, North Dakota, Oregon, South Dakota, Utah, Washington, and Wyoming.

If selected to receive support, WICHE exchange students pay reduced levels of tuition. This usually consists of paying resident tuition in public institutions or reduced tuition at private schools. The home state of the students then pays a support fee to the admitting schools to help cover the cost of the students' education. Another advantage that WICHE students receive is that they are given some preference in admission selection process. Each state determines just how many fields and students they are willing and able to support; veterinary medicine is usually one of these fields.

For veterinary medicine, maximum WICHE support is limited to four academic years. The following states are in compliance with the WICHE program and will support students who wish to pursue a DVM Arizona, Hawaii, Montana, Nevada, New Mexico, North Dakota, Utah, and Wyoming. States with additional support arrangements include North Dakota, Utah, and Wyoming. The following veterinary Schools are those who are willing to receive students under support of the WICHE program University of California

Davis, Colorado State University, Oregon State University, and Washington State University.

Educational Requirements in Various Countries

The educational requirements for the veterinarian vary with each country. Typically, it takes from four years to eight years of education after graduating from secondary school. Some countries grant a bachelor's degree - ie, the UK, Australia, Ireland, and India. Due to historical nomenclature modifications, other countries grant a doctoral degree - in the United States and Canada, the Doctor of Veterinary Medicine (DVM or VMD). In the rest of the world, the 'doctoral' designation of the degree granted in North America is viewed as an honorary one, as the degree does not result in a thesis, publication, or dissertation. Thus in the US and Canada, licensed vets are called "Doctor", but not always in other countries, where they are called "Surgeon". In the United States, holders of either degrees are allowed to practice as veterinarians if they succeed in passing national and state board examinations, and after passing three veterinary licensing exams - the Basic and Clinical Sciences Examination (BCSE), the National Board of Veterinary Medical Examiners' North American Veterinary Licensing Examination (NAVLE), and the Clinical Proficiency Examination (CPE), and a state veterinary law exam, foreign-educated veterinarians may practice as a general practice veterinarian.

Applicants must have earned or be close to earning bachelor's degrees before applying and must take the Veterinary College Admission Test (VCAT), Graduate Record Exam (GRE), or Medical College Admission Test (MCAT). The chances of admission in one state might be significantly different from those in another state, depending on the number of in-state applicants and the number of places available.

Admission competition in US Veterinary Schools

In the United States, the average veterinary medicine student admitted into 28 Veterinary Schools, has an undergraduate GPA of 3.5 (minimum) and a GRE score of approximately 1350/1600 (minimum). Some US Veterinary Schools, among total 28 US Veterinary schools, even accept a minimum GPA of 3.8/4.0 and minimum GRE score of 1500 out of 1600. Minimum Work Experience of 2–3 years as Veterinary Assistant or Animal Care Taker / Animal Care Attendant and Interview are also taken into consideration along with GPA and GRE Score, while considering admission into a DVM programme in US Veterinary Schools. In the U.S. and Canada, veterinary school lasts four years (again, normally after the completion of an undergraduate degree), with at least one year being dedicated to clinical rotations. In the U.S., one can enter veterinary school (DVM) after completing the undergraduate pre-veterinary requirements in as little as two years but chances of admission into DVM are less, but most veterinary school applicants have completed a bachelor's degree (most of them hold a 4 years BS in Animal Science/Biology/Microbiology/Zoology with minimum GPA of 3.5 or above) to entry into a **Doctor of Veterinary Medicine** program in 28 US Veterinary Schools. DVM applicants must have strong background in Animal Science, Math, Genetics, Biochemistry , Biology / Zoology with A+ grade.

In many other countries, the veterinary degree is granted upon completion of a bachelor's degree in veterinary medicine and is not usually a post-graduate program as in the U.S. and Canada. After completion of the national board examinations, some newly-accredited veterinarians choose to pursue residencies or internships in certain (usually more competitive) fields. The entry requirement for residency is 1 year of internship or 2 years of clinical experience plus research publication. The admission in residency program is highly competitive. Most of the veterinarians work as general practice veterinarian, only few become Veterinary Specialists.

In India, the Veterinary medical degree is known as **Bachelor of Veterinary Sciences and Animal Husbandry** (B.V.Sc. and A.H.). The program lasts for a period of five years with 4.5 years of course work and six months of clinical and farm training internships. Admission to the Veterinary Colleges are through the tests conducted by the Agricultural and Veterinary Universities in the respective states or through a National Level Joint Entrance Test. Admission into BVSc & AH program in India is competitive due to fewer Veterinary Colleges and seats.

In Pakistan UVAS (University of Veterinary and Animal Sciences-LAHORE) takes its own test for admission in M.Phil degree after DVM. Also interviews are held for the candidate with his choice of department which he is applying to join.

Salary

The mean salary for new graduates in 2010 was US$48,674 including nearly 50% going on to advanced study programs. Those not continuing their studies made US$67,359 at first. . **Those who have completed 1 Year Internship after License, start at US $ 140,000.** The average income for private practice rose from $105,510 in 2005 to $115,447 in 2007. These increased values exceed those of public practice including uniformed services and government . According to a survey done by the American Veterinary Medical Association, the average starting salaries of new graduates in 2006 depended upon their respective fields of practice. The Bureau of Labor Statistics in the Occupational Outlook Handbook, 2008-2009 Edition recorded the following:

Large animals, exclusively: $61,029
Small animals, predominantly: $57,117
Small animals, exclusively: $56,241
Private clinical practice: $55,031
Large animals, predominately: $53,397
Mixed animals: $52,254
Equine (horses): $40,130

Vets in the UK tend to make less than those in the US with average new graduate wages starting at an average of £25000.

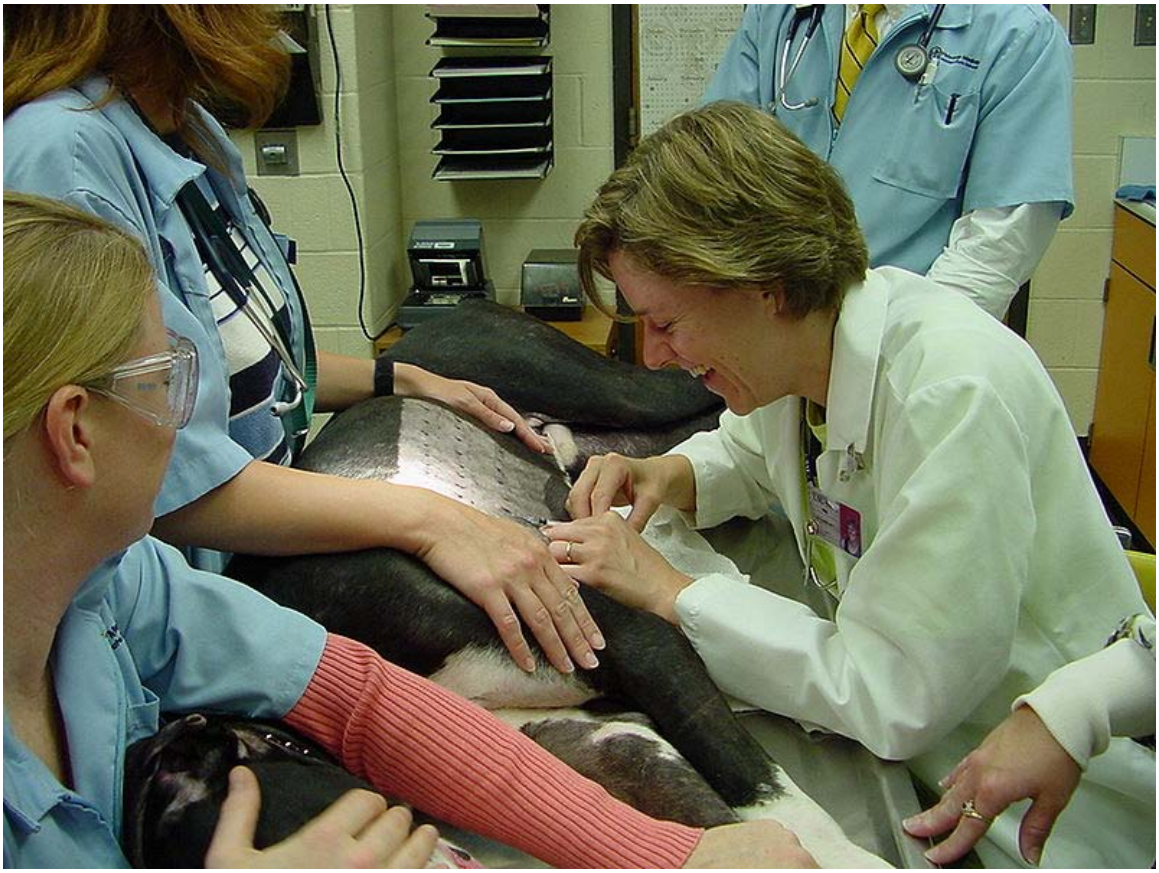
Work

Veterinarians may:

- Diagnose animal health problems, and perform diagnostic tests such as X-ray, EKG, ultrasound, blood tests, stool tests, and urinalysis.
- Vaccinates against diseases, such as distemper and rabies.
- Medicate animals suffering from infections or illnesses.
- Treat and dress wounds.
- Set fractures.
- Perform minor to complex surgery, depending on training.
- Advise owners about animal feeding, behavior, and breeding.
- Euthanize animals when necessary.
- Provide preventive care to maintain the health of food animals.
- Test for and vaccinates against diseases.
- Dental work

According to the American Veterinary Medical Association, about three-quarters of veterinarians were employed in either an individual or group practice. The remainder were employees in other settings, including colleges of veterinary medicine, medical schools, research laboratories, animal food companies, and pharmaceutical companies. The Bureau of Labor Statistics reports that around 1,400 civilian veterinarians are employed by the United States federal government, mainly in the Department of Agriculture, Department of Health and Human Services, and Department of Homeland Security. State and local governments also employ veterinarians.

Skills required of a general practice veterinarian



In many respects a veterinarian is similar to a pediatrician. Animals cannot talk like human beings, and much of the clinical history is obtained from the owner or client as a pediatrician would obtain the medical history from a child's parents. Excellent people skills, and communication skills are required. Veterinarians, like other physicians, require well-functioning physical and sensory faculties in order to diagnose and treat their patients. They also make use of diagnostic tests like x-ray, C.T., M.R.I., blood work, urinalysis, and fecal exams to diagnose patients. Veterinarians are well trained in laboratory medicine and parasitology.

The general practice veterinarian spends one third to one half of his or her time in surgery. Animal neutering operations are done in most veterinarians' offices. Many veterinarians also perform orthopedic procedures, bone setting, dentistry, and trauma surgery. Surgery requires good hand and eye coordination, and fine motor skills.

Focuses of practice

Many areas of focus exist for veterinary practices. These include:

- Exotic animal veterinarian - Generally considered to include reptiles, exotic birds such as parrots and cockatoos, and small mammals such as ferrets, rabbits, chinchillas, and degus.
- Conservation medicine - The study of the relationship between animal and human health and environmental conditions.
- Small animal practice - Usually dogs, cats, and other companion animals/household pets such as hamsters and gerbils. Some practices are canine-only or feline-only practices.
- Laboratory animal practice - Some veterinarians work in a university or industrial laboratory and are responsible for the care and treatment of laboratory animals of any species (often involving bovines, porcine species, felines, canines, rodents, and even exotic animals). Their responsibility is not only for the health and well being of the animals, but also for enforcing humane and ethical treatment of the animals in the facility.
- Large animal practice - Usually referring to veterinarians that work with, variously, livestock and other large farm animals, as well as equine species and large reptiles.
- Equine medicine - Some veterinarians are specialists in equine medicine. Horses are different in anatomy, physiology, pathology, pharmacology, and husbandry to other domestic species. Specialization in equine veterinary practice is something that is normally developed after qualification, even if students do have some interest before graduation.
- Food animal medicine - Some veterinarians deal exclusively or primary with animals raised for food (such as meat, milk, and eggs). Livestock practitioners may deal with ovine (sheep), bovine (cattle) and porcine (swine) species; such veterinarians deal with management of herds, nutrition, reproduction, and minor field surgery. Dairy medicine practice focuses on dairy animals. Poultry medicine practice focuses on the health of flocks of poultry; the field often involves extensive training in pathology, epidemiology, and nutrition of birds. The veterinarian treats the flock and not the individual animals.
- Food safety practice - Veterinarians are employed by both the food industry and government agencies to advise on and monitor the handling, preparation, and storage of food in ways that prevent foodborne illness.
- Wildlife medicine - A relatively recent branch of veterinary medicine, focusing on wildlife. Wildlife medicine veterinarians may work with zoologists and conservation medicine practitioners and may also be called out to treat marine species such as sea otters, dolphins, or whales after a natural disaster or oil spill.

Veterinary specialties

As opposed to human medicine, general practice veterinarians greatly out-number veterinary specialists. Most veterinary specialists work at the veterinary schools, or at a referral center in large cities. As opposed to human medicine, where each organ system has its own medical and surgical specialties, veterinarians often combine both the surgical and medical aspect of an organ system into one field. The specialties in

veterinary medicine often encompass several medical and surgical specialties that are found in human medicine.

Veterinary specialties are accredited in North America by the AVMA through the American Board of Veterinary Specialties. In Europe, specialties are accredited through the European Board of Veterinary Specialisation. In Australia, specialties are recognized by the Australian Veterinary Boards Council. While some veterinarians may have areas of interest outside of recognized specialties, they are not legally specialists.

- Anaesthesiology - A specialty limited to teaching in hospitals and schools. Most veterinarians practice anesthesiology in their own office. In North America, anesthesiologists are certified through the American College of Veterinary Anesthesia.
- Animal behavior - A relatively new specialty, with an increased interest in modulating abnormal animal behavior. In North America, behaviorists are certified through the American College of Veterinary Behaviorists.
- Cardiology and cardiothoracic surgery - Manages cardiac and conductance disorders; also performs cardiothoracic surgery for the treatment of congenital and acquired heart disease. In North America, cardiologists are certified through the American College of Internal Medicine as a subspecialty.
- Dentistry - Relates to prevention and treatment of dental disease. In North America, dentists are certified through the American Veterinary Dental College.
- Dermatology and dermatopathology - Relates to the skin. Veterinary dermatologists diagnose and treat skin disease. Dermatology in animals encompasses much of the field of allergy and immunology. In North America, dermatologists are certified through the American College of Veterinary Dermatology.
- Emergency Medicine and Critical Care - Also cover the field of emergency or trauma surgery. The veterinarian is trained in medicine, surgery, and critical care of the severely injured or ill animal. In North America, criticalists are certified through the American College of Veterinary Emergency and Critical Care.
- Epidemiology and public health - Focus on infectious disease in animals (including zoonotic disease, infectious diseases in animals which are transmitted (in some instances, by a vector) to humans. In North America, epidemiolologists are certified through the American College of Veterinary Preventive Medicine.
- Internal medicine - As opposed to human medicine, where an internist is often considered a primary care physician of adults; a veterinary internal medicine specialist, is a specialist. The specialty in the United States requires 3 years of residency training. They are trained to manage complex medical conditions, and often work at teaching universities and hospitals. In North America, internists are certified through the American College of Internal Medicine as a subspecialty. Internists subspecialize in either small animal or large animal medicine.
- Microbiology Work in the diagnosis and control of infectious diseases in animals. Specialists in this field often work in industry, the regulatory agencies, and teaching institutions. In North America, micriobiologists are certified through the

American College of Veterinary Microbiologists. There are subspecialties in Virology, Bacteriology/Mycology, and Immunology.

- Neurology and Neurologic Surgery - Veterinary neurologists are both surgeons and neurologists in practice. This is different than in human medicine, where neurologists are the medical side of the specialty, and neurosurgeon or orthopedic surgeons focus on the surgical side. In North America, neurologists are certified through the American College of Internal Medicine as a subspecialty.
- Nutrition - An important food animal medicine, and herd medicine. Specialists in this area include veterinarians and animal scientists. Most large animal veterinarians are also excellent nutritionists. Nutritionists also work in the pet food industry in quality assurance and research. In North America, nutritionists are certified through the American College of Veterinary Nutrition.
- Oncology - Covers the diagnosis and management of malignancies in animals. As animals are considered to be a part of the family, curative and pallative care is often demanded when malignancies develop. In North America, oncologists are certified through the American College of Internal Medicine as a subspecialty.
- Ophthalmology - Focuses on eyes, the diagnosis of eye diseases, and surgery of the eye and eyelid. In North America, veterinary ophthalmologists are certified by the American College of Veterinary Ophthalmology.
- Pharmacology - The study of drug action. As animals metabolize drugs in many different ways, veterinary clinical pharmacologists are important in the study of drug use in animals. In North America, veterinary pharmacologists are certified by the American College of Veterinary Clinical Pharmacology.
- Parasitology - Focuses on study of parasites such as whipworms, fleas, and ticks. While almost all veterinarians encounter parasites in some patients, parasitology specialists are usually found in teaching hospitals and universities. In Australia, parasitologists are recognized by the Fellowship of the Australian College of Veterinary Scientists. There is not yet a parasitology specialty college in North America, although development of a subspecialty in the American College of Veterinary Microbiologists is underway.
- Anatomic Pathology and Clincal Pathology - A broad field covering multiple species, organ systems, and domestic and foreign animal diseases. The veterinary pathologists perform necropsies (autopsies), collect specimens, and read pathological slides. They assist clinicians in the diagnosis of illnesses and seek causes of deaths in animals. In North America, veterinary pathologists are certified by the American College of Veterinary Pathologists, and subspecialize in either anatomic pathology or clinical pathology.
- Radiology and radiation oncology - Interpretation of imaging modalities, including X-rays, magnetic resonance imaging (MRI), computed tomography (CT scans), ultrasounds, echocardiograms, and Doppler devices; administration of radiation treatment for malignancies and endocrine diseases. In North America, veterinary radiologists are certified by the American College of Veterinary Radiology.
- Surgery - In North America, surgeons are certified by the American College of Veterinary Surgeons. Surgeons subspecialize in either small animal or large animal surgery.

- Theriogenology involves the study and treatment of reproductive disorders. Reproduction is an economically important aspect of bovine, porcine, ovine, and equine practices. In North America, theriogenologists are certified by the American College of Theriogenology.
- Zoological medicine - The treatment and care of captive zoo animals, free ranging wildlife species, aquatic animals, birds, reptiles and amphibians, and non-domestic companion animals. In North America, zoological medicine specialists are certified by the American College of Zoological Medicine.