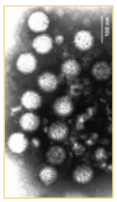
Slide 1	African Horse Sickness *Perdesiekte, Pestis Equorum, La Peste Equina, Peste Equina Africana*	African horse sickness (AHS) is also known as Perdesiekte, Pestis Equorum, La Peste Equina, and Peste Equina Africana. It is a serious viral infection that affects members of the Equidae family. The disease is spread by arthropod vectors (primarily *Culicoides* species– biting midges), with mortality in horses as high as 95%. Currently the disease is endemic in Africa but potential arthropod vectors for the disease may exist in the United States. AHS is considered as one of the most lethal of horse diseases and has therefore been classified as an OIE List A disease.
Slide 2	**Overview** • Organism • Economic Impact • Epidemiology • Transmission • Clinical Signs • Diagnosis and Treatment • Prevention and Control • Actions to take  Center for Food Security and Public Health Iowa State University - 2004	In today's presentation we will cover information regarding the organism that causes African horse sickness and the epidemiology of the disease. We will also talk about the economic impact the disease has had in the past and could have in the future. Additionally, we will talk about how it is transmitted, the species it affects, the clinical signs and necropsy findings, as well as the diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease as well as actions to take if AHS is suspected.
Slide 3	The Organism	
Slide 4	**African Horse Sickness** • Virus – Double stranded RNA – Family Reoviridae ▪ Genus Orbivirus – Arthropod-borne – Viscerotropic – Inactivated by low pH  Center for Food Security and Public Health Iowa State University - 2004	African horse sickness (AHS) is caused by a double-stranded RNA virus (Family Reoviridae: Genus Orbivirus). There are nine serotypes of this virus, which are viscerotropic (having a predilection for the abdominal and thoracic viscera). The AHS virus is relatively heat stable, particularly in the presence of protein. It is readily inactivated at pH values lower than 6.3, but is relatively stable between values ranging from 6.5 to 8.5. NOTE: AHS virus is comparable in morphology and molecular structure to bluetongue virus (which is considered the prototype virus of the genus Orbivirus). Photo is electron micrograph of Reoviridae from The National Center for Biotechnology Information at http://www.ncbi.nlm.nih.gov/ICTVdb/ICTVdB/60000000.htm.
Slide 5	Importance	

Slide 6

History

- 1780-1918: South Africa 7 epizootics
- Endemic in sub-Saharan Africa
- 1959-61: Middle East
  - 1st outbreak out of endemic Africa
- 1965-66: Morocco, Algeria, Spain
- 1987-91: Spain, Portugal
  - Imported zebra reservoirs
  - New *Culicoides* species
- 1989-91: Algeria, Morocco

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AHS was first described by Theiler in 1921, when he related seven major epizootics of AHS in South Africa between 1780-1918. Currently the disease is endemic in sub-Saharan Africa occurring annually or seasonally wherever susceptible Equidae are present. About every 20 years, major outbreaks occur in southern Africa and occasionally northern Africa. In 1959-61, the first documented outbreak of AHS out of its traditional enzootic region of Africa occurred in the Middle East (Israel, Iran, Pakistan, Afghanistan, India, Turkey, Iraq, Syria, Lebanon, Jordan, and Cyprus). During this outbreak, as many as 300,000 animals died or were destroyed. India reported a 90% mortality in Equidae involved. Another outbreak of AHS occurred in 1965-66 in the North African countries of Morocco and Algeria and also in Spain. Spain had another outbreak of AHS in 1987 (which later spread to Portugal). It is suspected that the disease reached the country via zebras imported from Namibia, Africa, which were subclinically infected with the virus. [Zebras show no clinical signs when infected with the virus but can have viremia (and therefore be infectious for the vector) for as long as 6 weeks!]. During this outbreak, the disease was also effectively spread by a non-traditional *Culicoides* species. This led to an increase in the list of potential vectors capable of transmitting the disease. Recent outbreaks of AHS have occurred in Algeria (1989) and Morocco (1989-91). However the disease has not occurred in these countries since 1991.

Slide 7

Economic Impact

- 1989: Spain and Portugal
  - 137 outbreaks - 104 farms
  - 206 equines died or destroyed
  - 170,000 equines vaccinated
    - 82 of vaccinated equines died
  - Eradication program cost $1.9 million
- U.S. Horse Industry (1998)
  - Value of sales from equine: $1.75 billion
  - Equine inventory:5.25 million horses
  - Equine sold: 558,000
- U.S. has arthropod vectors for AHS

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Following the 1989 outbreak, Portugal initiated an extensive vaccination and slaughter campaign. Of the 137 outbreaks (on 104 farms), 206 equines died (14%) or were destroyed (16%). Although an estimated 170,000 equines were vaccinated, 82 of the vaccinated equines died or were destroyed. One year after the program (December 1991), Portugal was declared free of AHS. Their estimated cost of the eradication was US$1.9 million.

As of 1998, the U.S. equine industry had an inventory of 5.25 million horses with 558,000 being sold that same year. The value of those sales were $1.75 billion (USDA, National Agricultural Statistics Service). In 1995, approximately seven million Americans were involved in the industry (i.e., horse owners, service providers, employees or volunteers). Since the disease has never occurred in the Western Hemisphere, our Equidae species are naïve and highly susceptible to the virus. The U.S. has arthropod vectors potentially able to transmit African horse sickness. Additionally, in the event of an outbreak of AHS in the U.S., an emergency disease declaration and immediate ban on the movement or trade of all Equidae species would be enacted. These factors demonstrate the potential economic impact this disease could have if introduced into the U.S.

Slide 8

Epidemiology

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Slide 9

Geographic Distribution

- Endemic in sub-Saharan Africa
- Outbreaks
  - Southern and Northern Africa
  - Near and Middle East
  - Spain and Portugal
- Peak: Late summer - early autumn
- Prevalence influenced by climate

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AHS is endemic in the central tropical region of Africa (sub-Saharan central and east Africa). This disease often spreads to epizootics in southern Africa and occasionally in northern Africa (Morocco and Algeria). Outbreaks have been seen in the Near and Middle East (1959-63), Spain (1966, 1987-90) and Portugal (1989). The peak incidence of disease occurs in the late summer and early autumn. Its prevalence being directly influenced by climatic conditions which favor insect breeding. Outbreaks are abruptly curtailed by severe frost. Spread of the disease may also occur through the importation of subclinically affected Equidae (i.e., zebras), which can serve as a reservoir for potential vector species. It has also been suggested that dispersal of vectors may occur by traveling on prevailing winds (a distance up to 700 km). Epizootics of AHS outside the enzootic sub-Saharan zone does not appear to be maintained for more than 2-3 consecutive years. Factors such as the absence of a long-term vertebrate reservoir, reduced prevalence and seasonal incidence of vectors and efficient control measures (vaccination and vector abatement) may play a role in preventing the disease from becoming endemic in these areas. The map depicts the present incidence and suspected incidence of African Horse Sickness.

Slide 10

Morbidity/Mortality

- Varies with exposure, species, immunity
- Horses: Mortality between 50-95%
  - Cardiac form 50-70%; Mixed form 80%
  - Pulmonary form always fatal
- Other Equidae
  - Mules: 50%
  - European or Asian donkeys: 5-10%
  - None in African donkeys and zebras

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Morbidity and mortality from AHS is variable and depends on factors such as exposure to the infected *Culicoides* species, virus serotype, as well as the Equidae species involved and their level of immunity. Horses are particularly susceptible to the disease with mortality ranging between 50-95%, depending on the form of the disease. The cardiac form of the disease results in a 50-70% morbidity rate. In the mixed form, morbidity is greater than 80%. The pulmonary form is almost always fatal. The horsesickness fever form rarely results in death. Horses that recover from an infection have a lifetime immunity to the particular causative viral serotype. Foals can acquire passive immunity from immunized mares. Other species of Equidae can also be affected, but disease is generally less severe. The mortality rate in mules is about 50 percent and in the European and Asian donkey about 5-10 percent. No mortality has been observed among African donkeys and zebras.

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Transmission

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Slide 12

Animal Transmission

- Not contagious
- Spread by arthropod vector
  - Biting midges: *Culicoides imicola; C. bolitinos; C. variipennis*
  - Other potential vectors: Mosquitoes, biting flies, ticks
- Viremia in Equidae
  - Horses: 12-40 days
  - Zebras, African donkeys: up to 6 weeks

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African horse sickness is not a contagious disease. It does not spread directly from horse to horse. The virus is transmitted by the arthropod-vector *Culicoides* (biting midges). The most common vector species reported are *Culicoides imicola* (most important vector) and *C. bolitinos*, however other *Culicoides* species (i.e., *C. variipennis*, which is common in many parts of the United States), should also be considered a potential vector. Transmission of AHS by other arthropods is thought to be a very minor source of infection. Mosquitoes may be potential biological vectors, while biting flies (i.e., *Stomoxys* and *Tabanus*) may potentially serve as mechanical vectors. Experimentally, some tick species (*Hyalomma dromadarii* and *Rhipicephalus sanguineus sanguineus*) were found able to transmit the virus from infected dogs and horses to healthy dogs and horses. (Although dogs have been experimentally infected, they are not thought to spread the infection naturally). Viremia developed in Equidae species is high enough to infect competent vectors, however, the length of viremia varies with Equidae species, lasting 12-40 days in horses and up to 6 weeks in zebras and African donkeys.

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Culicoides spp.

- Biting midges, "punkies", "no-see-ums"
- Extremely small ~1/8"
- Distinct wing pattern
- Only females bite
- Greatest biting activity around dawn and dusk

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Culicoides species (Family Ceratopogonidae), are also called biting midges, "punkies" and "no-see-ums". They are extremely small arthropods, typically measuring less than 1/8 inch with have a distinctive wing pattern. Only females feed on blood with the greatest time of biting activity occurring near dusk and around dawn. Environmental temperature and moisture are the main factors determining their prevalence in the environment. Eggs are usually deposited in masses of 25-300 in water and hatch in about 3 days at 80^{o}F. Margins of streams and lakes, mud holes, tree holes, salt marshes, tide pools swamps, rice fields and runoff from dairy and feedlot pens are all habitats for *Culicoides* spp. Photo: A culicoides biting midge courtesy of www.defra.gov.uk

Slide 14

Animals and African Horse Sickness

Slide 15

Clinical Signs

- Incubation period: 2-14 days
  - Clinical signs typically seen 5-7 days
- Four forms of the disease
  - Pulmonary (peracute)
  - Cardiac (subacute edematous)
  - Mixed (acute)
  - Horsesickness fever

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The incubation period for AHS can range from 2-14 days. Clinical signs typically appear 5 to 7 days after infection. They are characterized by damage to the respiratory and circulatory system as a result of increased vascular permeability. There are four different forms of the disease: the peracute or pulmonary form, the subacute edematous or cardiac form, the acute or mixed form and horsesickness fever.

Slide 16

Pulmonary (Peracute) Form

- Acute fever
- Sudden, severe respiratory distress
- Dyspnea and tachypnea
- Profuse sweating
- Spasmodic coughing
- Frothy serofibrinous nasal exudate
- Rapid death

Foam from the nares due to pulmonary edema.

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Clinical signs seen with the pulmonary (peracute) form of AHS include an acute fever (104-106 ^{o}F), followed by the sudden onset of severe respiratory distress. This form of the disease has a shorter incubation period of 3-5 days (vs. 5-7 days). Infected animals may show signs of dyspnea, such as standing with their forelegs spread apart, their head extended and nostrils fully dilated. Other clinical signs may include tachypnea, forced expiration, profuse sweating, spasmodic coughing, and a frothy serofibrinous nasal exudate. The disease usually progresses rapidly and the animal often dies within a few hours after the respiratory signs appear. Mortality is near 100% for this form of AHS. Photos courtesy of USAHA 'The Grey Book'.

Slide 17

Cardiac (Subacute) Form

- Edema
  - Supraorbital fossae, eyelids, intermandibular space
  - Neck, thorax, brisket and shoulders
- Terminal stages
  - Petechiae: Ventral tongue, conjunctiva
- Death within 1 week

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Clinical signs for the cardiac form of AHS include a fever (102-106^{o}F) which lasts for 3 to 6 days. As the fever starts to subside, edematous swellings appear in the supraorbital fossae (top photo), palpebral conjunctivae (bottom photo) and intermandibular space. Edema then spreads to involve the cheeks, lips, tongue, laryngeal region and sometimes the neck, shoulders and chest. Typically no edema is seen in the lower limbs. If the animal recovers, the swellings gradually subside over the next 3 to 8 days. Clinical signs seen in the terminal stage of the disease can include severe depression, colic, and petechiae under the ventral surface of the tongue and in the conjunctivae. Death is typically due to cardiac failure. The mortality rate in horses with the cardiac form of AHS ranges from 50-70%, with death occurring 4-8 days after onset of clinical signs. Photos courtesy of USAHA 'The Grey Book'.

Slide 18

Mixed (Acute) Form

- Pulmonary and cardiac forms
- Cardiac signs usually subclinical
  - Followed by severe respiratory distress
- Mild respiratory signs
  - Followed by edema and death
- Diagnosed by necropsy

The mixed form of AHS involves concurrent symptoms of both the pulmonary and cardiac forms of the disease. Typically the cardiac signs are subclinical and are then followed by severe respiratory distress. Occasionally, mild respiratory signs may be followed by edema and death from cardiac failure. The mixed form of the disease is rarely diagnosed clinically, but is often seen at necropsy in horses and mules. Incubation and mortality rates vary with the mixed form.

Slide 19

Horsesickness Fever

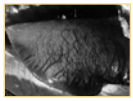
- Mild clinical signs
- Characteristic fever (3-8 days)
  - Morning remission (undetectable)
  - Afternoon exacerbation
- Other signs
  - Mild anorexia or depression
  - Congested mucous membranes
  - Increased heart rate
- This form is rarely fatal

Clinical signs of horsesickness fever are typically mild. The fever usually lasts for 3 to 8 days, rarely exceeds 104 ^{o}F, and is characterized by morning remissions and afternoon exacerbations. Other signs are also mild and may include anorexia or depression, congested mucous membranes, and an increased heart rate. This form of the disease is rarely fatal.

Slide 20

Post Mortem Lesions

- Pulmonary form
  - Hydrothorax
  - Severe pulmonary edema
- Cardiac form
  - Yellow gelatinous infiltrate
    - Fascia of head, neck, shoulders
  - Hydropericardium
- Mixed form
  - Mixture of above findings



Excessive fluid in the thoracic cavity and pulmonary edema; note the distended interlobular septa.

In the pulmonary form of AHS, the predominant post mortem lesion seen is severe diffuse pulmonary edema, sometimes associated with pleural effusion (see photo). Hydrothorax can also be common. The thoracic and abdominal lymph nodes are usually enlarged and edematous. In the cardiac form, a yellow gelatinous infiltrate can be seen in the subcutaneous and intermuscular fascia of the head, neck, shoulders and occasionally the brisket, ventral abdomen and rump. Hydropericardium is a consistent finding, however the lungs are usually normal or may be slightly engorged. The thoracic cavity rarely contains excess fluid. The mixed form of AHS is generally diagnosed at necropsy and is characterized by the both pulmonary and subcutaneous edema, pleural and pericardial effusion with typical cardiac hemorrhages. No gross or microscopic lesions have been reported for the horsesickness fever form of AHS. All affected horses recover. Photos courtesy of USAHA 'The Grey Book'.

Slide 21

Differential Diagnosis

- Anthrax
- Equine encephalosis
- Equine viral arteritis
- Equine infectious anemia
- Equine morbillivirus pneumonia
- Purpura hemorrhagica
- Equine piroplasmosis

Differentials for African horse sickness include anthrax, equine encephalosis, equine viral arteritis, equine infectious anemia, equine morbillivirus pneumonia, purpura hemorrhagica and equine piroplasmosis.

Slide 22

Sampling

- Before collecting or sending any samples, the proper authorities should be contacted
- Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease

Before collecting or sending any samples from animals with a suspected foreign animal disease, the proper authorities should be contacted. Samples should only be sent under secure conditions and to authorized laboratories to prevent the spread of the disease.

Slide 23

Diagnosis and Treatment

- Clinical signs
  - Supraorbital swelling is characteristic
  - History
- Laboratory diagnosis
  - Virus isolation & identification
  - Serology (tentative)
  - Necropsy: spleen, lung, lymph node
- No efficient treatment

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African horse sickness should be suspected in Equidae demonstrating the previously mentioned clinical signs. Supraorbital swellings are particularly characteristic of this disease. Additionally, a history of prevalence or exposure to competent vectors or of travel from an enzootic area can be important factors. Tentative diagnosis of AHS may be obtained by serology, if blood is taken during the febrile stage. However, laboratory confirmation is essential for definitive diagnosis and serotype determination will be important for control measures. Virus isolation and identification may be done from a number of tissues; samples taken should include small (2-4 g) sections of the spleen, lung and lymph nodes. Samples for virus isolation should be stored and transported at 4^{o} C. Currently there is no efficient treatment for African horse sickness. Surviving Equidae develop solid immunity to the homologous serotype but remain susceptible to heterologous serotypes. Vaccines have been developed for all 9 serotypes of the virus but are not currently available in the U.S.

Slide 24

AHS and Other Species

- Dogs
  - Experimentally
  - Ingestion of infected horse meat
  - Not usually by insect bites
  - No role in spread or maintenance
- Camels, Zebras
  - Inapparent infection

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Experimentally, dogs have been found to be susceptible to the AHS virus. Infection typically occurs following the consumption of virus-infected horse meat. Canines are not thought to become naturally infected with AHS through vector bites and are also not considered important in the spread or maintenance of the virus. It is generally accepted that dogs play no role in the spread or maintenance of AHS. Camels and zebras can be inapparently infected with AHS virus.

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African Horse Sickness in Humans

Slide 26

AHS in Humans

- No natural infection in humans
- Transnasal infection with certain neurotropic vaccine strains
  - Encephalitis
  - Retinitis

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There is no evidence that humans can become infected with field strains of AHS virus, either through contact with infected animals or from working in laboratories. However, it has been shown that certain neurotropic vaccine strains may cause encephalitis and retinitis in humans following transnasal infection. **Modified live vaccine strains of AHS should be handled with caution**.

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Prevention and Control

Slide 28

Recommended Actions

- Notification of Authorities
  - Federal:
    Area Veterinarian in Charge (AVIC)
    www.aphis.usda.gov/vs/area_offices.htm
  - State veterinarian
    www.aphis.usda.gov/vs/sregs/official.htm
- Quarantine

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If you suspect a case or outbreak of AHS, contact your state and/or federal veterinarian immediately. If AHS is detected in a non-endemic country, a strict quarantine zone should be established.

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Disinfection

- Inactivation of virus
  - Formalin, β-propiolactone, acetyl-ethyleneimine derivatives
  - Radiation
- Disinfectants
  - Sodium hypochlorite (bleach)
- Killed
  - pH less than 6
  - pH greater than 12

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The AHS virus can be inactivated by formalin, -propiolactone, acetyl-ethyleneimine derivatives or radiation. Sodium hypochlorite (bleach) is an effective disinfectant against the virus. The virus is also destroyed at a pH less than 6 or greater than 12.

Slide 30

Prevention

- Quarantine
  - Equidae from endemic areas
    - Asia, Africa and Mediterranean
  - Minimum 60 days at point of entry
- Vaccination
  - In infected areas
  - Surrounding protection zone
  - Not available in the U.S.

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Current efforts to prevent the introduction of AHS into the U.S. include a mandatory 60-day (minimum) quarantine in an insect-proof facility of all Equidae from endemic countries (Asia, Africa and the Mediterranean) at the point of entry. In endemic areas, vaccination is strongly recommended for susceptible Equidae. Additionally, areas around the affected area should vaccinate as well to produce a surrounding protection zone. Vaccines have been developed for all 9 serotypes of the virus but are not currently available in the U.S.

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Control

- Vector control and protection
  - Insect repellants
  - Stable in insect-proof housing from dusk to dawn
- Monitor temperature of all Equidae
- Euthanize or isolate febrile Equidae
  - In insect-free stable until cause is determined
- Vaccination

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The main control measures for AHS include arthropod vector control, vaccination of susceptible animals, (preferably with the monovalent vaccine for the outbreak serotype) and euthanasia or isolation (in vector proof facilities) of Equidae showing clinical signs. Vector control measures for AHS should include spraying all Equidae with insect repellants and, at a minimum, stable them from dusk to dawn. Insecticides of the pyrethrin group have shown adequate efficacy against AHS vectors. Each susceptible animal should have its temperature taken regularly (optimally, twice daily). Animals that develop a fever should either by euthanized or isolated in an insect-free stable until the cause of the fever has been established. In the event of a large-scale outbreak, all affected Equidae should be eliminated immediately. Noninfected Equidae should be vaccinated with the monovalent vaccine specific for the viral serotype of the outbreak.

Slide 32

Vaccination

- Attenuated live vaccine available for horses, mules and donkeys
- Recovering animals
  - Lifelong immunity to that serotype
- OIE International Animal Health Code
  - All AHS vaccinated Equidae must be permanently marked at time of vaccination

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Vaccines have been developed for the 9 serotypes of AHS virus. Attenuated (monovalent and polyvalent) live (Vero cell) vaccines for use in horses, mules and donkeys, are currently available in some countries (but not the U.S. at this time). New vaccines, including a subunit vaccine, have been evaluated experimentally. Once a diagnosis of AHS has been confirmed, vaccination of all Equidae should be considered. Animals that recover from the disease develop solid life-long immunity against the infecting viral serotype. The OIE International Animal Health Code (2002), requires that all AHS vaccinated Equidae in an infected zone must be clearly identified with a permanent mark at the time of vaccination.

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Additional Resources

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Additional Resources

- World Organization for Animal Health (OIE) International Animal Health Code
  - www.oie.int
- USAHA Foreign Animal Diseases – "The Gray Book"
  - www.vet.uga.edu/vpp/gray_book
- USDA – APHIS
  - www.aphis.usda.gov/vs/ep/fad_training/bibpage.htm

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Acknowledgments

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Acknowledgments

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