Mammal Handling Protocol

v. 1.0 March 2006
Mammal Handling Protocol.

Persons handling mammals while conducting fieldwork incur a small but increased risk of zoonotic infection, together with the possibility of physical injury. This protocol, which is to be followed by all faculty, staff, and students involved in direct contact with mammals, provides background information on potential risks, procedures to mitigate these risks and ensure the humane treatment of mammals, and specific rules to be followed whilst handling mammals on the property of the Firestone Center or the adjacent Hacienda Baru Reserve.

Mammal studies that are limited to non-contact observation, e.g. track studies and camera-trapping, and not subject to this protocol.

RULES.

1) Faculty, staff, students and authorized visitors MAY NOT handle wild mammals without first receiving professional training. Persons currently approved to provide training are:
   Donald A. McFarlane, Professor of Biology, Pitzer College
   Keith Christenson, Wildlife Biologist, Falls Church, VA.

2) Faculty, staff and students involved in direct contact with wild mammals MUST read and sign the Mammal Handling Protocol prior to beginning work.

3) Faculty, staff and students MUST report any injuries, suspected zoonotic exposure, or animal casualties on the form provided.
Mammal Handling Protocol.

Version 1. March 2006

All wild mammals are potentially dangerous to persons coming into close contact with them, either from direct traumatic injury (bites, scratches) or from transmissible infectious diseases or parasites. Workers should err on the side of caution and assume that all captured mammals pose a risk. This risk can be substantially reduced by good personal hygiene (frequent washing of hands with soap and water) together with training in handling techniques and the use of appropriate gloves and respirators.

Principal Zoonotic Diseases of Wild Mammals.

Disease organisms of concern include but are not limited to rabies (primarily bats and carnivores), hanta viruses (rodents), leptospirosis (rodents, carnivores) plague (squirrels), leishmaniasis and tetanus. Overall, risks are extremely low but these diseases can be very serious or fatal. Center for Disease Control fact sheets on these diseases are appended.

Immunizations.

Persons working with mammals should ensure that their tetanus immunizations are up to date. Rabies vaccination is recommended; rabies vaccination is REQUIRED for persons handling bats or carnivores.

Precautions.

Gloves: leather gloves appropriate to the size of animal MUST be worn when removing mammals from live-traps. Latex gloves should be worn when cleaning traps.

Respirators: Persons handling rodents or working with (including cleaning) traps that have contained rodents MUST wear half-mask respirators with P-100 filters, and safety goggles.

Arthropod repellent: Persons working with wild mammals and traps shall apply insect repellent to exposed skin prior to commencing work. The CDC recommends the use of DEET based repellents, e.g. “OFF!” products containing 25% DEET in spray or towelette form (http://www.offprotects.com/insect-bites/)

At the completion of fieldwork, traps shall be soaked for 1 hour in a bucket of water containing 5% bleach solution prior to cleaning, drying and storage. Latex gloves should be worn and care should be taken not to damage clothing with bleach.

First Aid.

Mammal bites and scratches should be immediately cleaned with copious amounts of water and soap. Soap should be worked deep into the wound. Persons without prior rabies vaccination should seek medical attention.
Animal Care Considerations.

Persons working with wild mammals have a responsibility to the integrity of their research and to the animals to ensure that humane methods of capture and handling are used. Only mist nets, harp traps, and live-traps of cage/box design are approved for use on the property of the Firestone Center. Traps should be of a size appropriate to the target species. Leg-hold, snap traps and snares are prohibited. Live traps for nocturnal species should be opened no more than 1 hour before dusk and MUST be checked within 1 hour of dawn, irrespective of weather and other conditions – the number of traps deployed should therefore be limited by the number and energy of the persons responsible for them. Open mist nets and harp traps must be attended continuously.

Projects involving tissue collection, euthanasia, toe-clipping, banding and tagging require additional prior written approval from the Animal Care and Use Committee, (Joint Science Department, Pitzer College, Claremont, California)
Appendix 1. Rabies,

(source: Center for Disease Control, March 2006)

Rabies is a preventable viral disease of mammals most often transmitted through the bite of a rabid animal. The vast majority of rabies cases reported to the Centers for Disease Control and Prevention (CDC) each year occur in wild animals like raccoons, skunks, bats, and foxes. Domestic animals account for less than 10% of the reported rabies cases, with cats, cattle, and dogs most often reported rabid.

Rabies virus infects the central nervous system, causing encephalopathy and ultimately death. Early symptoms of rabies in humans are nonspecific, consisting of fever, headache, and general malaise. As the disease progresses, neurological symptoms appear and may include insomnia, anxiety, confusion, slight or partial paralysis, excitation, hallucinations, agitation, hypersalivation, difficulty swallowing, and hydrophobia (fear of water). Death usually occurs within days of the onset of symptoms.

Over the last 100 years, rabies in the United States has changed dramatically. More than 90% of all animal cases reported annually to CDC now occur in wildlife; before 1960 the majority were in domestic animals. The principal rabies hosts today are wild carnivores and bats. The number of rabies-related human deaths in the United States has declined from more than 100 annually at the turn of the century to one or two per year in the 1990’s. Modern day prophylaxis has proven nearly 100% successful. In the United States, human fatalities associated with rabies occur in people who fail to seek medical assistance, usually because they were unaware of their exposure.

Rabies virus causes an acute encephalitis in all warm-blooded hosts, including humans, and the outcome is almost always fatal. Although all species of mammals are susceptible to rabies virus infection, only a few species are important as reservoirs for the disease. In the United States, several distinct rabies virus variants have been identified in terrestrial mammals, including raccoons, skunks, foxes, and coyotes. In addition to these terrestrial reservoirs, several species of insectivorous bats are also reservoirs for rabies.

Transmission of rabies virus usually begins when infected saliva of a host is passed to an uninfected animal. Various routes of transmission have been documented and include contamination of mucous membranes (i.e., eyes, nose, mouth), aerosol transmission, and corneal transplantations. The most common mode of rabies virus transmission is through the bite and virus-containing saliva of an infected host.

Following primary infection, the virus enters an eclipse phase in which it cannot be easily detected within the host. This phase may last for several days or months. Investigations have shown both direct entry of virus into peripheral nerves at the site of infection and indirect entry after viral replication in nonnervous tissue (i.e., muscle cells). During the eclipse phase, the host immune defenses may confer cell-mediated immunity against viral infection because rabies virus is a good antigen. The uptake of virus into peripheral nerves is important for progressive infection to occur (see Figure, number 3).
After uptake into peripheral nerves, rabies virus is transported to the central nervous system (CNS) via retrograde axoplasmic flow. Typically this occurs via sensory and motor nerves at the initial site of infection. The incubation period (see figure, number 4) is the time from exposure to onset of clinical signs of disease. The incubation period may vary from a few days to several years, but is typically 1 to 3 months. Dissemination of virus within the CNS is rapid, and includes early involvement of limbic system neurons (see Figure, number 5). Active cerebral infection is followed by passive centrifugal spread of virus to peripheral nerves. The amplification of infection within the CNS occurs through cycles of viral replication and cell-to-cell transfer of progeny virus. Centrifugal spread of virus may lead to the invasion of highly innervated sites of various tissues, including the salivary glands. During this period of cerebral infection, the classic behavioral changes associated with rabies develop.

**Signs and symptoms**
The first symptoms of rabies may be nonspecific flu-like signs — malaise, fever, or headache, which may last for days. There may be discomfort or paresthesia at the site of exposure (bite), progressing within days to symptoms of cerebral dysfunction, anxiety, confusion, agitation, progressing to delirium, abnormal behavior, hallucinations, and insomnia. The acute period of disease typically ends after 2 to 10 days (6). Once clinical signs of rabies appear, the disease is nearly always fatal, and treatment is typically supportive. Disease prevention is entirely prophylactic and includes both passive antibody (immune globulin) and vaccine. Non-lethal exceptions are extremely rare. To date only six documented cases of human survival from clinical rabies have been reported and each included a history of either pre- or postexposure prophylaxis.

There is no treatment for rabies after symptoms of the disease appear. However, two decades ago scientists developed an extremely effective new rabies vaccine regimen that provides immunity to rabies when administered after an exposure (postexposure prophylaxis) or for protection before an exposure occurs (preexposure prophylaxis). Although rabies among humans is rare in the United States, every year an estimated 18,000 people receive rabies preexposure prophylaxis and an additional 40,000 receive postexposure prophylaxis.

**Preexposure prophylaxis**
Preexposure vaccination is recommended for persons in high-risk groups, such as veterinarians, animal handlers, and certain laboratory workers. Other persons whose activities bring them into frequent contact with rabies virus or potentially rabid bats, raccoons, skunks, cats, dogs, or other species at risk of having rabies should also be considered for preexposure prophylaxis. In addition, international travelers likely to come in contact with animals in areas of enzootic dog rabies which lack immediate access to appropriate medical care, including biologics, should be considered for preexposure prophylaxis.

**Purpose of preexposure prophylaxis**
Preexposure prophylaxis is given for several reasons. First, although preexposure vaccination does not eliminate the need for additional medical attention after a rabies
exposure, it simplifies therapy by eliminating the need for human rabies immune globulin (HRIG) and decreasing the number of vaccine doses needed – a point of particular importance for persons at high risk of being exposed to rabies in areas where immunizing products may not be available, and it minimizes adverse reactions to multiple doses of vaccine. Second, it may enhance immunity in persons whose postexposure therapy might be delayed. Finally, it may provide protection to persons with inapparent exposures to rabies.

Preexposure prophylaxis consists of three doses of rabies vaccine given on days 0, 7, and 21 or 28. Postexposure prophylaxis (PEP) is indicated for persons possibly exposed to a rabid animal. Possible exposures include animal bites, or mucous membrane contamination with infectious tissue, such as saliva. PEP should begin as soon as possible after an exposure. There have been no vaccine failures in the United States (i.e. someone developed rabies) when PEP was given promptly and appropriately after an exposure.

Administration of rabies PEP is a medical urgency, not a medical emergency. Physicians should evaluate each possible exposure to rabies and as necessary consult with local or state public health officials regarding the need for rabies prophylaxis.

**Postexposure prophylaxis regimen**
In the United States, PEP consists of a regimen of one dose of immune globulin and five doses of rabies vaccine over a 28-day period. Rabies immune globulin and the first dose of rabies vaccine should be given as soon as possible after exposure. Additional doses of rabies vaccine should be given on days 3, 7, 14, and 28 after the first vaccination. Current vaccines are relatively painless and are given in your arm, like a flu or tetanus vaccine.

**What to do after a possible exposure**
If you are exposed to a potentially rabid animal, wash the wound thoroughly with soap and water, and seek medical attention immediately. A health care provider will care for the wound and will assess the risk for rabies exposure. The following information will help your health care provider assess your risk:

- the geographic location of the incident
- the type of animal that was involved
- how the exposure occurred (provoked or unprovoked)
- the vaccination status of animal
- whether the animal can be safely captured and tested for rabies

Steps taken by the health care practitioner will depend on the circumstances of the bite. Your health care practitioner should consult state or local health departments, veterinarians, or animal control officers to make an informed assessment of the incident and to request assistance. The important factor is that you seek care promptly after you are bitten by any animal.
Appendix 2. Hanta Virus

(source: Center for Disease Control, March 2006)

**Hantavirus Pulmonary Syndrome (HPS)**

Hantavirus pulmonary syndrome (HPS) is a deadly disease transmitted by infected rodents through urine, droppings, or saliva. Humans can contract the disease when they breathe in aerosolized virus. HPS was first recognized in 1993 and has since been identified throughout the United States. Although rare, HPS is potentially deadly. Rodent control in and around the home remains the primary strategy for preventing hantavirus infection.

In the United States, deer mice (along with cotton rats and rice rats in the southeastern states and the white-footed mouse in the Northeast) carry hantaviruses that cause hantavirus pulmonary syndrome. Rodents shed the virus in their urine, droppings, and saliva. The virus is mainly transmitted to people when they breathe in air contaminated with the virus. When fresh rodent urine, droppings or nesting materials are stirred up, tiny droplets containing the virus get into the air. This process is known as "aerosolization."

There are several other ways rodents may spread hantavirus to people:

- If a rodent with the virus bites someone, the virus may be spread to that person—but this type of transmission is rare.
- Researchers believe that people may be able to get the virus if they touch something that has been contaminated with rodent urine, droppings, or saliva, and then touch their nose or mouth.
- Researchers also suspect people can become sick if they eat food contaminated by urine, droppings, or saliva from an infected rodent.

These possibilities demonstrate why disinfecting rodent-infested areas is so important in preventing transmission of the virus. Transmission can happen any place that infected rodents have infested. This could include barns, sheds, or other outbuildings, warehouses, and summer cottages that have been closed up for the season. Carrier rodents can infest homes as well. Therefore, the most sensible way to avoid contact with infected rodents is to prevent rodents from infesting the places where you live and work and to follow safety precautions if you do stumble into a rodent-infested area.

**Early symptoms**

Early symptoms include fatigue, fever and muscle aches, especially in the large muscle groups-thighs, hips, back, and sometimes shoulders. These symptoms are universal. There may also be headaches, dizziness, chills, and abdominal problems, such as nausea, vomiting, diarrhea, and abdominal pain. About half of all HPS patients experience these symptoms.
Late symptoms
Four to 10 days after the initial phase of illness, the late symptoms of HPS appear. These include coughing and shortness of breath, with the sensation of, as one survivor put it, a "...tight band around my chest and a pillow over my face" as the lungs fill with fluid. Earache, sore throat, runny nose, and rash are very uncommon symptoms of HPS.

How long after contracting the virus do symptoms appear?
Due to the small number of HPS cases, the "incubation time" is not positively known. However, on the basis of limited information, it appears that symptoms may develop between 1 and 5 weeks after exposure to urine, droppings, or saliva of infected rodents.

Another important point to remember from the data that the CDC Special Pathogens Branch keeps on all reported cases of HPS, is that it appears many people who have become ill were in a situation where they did not see rodents or rodent droppings. Other people have had frequent contact with rodents and their droppings before becoming ill. This apparent inconsistency makes it very difficult to pin down the precise time when the virus was transmitted.

Reservoir and Reservoir Distribution: United States
All hantaviruses known to cause hantavirus pulmonary syndrome (HPS) are carried by New World rats and mice of the family Muridae, subfamily Sigmodontinae. The subfamily Sigmodontinae contains at least 430 species, which are widespread in North and South America. The rodent hosts of HPS are generally not associated with urban environments, although some species, including the deer mouse, Peromyscus maniculatus, and white-footed mouse, Peromyscus leucopus, will enter human habitation in rural and suburban areas.

Several hantaviruses that are pathogenic for humans have been identified in the United States. In general, each virus has a single primary rodent host. Other small mammals can be infected as well but are much less likely to transmit the virus to other animals or humans. The deer mouse is the host for Sin Nombre virus (SNV), the primary causative agent of HPS in the United States. The deer mouse is common and widespread in rural areas throughout much of the United States. Although prevalence varies temporally and geographically, on average about 10% of deer mice tested throughout the range of the species show evidence of infection with SNV.

Other hantaviruses associated with sigmodontine rodents and known to cause HPS include New York virus, which is hosted by the white-footed mouse; Black Creek Canal virus, which is hosted by the cotton rat, Sigmodon hispidus; and Bayou virus, which is hosted by the rice rat, Oryzomys palustris. Nearly the entire continental United States falls within the range of one or more of these host species. Several other sigmodontine rodent species in the United States are associated with additional hantaviruses that have yet to be implicated in human disease.

Recent studies have confirmed that infected deer mice are present in every habitat type--from desert to alpine tundra, although the prevalence of infection is higher in
certain middle-altitude habitats. Surveys of rodents throughout the United States suggest that SNV is distributed in all locations where P. maniculatus is found. Related hantaviruses are also found throughout the geographic range of their rodent carriers. Given that P. maniculatus and P. leucopus are commonly found in the peridomestic setting and typically have higher population densities than other rodents, cases of HPS can be expected to occur throughout the range of these rodent species. Other implicated species, such as S. hispidus and O. palustris, generally do not live in such close proximity to human habitats, and this factor may decrease the probability of human exposure to viruses shed by these rodents. Lower population density, a lesser propensity for peridomestic encroachment and a narrower geographic and ecologic distribution (and perhaps differing virulence) may explain the lack of human disease associated with hantaviruses (or genetic sequences thought to represent additional hantaviruses) from meadow and California voles (Microtus pennsylvanicus and californicus, respectively), the western harvest mouse (Reithrodontomys megalotis), and the brush mouse (Peromyscus boylii).

Reservoir Distribution Outside the United States

HPS is more common in South America than in North America. Cases have been identified in Argentina, Chile, Uruguay, Paraguay, Brazil, and Bolivia. Andes virus causes HPS in Argentina and Chile and is the only hantavirus known to have been transmitted from person to person. Andes, Bermejo, Hu39694, Lechiguanas, Maciel, Oran, and Pergamino viruses have been linked to HPS cases in Argentina. Bermejo and Laguna Negra virus cause HPS in Bolivia, and Laguna Negra virus is also linked to HPS in Paraguay. Araraquara, Castelo dos Sonhos, and Juquitiba viruses have been associated with HPS in Brazil.

In 1999, an outbreak in Panama marked the first cases of HPS identified in Central America. This outbreak led to the identification of another hantavirus, Choclo virus, which is associated with the rodent host Oligoryzomys fulvescens. The broad geographic distribution of sigmodontine rodents suggests that human cases of HPS will eventually be identified from all countries in the Americas.

Numerous other New World hantaviruses associated with sigmodontine rodents have been identified by molecular methods, but so far, few of them have not been linked to human illness. It is likely that HPS does not occur in the Old World and is confined to the New World distribution of Sigmodontine rodents.

Infection in the Host

Hantaviruses do not cause overt illness in their reservoir hosts. Although infected rodents shed virus in saliva, urine, and feces for many weeks or months or for life, the quantity of virus shed can be at its greatest approximately 3--8 weeks after infection. Field data suggest that transmission in host populations occurs horizontally and that this occurs more frequently among male than female rodents. Transmission from rodent to rodent is believed to occur primarily after weaning and through physical contact, perhaps through aggressive behavior, such as fighting. Studies of the genomic sequences indicate
that the virus has probably evolved concurrently with its rodent host over a long period of time.

Occasional evidence of infection (antibody) is found in numerous other species of rodents and their predators (e.g., dogs, cats, and coyotes), indicating that many (perhaps any) mammal species coming into contact with an infected host might become infected. No evidence supports the transmission of infection to other animals or to humans from these "dead-end" hosts. However, domestic cats and dogs may bring infected rodents into contact with humans.

Ticks, fleas, mosquitoes, and other biting insects have not been implicated in the transmission of HPS. Nevertheless, species of Peromyscus in the western United States are susceptible to infection with the plague bacterium (Yersinia pestis), and may act as hosts for plague-carrying fleas. Control of rodents without concurrent control of fleas might therefore increase the risk of human plague as the rodent fleas seek an alternative food source.

Transmission

Human infection occurs most commonly through the inhalation of infectious aerosolized saliva or excreta. Persons visiting laboratories where infected rodents were housed have been infected after only a few minutes of exposure to animal holding areas. Transmission can occur when dried materials contaminated by rodent excreta are disturbed and inhaled, directly introduced into broken skin or conjunctiva, or possibly, when ingested in contaminated food or water. Persons have also acquired HPS after being bitten by rodents. High risk of exposure has been associated with entering or cleaning rodent-infested structures.

Person-to-person transmission has not been associated with HPS cases in the United States. However, person-to-person transmission, including nosocomial transmission of Andes virus, was well documented for a single outbreak in southern Argentina, and it was suspected to have occurred much less extensively in another outbreak in Chile that was associated with the same virus. Therefore, universal precautions are recommended for healthcare workers treating HPS patients.
Appendix 3. Leptospirosis

(source: Center for Disease Control, March 2006)

**Clinical Features** Symptoms include fever, headache, chills, muscle aches, vomiting, jaundice, anemia, and sometimes a rash. The incubation period usually is 7 days, with a range of 2-29 days. If not treated, the patient could develop kidney damage, meningitis, liver failure, and respiratory distress. In rare cases, death occurs. **Etiologic Agent** Leptospires are long, thin motile spirochetes. They may be free-living or associated with animal hosts and survive well in fresh water, soil, and mud in tropical areas. Organisms are antigenically complex, with over 200 known pathogenic serologic variants. Molecular taxonomic studies at CDC and elsewhere have identified 13 named and 4 unnamed species of pathogenic leptospires. Although certain geographic regions contain specific leptospiral serovars and species, the serologic characterization of an isolate is not an absolute predictor of its species designation. **Incidence** Estimated that 100-200 cases are identified annually in the United States with about 50% of cases occurring in Hawaii. However, leptospirosis is no longer a reportable disease in the United States. Although incidence in the United States is relatively low, leptospirosis is considered to be the most widespread zoonotic disease in the world. **Sequelae** Clinical course is highly variable. The serious icteric form (Weil's disease) is not common, but hemorrhage, hepatomegaly, pulmonary hemorrhage, ARDS, and jaundice are among the severe features. Case fatality rate is 1 to 5%. **Transmission** Occurs through direct or indirect transmission from a mammalian host. Indirect transmission via contact with Leptospira contaminated water or soil, is thought to be responsible for most cases. **Risk Groups** Workers in rice fields, sugar cane plantations, mines, sewer systems, and slaughterhouses; animal caretakers and veterinarians; and travelers to tropical parts of the world involved in recreational activities in fresh water. Recreational exposures can include rafting, kayaking, and swimming, in tropical and temperate climates. **Surveillance** Currently not reportable nationally, leptospirosis is reported in numerous states (including Hawaii). To determine the incidence of leptospirosis in high-risk areas, active surveillance will be required. **Trends** Leptospirosis continues to re-emerge as a notable source of morbidity and mortality in the Western Hemisphere. The largest recorded outbreak in the continental United States (110 cases in a group of 775 exposed persons who participated in triathlons, which included swimming in a lake) occurred in June and July 1998. Significant increases in incidence were also reported from Peru and Ecuador following heavy rainfall and flooding in the spring of 1998. Thailand has also reported a rapid increase in incidence between 1995 and 2000.
Appendix 4. Plague

(source: Center for Disease Control, March 2006)

Plague is an infectious disease of animals and humans caused by a bacterium named *Yersinia pestis*. People usually get plague from being bitten by a rodent flea that is carrying the plague bacterium or by handling an infected animal. Millions of people in Europe died from plague in the Middle Ages, when human homes and places of work were inhabited by flea-infested rats. Today, modern antibiotics are effective against plague, but if an infected person is not treated promptly, the disease is likely to cause illness or death.

**Risk:** Wild rodents in certain areas around the world are infected with plague. Outbreaks in people still occur in rural communities or in cities. They are usually associated with infected rats and rat fleas that live in the home. In the United States, the last urban plague epidemic occurred in Los Angeles in 1924-25. Since then, human plague in the United States has occurred as mostly scattered cases in rural areas (an average of 10 to 15 persons each year). Globally, the World Health Organization reports 1,000 to 3,000 cases of plague every year. In North America, plague is found in certain animals and their fleas from the Pacific Coast to the Great Plains, and from southwestern Canada to Mexico. Most human cases in the United States occur in two regions: 1) northern New Mexico, northern Arizona, and southern Colorado; and 2) California, southern Oregon, and far western Nevada. Plague also exists in Africa, Asia, and South America (see map).

**Transmission:** Plague is usually transmitted to humans by the bites of infected rodent fleas. During rodent plague outbreaks, many animals die and their hungry fleas seek other sources of blood to survive. Persons and animals that visit places where rodents have recently died from plague risk getting the disease from flea bites. Persons also can become directly infected through handling infected rodents, rabbits, or wild carnivores that prey on these animals, when plague bacteria enter through breaks in the person's skin. House cats also are susceptible to plague. Infected cats become sick and may directly transmit plague to persons who handle or care for them. Also, dogs and cats may bring plague-infected fleas into the home. Inhaling droplets expelled by the coughing of a plague-infected person or animal (especially house cats) can result in plague of the lungs (plague pneumonia). Transmission of plague pneumonia from person to person is uncommon but sometimes results in dangerous epidemics that can quickly spread.
Appendix 5. Leishmaniasis.

(source: Center for Disease Control, March 2006)

Leishmaniasis is a vector-borne disease that is transmitted by sandflies and caused by obligate intracellular protozoa of the genus *Leishmania*. Human infection is caused by about 21 of 30 species that infect mammals. These include the *L. donovani* complex with 3 species (*L. donovani*, *L. infantum*, and *L. chagasi*); the *L. mexicana* complex with 3 main species (*L. mexicana*, *L. amazonensis*, and *L. venezuelensis*); *L. tropica*; *L. major*; *L. aethiopica*; and the subgenus *Viannia* with 4 main species (*L. (V.) braziliensis*, *L. (V.) guyanensis*, *L. (V.) panamensis*, and *L. (V.) peruviana*). The different species are morphologically indistinguishable, but they can be differentiated by isoenzyme analysis, molecular methods, or monoclonal antibodies.

Leishmaniasis is transmitted by the bite of female phlebotomine sandflies. The sandflies inject the infective stage, promastigotes, during blood meals. Promastigotes that reach the puncture wound are phagocytized by macrophages and transform into amastigotes. Amastigotes multiply in infected cells and affect different tissues, depending in part on the *Leishmania* species. This originates the clinical manifestations of leishmaniasis. Sandflies become infected during blood meals on an infected host when they ingest macrophages infected with amastigotes). In the sandfly's midgut, the parasites differentiate into promastigotes, which multiply and migrate to the proboscis.

Leishmaniasis is found in parts of about 88 countries. Approximately 350 million people live in these areas. Most of the affected countries are in the tropics and subtropics. The settings in which leishmaniasis is found range from rain forests in Central and South America to deserts in West Asia. More than 90 percent of the world's cases of visceral leishmaniasis are in India, Bangladesh, Nepal, Sudan, and Brazil.

Leishmaniasis is found in Mexico, Central America, and South America—from northern Argentina to southern Texas (not in Uruguay, Chile, or Canada), southern Europe (leishmaniasis is not common in travelers to southern Europe), Asia (not Southeast Asia), the Middle East, and Africa (particularly East and North Africa, with some cases elsewhere).

Human leishmanial infections can result in 2 main forms of disease, cutaneous leishmaniasis and visceral leishmaniasis (kala-azar). The factors determining the form of disease include leishmanial species, geographic location, and immune response of the host. Cutaneous leishmaniasis is characterized by one or more cutaneous lesions on areas where sandflies have fed. Persons who have cutaneous leishmaniasis have one or more sores on their skin. The sores can change in size and appearance over time. They often end up looking somewhat like a volcano, with a raised edge and central crater. A scab covers some sores. The sores can be painless or painful. Some people have swollen glands near the sores (for example, in the armpit if the sores are on the arm or hand).
Persons who have visceral leishmaniasis usually have fever, weight loss, and an enlarged spleen and liver (usually the spleen is bigger than the liver). Some patients have swollen glands. Certain blood tests are abnormal. For example, patients usually have low blood counts, including a low red blood cell count (anemia), low white blood cell count, and low platelet count. Some patients develop post kala-azar dermal leishmaniasis. Visceral leishmaniasis is becoming an important opportunistic infection in areas where it coexists with HIV.

Physicians may consult CDC to obtain information on how to treat leishmaniasis. The drug sodium stibogluconate is available under an Investigational New Drug protocol from the CDC Drug Service.
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   Donald A. McFarlane, Professor of Biology, Pitzer College
   Keith Christenson, Wildlife Biologist, Falls Church, VA.

5) Faculty, staff and students involved in direct contact with wild mammals MUST read and sign the Mammal Handling Protocol prior to beginning work.

6) Faculty, staff and students MUST report any injuries, suspected zoonotic exposure, or animal casualties on the form provided.

I have read and understand the Pitzer College SA/Firestone Center Mammal Handling Protocol. I understand the nature of the hazards associated with handling wild mammals. I have received training in handling wild mammals, I have had the opportunity to ask questions, and I have received adequate answers to any such questions.

Signed: ___________________________  Date: __________________
Printed Name: ____________________________________________
Mammal Contact Injury Form

Name: ______________________________________________________

Date of Incident: ____________________________

Description of incident: __________________________________________________
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Treatment Sought:
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Signed: _________________________________ Date: __________

Submit form to Carol_Brandt@pitzer.edu without delay. (Fax: [01] 909 621 0518)