One Health Newsletter

A quarterly newsletter highlighting the interconnectedness of animal and human health

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This newsletter was created to lend support to the One Health Initiative and is dedicated to enhancing the integration of animal, human, and environmental health for the benefit of all.

Chikungunya--Could It Be The Next West Nile?

Thomas P. Monath, MD

Chikungunya is a mosquito-borne alphavirus, a member of the same taxonomic group as eastern equine encephalomyelitis (EEE) virus, which is familiar to Floridian public health physicians and veterinarians.

The term 'chikungunya' is a Makonde word for 'that which bends up', referring to the crippling muscle and joint pains that double over the patient during the acute disease. Chikungunya was first described in 1952-53 during an epidemic in East Africa. Though rarely fatal, the disease is prostrating, with high fever, rash, pains, and (in some patients) long-lasting rheumatoid symptoms. Encephalitis is a rare complication.

Chikungunya virus is distributed widely in Africa, the Indian subcontinent, SE Asia, and the Western Pacific, where outbreaks have occurred from time to time. It is a zoonosis, and wild monkeys serve as the reservoir hosts. Not unexpectedly, humans are effective viremic hosts and can serve as the source of mosquito infection. Hence, major outbreaks are sustained by inter-human transmission by mosquitoes, including *Aedes aegypti.*

Aedes aegypti (Courtesy of CDC)

In the past several years there has been a dramatic explosion of chikungunya virus activity affecting three continents (1). Explosive epidemics involving millions of people have affected the Comoros Is. (2005); Mauritius, the Seychelles Is. and Réunion (2006); India (2006); and ultimately southern Europe (Italy, 2007), demonstrating long-distance spread by viremic air travelers and the potential for spread in this way to the Americas. Imported cases have been reported among travelers in Australia.

The historical record suggests that chikungunya caused human epidemics in the Caribbean and South America in the early 19th Century, when it was confused with dengue. The principal vector in the recent outbreaks in the Indian Ocean was *Aedes albopictus*, which is now widely distributed in the United States and is a major pest species, after being introduced in imported truck tires from Asia (2). The virus strain causing the recent outbreaks has adapted to efficient transmission by *Ae. albopictus* by virtue of a specific mutation (3). In Réunion, an island with a population of 770,000, 1 out of 7 people was affected

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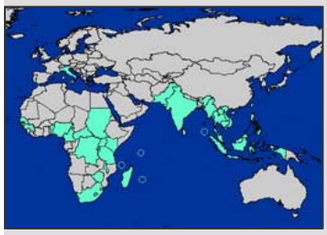

Approximate distribution of chikungunya virus, 2007
(Courtesy CDC)

The virus strain causing the recent outbreaks has adapted to efficient transmission by Aedes albopictus, a mosquito species which is now widely distributed in the United States.

Introduction of chikungunya virus into the United States would be a dramatic reminder of our vulnerability to foreign viral threats!

within a few months, illustrating a tremendous force of infection and potential for epidemic spread.

Introduction of chikungunya virus into the United States would be a dramatic reminder of our vulnerability to foreign viral threats. The ensuing outbreak would not resemble those in Africa and Asia because exposure to mosquito bite is more limited; nevertheless there would be considerable alarm as there was following the introduction of West Nile in 1999. Elderly people are more susceptible to severe disease following chikungunya (as for West Nile), and it is in this age group that deaths occur. Unlike West Nile, which rapidly inserted itself into wildlife (avian) transmission cycles and overwinters, chikungunya probably does not have this potential and would likely die off after control of inter-human transmission or a winter freeze. However, at least one African rodent species develops high viremia after inoculation, so that the possibility of escape into a natural transmission cycle cannot be excluded. There is no known veterinary disease associated with chikungunya, but given its close genetic relationship to some other alphaviruses (e.g., Semliki Forest virus) that cause disease in horses, it would not be surprising to find that chikungunya was also an equine pathogen. Antibodies have been found in horses indicating that they develop active infections. Experimental studies in kittens showed them susceptible to viremia. If an outbreak occurred in the US there should be a coordinated effort to conduct surveillance and disease investigations involving both human and animal populations, especially captive non-human primates in zoological parks and primate centers. If the virus were introduced into tropical America, there is a potential for continued transmission in wild monkeys.

The US has not developed an aggressive posture to prepare for the introduction of, or conduct surveillance for this disease, and no laboratory diagnostic test is readily available. This disease illustrates both weaknesses in our public health system and the conundrum of how to prioritize rare public health resources.

There is no antiviral drug. An experimental, live, attenuated vaccine for human use developed by the US Army in the 1980s is now being re-developed in France.

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Chikungunya virus in Florida: Lessons from Italy, 2007

Jonathan F. Day, Ph.D.

Aedes albopictus, the Asian tiger mosquito, was introduced into Florida in 1986 and has since spread throughout the state.

The Asian tiger mosquito *Aedes albopictus*
(Courtesy of CDC)

The chikungunya virus could be introduced into local Florida mosquitoes via an infected traveler returning from overseas.

At one time Florida experienced widespread yellow fever and dengue epidemics, both diseases are transmitted by the mosquito *Aedes aegypti*. *Aedes albopictus*, the Asian tiger mosquito, is another important vector of dengue and yellow fever. This 6-legged tiger was introduced into Florida in 1986 and has since spread throughout the state. The yellow fever and Asian tiger mosquitoes are also the main vectors for chikungunya virus, the alphavirus responsible for widespread epidemics of chikungunya fever in Asia and Africa in 2006-2007. So what chance does a virus like chikungunya have to gain a foot-hold in Florida, a state where, at one time, yellow fever and dengue ran wild?

Perhaps the most accurate scenario of the potential invasion of Florida by chikungunya virus already occurred in Italy during the summer and autumn of 2007. In early August, local health authorities in the Province of Ravenna detected an unusually high number of cases of febrile illness in two small neighboring towns with a total population of 3,767 located 6 km from the Adriatic coast. The two villages were separated by a slow moving stagnant river and lock system that produced large numbers of mosquitoes, including *Aedes albopictus*.

By the end of August, serological testing on suspected human cases confirmed the diagnosis of chikungunya virus. In addition, chikungunya virus was isolated by PCR from pools of *Aedes albopictus*. The initial establishment of *Aedes albopictus* in Italy was in the Veneto Region and was traced to the importation of used tires from Atlanta, Georgia in 1992. *Aedes albopictus* was first identified in the two epicenter villages in 2006, the year before the chikungunya epidemic.

Conditions around the houses in these villages seem optimal for vector blood feeding, reproduction, and dispersal. Houses were typically one story and were surrounded by small gardens with many flowers, plants and, most importantly, numerous flower pots. Open sewer systems containing stagnant water were evident just below street level.

The index case of the epidemic is presumed to be a resident of the Italian Provence containing the two epicenter towns. This individual traveled to the chikungunya-active Kerala State in India during June of 2007. He presented with two episodes of fever in late June, 2007. While ill he visited his cousin in one of the affected towns. The cousin, the second case in this outbreak, became ill on July 4th. These two cases were first identified as possible chikungunya infections to epidemiologists and vector control specialists in late August 2007. This reporting delay of eight weeks was crucial to later difficulties associated with control of the outbreak.

An active human disease surveillance system was set up for the entire Italian Provence on August 29, 2007. Control measures included adulticiding and larviciding on public and private land within 100 meters of the residence of all confirmed and suspected chikungunya cases. As of September 21, 292 chikungunya cases were identified within the transmission zone. Most of the cases continued to be reported from residents and visitors in the initial village

epicenters. However, beginning at the end of August, cases were reported with no known exposure in the two villages. This indicated that local transmission in adjacent areas was possibly fueled by dispersing infective *Aedes albopictus* females.

In spite of intense and prolonged vector control efforts the transmission of chikungunya virus continued into the third week of September, indicating the difficulty of managing arboviral epidemics once large numbers of adult mosquitoes become infective within a transmission zone. By the end of September the number of new cases began to diminish and the epidemic burned itself out with the onset of cold weather in mid October.

Communication and coordination between human and public health professionals and mosquito control are essential to prevent an introduced chikungunya epidemic in Florida.

For Florida to realize an epidemic of chikungunya virus we will likely need to have at least three pre-existing conditions in place:

1. First, focal areas within the state where *Aedes albopictus* populations are routinely "off scale."
2. Second, introduction of virus into one of these areas via an infected traveler or an already infected mosquito.
3. And third, a susceptible local human population with an unusually high level of day-to-day mosquito (*Aedes albopictus*) exposure within the introduction zone.

I believe that we can reduce the risk of an introduced chikungunya epidemic in Florida by addressing any one of these three conditions through effective *Aedes albopictus* vector-control programs, a sensitive human disease surveillance system, and excellent communication and coordination between human and public health professionals and mosquito control.

Dr. Jonathan F. Day is a Professor of Medical Entomology at the University of Florida, Florida Medical Entomology Laboratory in Vero Beach.

One Health and Biodiversity Loss

Leonard C. Marcus, VMD, MD

"One Health" is a concept that includes concern over the health status of all living things.

"One Health" is a concept that includes concern over the health status of all living things. Extinction of plant and animal species on a massive scale is imminently possible, so biodiversity loss should be a major priority for those promoting One Health. Such a dire prediction might suggest cartoon images of robed and bearded fanatics carrying signs warning the end of the earth is near, but, in fact, is in the report of the Intergovernmental Panel on Climate Change (IPCC) (http://www.ipcc.ch/) which shared the Nobel Peace Prize with Albert Gore in 2007.

The International Union for the Conservation of Nature (http://cms.iucn.org/) publishes Red Lists which indicate the number of species whose populations are in various degrees of significant decline, including the

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A cutaneous secretion from the paradoxical frog could be useful in the treatment of diabetes.

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whale watching

possibility of extinction. The following information is derived from their web site, Table 1 in the IUCN Red List.

Numbers of threatened species by major groups of organisms

	Number of described species	Number of species evaluated by 2007	Number of threatened species in 2007	Number threatened in 2007 as % of species described	Number threatened in 2007 as % of species evaluated
Mammals	5,416	4,863	1,094	20%	22%
Birds	9,956	9,956	1,217	12%	12%
Reptiles	8,240	1,385	422	5%	30%
Amphibians	6,199	5,915	1,808	29%	31%
Fishes	30,000	3,119	1,201	4%	39%
Insects	950,000	1,255	623	0.07%	50%

Other animal groups and plants are represented in the IUCN tables, but I have just listed vertebrates and insects above to illustrate how severe the known problem is and how ignorant we are of the total picture.

Mass extinction of species has occurred before, but the current crisis is unique because humans are here to witness it, suffer its consequences and are the principle cause of the problem. The causes of biodiversity loss include habitat loss, pollution of land, water and air, excess harvesting (hunting, fishing and collecting) and climate change. The IPCC report on climate change states “Approximately 20-30 % of plant and animal species assessed so far are likely to be at increased risk of extinction if increases in global average temperature exceed 1.5- 2.5 degrees Celsius”.

Biodiversity loss results in the loss of food, exemplified in the widespread depletion of fish stocks and in decreasing numbers of pollinator species. Many of the drugs used in veterinary and human medicine are derived from natural sources and this resource is threatened as species disappear. It is paradoxical that simultaneous with the discovery that a cutaneous secretion from the paradoxical frog (*Pseudis paradoxa*) could be useful in the treatment of diabetes there is increasing concern that frogs around the world are threatened with extinction from infection with a chytrid fungus (*Batrachochytrium dendrobatidis*). An immeasurable consequence of biodiversity loss is esthetic. How do you put a value on the joy of seeing, hearing and experiencing wilderness? (One answer to that seemingly rhetorical question is in the many millions of dollars spent annually in birding, whale watching and ecotourism.) Furthermore, our understanding of ecology is so limited that we cannot predict the consequences of any interruption in the chain of life.

It is very discouraging to see the lack of knowledge or concern about these matters in the general public and even in the medical community. Environmental issues rank very low when Americans are polled about issues that are important to them as shown in the web site www.pollingreport.com/prioriti.htm . The Nobel

Prize given to Al Gore and IPCC was for Peace, not for any field of science. Perhaps people will get more interested in environmental issues when wars break out over diminishing resources. At the very least the public should have a heightened awareness of environmental issues and should know how candidates for office stand on these issues. Such information can be obtained on the web site of the League of Conservation Voters, (www.lcv.org.)

The medical professions should act to better educate themselves and the public about the importance and relevance of environmental issues including biodiversity loss.

The medical professions should act to better educate themselves and the public about the importance and relevance of environmental issues including biodiversity loss. Premedical and preveterinary students should be encouraged to take biology courses that emphasize ecology. More research money should be directed at environmental issues even if it means diverting some funds from molecular studies. More emphasis on environmental issues should be in professional school education and could easily be incorporated in subjects like microbiology, epidemiology, etc.

In government at all levels offices dealing with natural resources should be involved in more public health decisions. For example, departments of fish and game should be consulted before measures are taken to control mosquitoes or other disease vectors to consider what collateral damage might occur.

I hope the medical community will become more involved in environmental issues as recommended in a recent editorial in the Journal of the American Medical Association (Auerbach, PS. Physicians and the Environment. JAMA 2008; 299:956-958). The stakes are high and the issues are urgent.

The stakes are high and the issues are urgent.

Dr. Leonard Marcus is a physician and veterinarian who recently retired from private practice in Newton, MA. He specialized in travelers' health, tropical medicine, parasitology, zoonoses and vector-borne diseases. Dr. Marcus is a member of the American Veterinary Medical Association's One Health Initiative Task Force.

The opinions stated in this article are his own and are not necessarily those of the Task Force or the AVMA, as their report is pending.

Florida Red Tide (*Karenia brevis*): Human and Animal Exposures and Health Effects

Lora E Fleming MD PhD and Jan H. Landsberg, PHD

What are Harmful Algal Blooms (HABs) and how are humans and animals exposed? Microalgae are microscopic organisms which form the basis of the food web in aquatic environments. Periodically, these microalgae grow exuberantly in what is known as "algal blooms." When algal blooms produce and release toxins into the water that adversely affect people, animals or the environment, they are called "harmful algal blooms" or "HABs."

Aquatic HABs can pose a key threat both to natural resources, and to human and animal health (32, 15, 41, 25, 6, 27, 28). Humans and animals are primarily affected by HAB toxins released into the water or that enter the food chain. As well as being toxic, proliferation of algae can lead to poor water quality, including low dissolved oxygen which can result in significant fish kills and fishery declines. Many HAB toxins are neurotoxins which can cause rapid death in aquatic

organisms and In terrestrial mammals, and can cause acute and chronic morbidity in humans. In addition, other HAB toxins are also hepatotoxic, dermatotoxic, or can act as tumor promoters (25, 3). Exposure to the HAB toxins can result from: a) consumption of toxic seafood (38, 3); b) inhalation of toxic aerosols (4, 1, 5, 12, 9, 20); c) direct skin contact (25); and d) in the case of freshwater HABs, consumption of toxic drinking water (17).

Karenia brevis
(Courtesy FWRI/FWC)

What is Florida red tide? *Karenia brevis* is a microalgal dinoflagellate that forms HABs known as "Florida red tides," an annual event common in Florida, but also found throughout the Gulf of Mexico (37). Occasionally, Florida red tides have been transported along the Gulf Stream as far north as North Carolina (39). *K. brevis* blooms are associated with the production of a group of powerful natural neurotoxins known as brevetoxins (37, 7, 22).

What risks do Florida red tides pose to the environment and health? Blooms of brevetoxin-producing *K. brevis* have been associated with massive fish kills, as well as the deaths of marine mammals, including endangered Florida manatees and bottlenose dolphins, sea turtles, marine birds, and invertebrates (37, 35, 33, 26, 24, 25, 42, 31). Since 1996, five major spring-time red tide events in southwest Florida have contributed to manatee mortalities representing more than 10% of the local population. During August 1999-February 2000, and again in 2004, unusual red tides in the Florida panhandle were responsible for more than 200 bottlenose dolphin deaths, and for increased turtle strandings (26, 42, 27, 18). In 2003, possibly for the first time, more than eight domestic dogs from the beach area of Little Gasparilla Island in southwest Florida were admitted to local veterinary clinics after being reportedly affected by exposure to brevetoxins during a highly concentrated Florida red tide event (Flewelling et al. Florida Fish and Wildlife Conservation Commission (FWC), unpublished data).

Blooms of Karenia brevis have been associated with massive fish kills, as well as the deaths of marine mammals, including endangered Florida manatees and bottlenose dolphins, sea turtles and marine birds.

People who eat shellfish contaminated with brevetoxins can experience neurotoxic shellfish poisoning (NSP) (38, 43), a disease with acute gastrointestinal and neurologic symptoms that apparently resolve within a few days of onset, however chronic health sequellae have never been explored (22, 3). Human shellfish poisonings were known in Florida since the 1880s, but the connection with filter-feeding shellfish, toxicity, and the *K. brevis* red tide was not identified until the 1960s. Thus far, NSP has only been caused by the consumption of toxic bivalves and rarely gastropods, and, luckily, there have been no reported human fatalities. Shellfish monitoring for public health protection is managed by the Florida Department of Agriculture and Consumer Services (DACS) using red tide distribution data and analytical results of brevetoxins in shellfish provided by the FWC's Fish and Wildlife Research Institute (FWRI) under the National Shellfish Sanitation Guidelines. Despite regulation, however, NSP cases may occur when people consume unregulated shellfish species, when shellfish are illegally harvested, or when blooms expand to other geographic areas.

Endangered Florida manatee
(Courtesy FWRI/FWC)

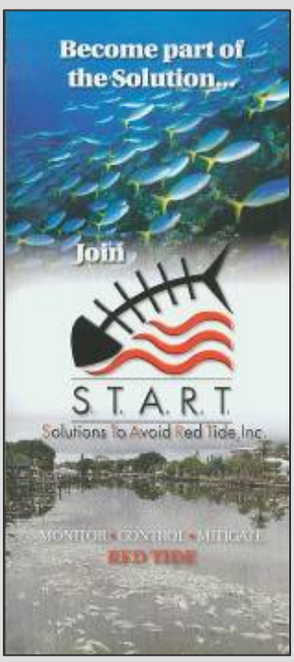
In addition to NSP caused by ingesting brevetoxin-contaminated shellfish, other environmental exposures can cause adverse human health effects (22). The *K. brevis* organism is quite fragile and is readily broken up in the surf, allowing for the production of aerosolized toxin (34). In fact, people living and working on or

The K. brevis organism is readily broken up in the surf, allowing for the production of aerosolized toxin which can cause respiratory irritation.

near coastal areas have complained for years about upper and lower respiratory irritation associated with onshore winds and aerosol exposures during active Florida red tides (44, 30, 7). Exposure to aerosolized brevetoxins induces respiratory irritation in healthy people and acute exacerbations in people with asthma (44, 4, 5, 16, 13, 29). Studies of asthmatics after only 1 hour of exposure to Florida red tide toxins have demonstrated continued symptoms from 5-10 days after exposure (Kirkpatrick et al. in press).

Although over the past decade there has been a renaissance in HAB research, a host of uncertainties still remain about the human and animal health risks associated with long term exposure to brevetoxins, and further study is warranted.

Florida Red Tide: Outreach and Education. There have been multiple efforts by state and federal agencies, grassroots groups, scientists, and even the tourism industry to inform people about the exposures and effects of the Florida red tides and their toxins. Some of these efforts include:



- the Aquatic Toxins Group at the Florida Dept of Health (http://www.doh.state.fl.us/ENVIRONMENTAL/community/aquatic/index.html)
- the 24/7 Aquatic Toxins Hotline (**888-232-8635**) at the Florida Poison Information Center (disease surveillance and a sources of health information)
- the NOAA HAB Bulletin (http://coastwatch.noaa.gov/hab/bulletins_ns.htm)
- the FWC (remote sensing and water monitoring for *K. brevis* cells to identify the locations of the red tides)(http://research.myfwc.com/ features/ category_sub.asp?id=4434)
- a new Beach monitoring system reported by lifeguards in Sarasota County (FL) giving real time beach conditions (http://coolgate.moteoorg/beachconditions/)(14).
- The public can report dead or dying animals during red tides to the FWC fish kill hotline at 1-800-636-0511 or online at (http://research.myfwc.com/fishkill/submit.asp).
- Daily information on shellfish bed closures cab be obtained from FDACS website (http://shellfish.floridaaquaculture.com/seas/seas_statusmap.htm)

References –

www.doh.state.fl.us/environment/community/One_Health/HABrefs_Spring2008.pdf

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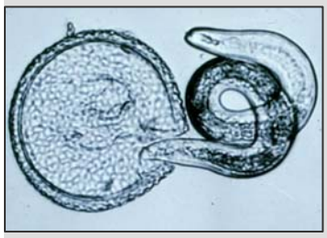
Human Larva Migrans Caused by Toxocara canis and Baylisascaris procyonis

Albert L. Vincent, Ph. D. and John N. Greene, M.D., F.A.C.P.



A second or third stage larva of *Toxocara canis*, 350-445 micrometers in length, emerges from its thick, resistant shell in the duodenum. (Courtesy of CDC)

Toxocara canis and T*. cati* are cosmopolitan, ascarid roundworms, primarily of domestic dogs and cats. Humans do not support full development of the worms to the egg-laying adult fifth stage worm in the intestine. Rather, following ingestion of their infective eggs by humans, hatched second or third stage larvae migrate through various tissues until becoming enveloped in granulomas, often in the liver. This aberrant behavior is the basis of two well-defined clinical syndromes, visceral larva migrans (VLM) and ocular larva migrans (OLM). *Toxocara* eggs are acquired from contaminated sandboxes, on playgrounds, in city parks or on public beaches where dogs are not "poop-scooped" or are allowed to roam unattended. VLM is largely disease of children under five years of age, who typically present with fever, hepatomegaly, eosinophilia sometimes approaching 70%, a bronchospasm which resembles asthma and hypergammaglobulinemia. Human toxocariasis is rarely fatal but penetration into an eye may result in endophthalmitis and blindness. OLM should be suspected in any child with unilateral vision loss and strabismus. In addition to VLM and OLM, other less well documented "covert syndromes" include the so-called idiopathic seizure, intestinal and skin disorders, a form of eosinophilic arthritis, and asthma.

Diagnostic tests are primarily immunological, currently employing a moderately specific enzyme-linked immunosorbent assays (ELISA) detection of antigens secreted by the second-stage larvae. In the U.S. the seroprevalence is about 3 percent. Because the infection is self-limited, steroid and antihistamine therapy directed at symptoms is usually sufficient. Mebendazole and albendazole are appropriate anthelminthics for the giant intestinal roundworm, *Ascaris lumbricoides,* of humans but may trigger severe adverse reactions in VLM or OLM due to *Toxocara* and should probably be avoided altogether.

As many as 95% of newborn puppies are infected even before leaving the litter.

Dogs become infected with adult *Toxocara* worms by 1) ingesting infective eggs from a contaminated environment, soil invertebrates which carry eggs or by coprophagy, 2) transplacental passage of larvae to the embryonic pups and 3) transmammary migration of larvae into sucking pups in the mother's milk. Thus, as many as 95% of newborn puppies are infected even before leaving the litter. Deworming of puppies and kittens should begin at three weeks of age, repeated three times at two week intervals and for once every six months thereafter. Effective chemotherapies for dog ascarids may include piperazine, ivermectin, pyrantel pamoate, diethylcarbamazine and fenbendazole. Partly because people are less likely to come into contact with cat feces, *T. cati* is a lesser cause of this problem. (*Toxocara* life cycle: http://www.dpd.cdc.gov/dpdx/HTML/Toxocariasis.htm)

Control of human exposure to *Toxocara* in large urban centers is based on preventing the indiscriminate deposition of dog and cat feces in play areas, sandboxes and on beaches frequented by children. Hands should be washed after

handling soil and before eating. Most pet owners are not aware that their dogs and cats may carry *Toxocara* and other parasites dangerous to other people. Veterinarians are ideally positioned to promote *Toxocara* control by recommending regular fecal examinations, providing appropriately timed anthelmintic treatments and counseling clients on appropriate precautionary measures, especially the need for prompt collection and disposal of pet feces.

Veterinarians are ideally positioned to promote Toxocara control and to counsel clients on precautionary measures, especially the need for prompt collection and disposal of pet feces.

The raccoon ascarid, *Baylisascaris procyonis*, produces a devastating and potentially fatal neural larva migrans syndrome (NLM) that usually presents as an acute meningoencephalitis of humans. Larvae increase in size during aggressive migration into the brain while secreting products that provoke intense eosinophilic inflammation and widespread necrosis. The presence of eosinophils in the CSF is a helpful clue. Neither anthelminthics nor steroids affect the outcome. Among documented survivors of this emerging infectious disease, only one has emerged neurologically intact. Infection typically occurs in young children practicing pica or geophagia or after the ingestion of infective *B. procyonis* eggs on contaminated objects. Although still sporadic and rare, human *Baylisascaris* infections are increasing in parallel with the rising peridomestic raccoon populations. Of greater concern is the appearance of egg-laying adult *B. procyonis* worms in puppies and dogs with their indiscriminate defecation habits. Raccoons by contrast typically habituate sylvan communal latrines which accumulate enormous numbers of their highly resistant eggs and serve as sources of infection to many species of smaller visiting scavengers. Human ingestion of *B. procyonis* eggs is most likely to occur during exposure to baby or pet raccoons; by indoor storage of contaminated timber, wood chips or bark for firewood; indoor contamination by raccoon dens in chimneys and fireplaces, on flat roofs, patios or in outdoor play areas. Fresh raccoon feces can be recognized by their tubular shape (7-15 cms), dark color and pungent odor.

The raccoon ascarid, Baylisascaris procyonis, produces a devastating and potentially fatal neural larva migrans syndrome (NLM).

Known endemic areas include the Midwest, northeast and west coast but *Baylisascaris* is to be expected anywhere raccoons occur. Thus, veterinarians, physicians and public health officials anticipate the appearance of zoonotic *Baylisascaris* infection elsewhere, specifically in the southeastern United States.

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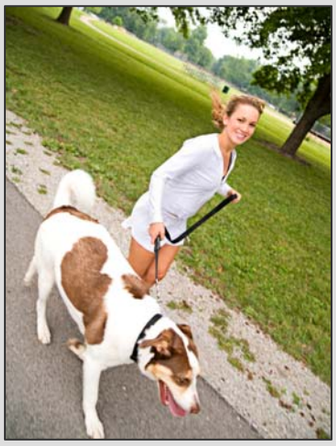
One in four dogs is overweight or obese.

The two major contributors to obesity in humans and animals are too many calories and not enough exercise.



Increasing activity can be as simple as taking your pet on a daily walk or jog; a healthy benefit for both.

Obesity epidemic occurring concurrently in people and pets

Justin Sobota and Travis Meyer

Obesity is not just an increasing problem among children; the percentage of clinically defined obese adults is also at an all time high. Globally, the WHO estimates 1.6 billion people to be overweight and more than 400 million as obese. Simultaneously, obesity is also increasing in companion animals, most commonly pet dogs and cats. Animal obesity is now an epidemic with 1 in 4 dogs now overweight or obese.

Overweight is defined with a BMI greater than 25, or more than 25 percent or 30 percent body fat for men and women, respectively. Obesity is defined as a BMI greater than 30 in adults.

Obesity in animals is not as easy to calculate, but can be visually within limits of clinical accuracy. Veterinarians and small animal nutritionists advise dogs should have a flat to concave stomach, and a thinning of their waistline when viewed from above. Cats are somewhat less likely to be obese than dogs, but still, if they look overweight, they probably are.

Animal obesity is currently the most common medical condition seen by veterinarians. The condition carries many of the same risks as it does for humans. Increased incidence of diabetes mellitus and heart disease are among the leading mortality factors that are increased with obesity, but others include hypothyroidism, hyperadrenocorticism (Cushing's disease), hypoadrenocorticism (Addison's disease), hip dysplasia, osteoarthritis, cruciate ligament and meniscal injury, osteochondrosis dessicans, pancreatitis, and kidney disease.

The two major contributors to obesity in humans and animals are too many calories and not enough exercise. No time in history has it been easier for humans as well as companion animals to obtain as many available calories for less fiscal and physical expenditure.

Although we might be able to consciously cut down on our own caloric intake, animals retain the primitive instinct to eat when food is available. Also, there are species differences. Dogs are behaviorally gorge feeders and cats are one of the world's true obligate carnivores. Human interactions with pets including giving second helpings, table scraps, or keeping food available all day coupled with a decrease in exercise initiated by owners leads to increased pounds.

Increasing activities can be as simple as taking your pet on a daily walk or jog; a healthy benefit for both. Consider weight control rations and important feeding tips offered by pet food companies and include your dog or cat in your diet plans. In all circumstances, pets do not benefit from being fed table scraps or other treats. Create weight loss goals and set rewards, such as buying them a favorite toy when they reach their goal. To accomplish this, one must weigh their pet and monitor their weight loss. Most importantly, ask your veterinarian what is

One Health Newsletter

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the appropriate type and amount of food for your animal and be realistic about how often and to what extent exercise will be employed.

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Coming Events:

- **AVMA 145th Annual Convention**
  July 19 – 22, 2008

  New Orleans Morial Convention Center
  900 Convention Center Blvd., New Orleans, LA 70130

  http://www.avmaconvention.org/

  PREVENTION for ONE HEALTH ONE MEDICINE FORUM:
  Two Day Program
  Monday, July 21 and Tuesday July 22

- **International EcoHealth Forum 2008**
  "EcoHealth: Healthy Environments, Healthy People"

  December 1– 5, 2008, Mérida, México

  www.ecohealth2008.org

Recent One Health Publications:

- The Lancet 2008; **371**:386 [Feb. 2] **"Improving vaccine coverage in Africa" using One Medicine-One Health collaboration**
  http://www.thelancet.com
- Army Medical Department Journal, July-September 2007 **Healthy Animals, Health People: Inextricably Linked**
  http://www.veterinaryservice.army.mil/corps_chief/AMEDDJournalJulySeptember2007.pdf
- The Journal of American Medicine, Volume 121, Issue 3, Pages 169-170 (March 2008). **"Teaching One Medicine, One Health**
  http://www.amjmed.com/current
- Laboratory Investigation (2008) 88, 18-26; published online November 26, 2007. **'One medicine—one pathology: are veterinary and human pathology prepared?**
  http://www.nature.com/labinvest/journal/v88/n1/full/3700695a.html
- Emerging Diseases, **One Health Initiative will unite veterinary, human medicine**: Experts urge collaboration between veterinarians, physicians in wake of emerging zoonotic diseases, potential epidemics, February, 2008
  http://www.infectiousdiseasenews.com/200802/veterinary.asp