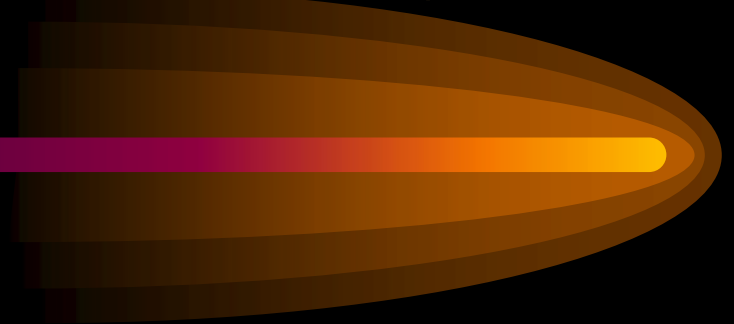


West Nile Fever

West Nile Virus



What is West Nile Fever?

- **West Nile Virus (WNV)**

First isolated from a woman in the West Nile district of Uganda in 1937

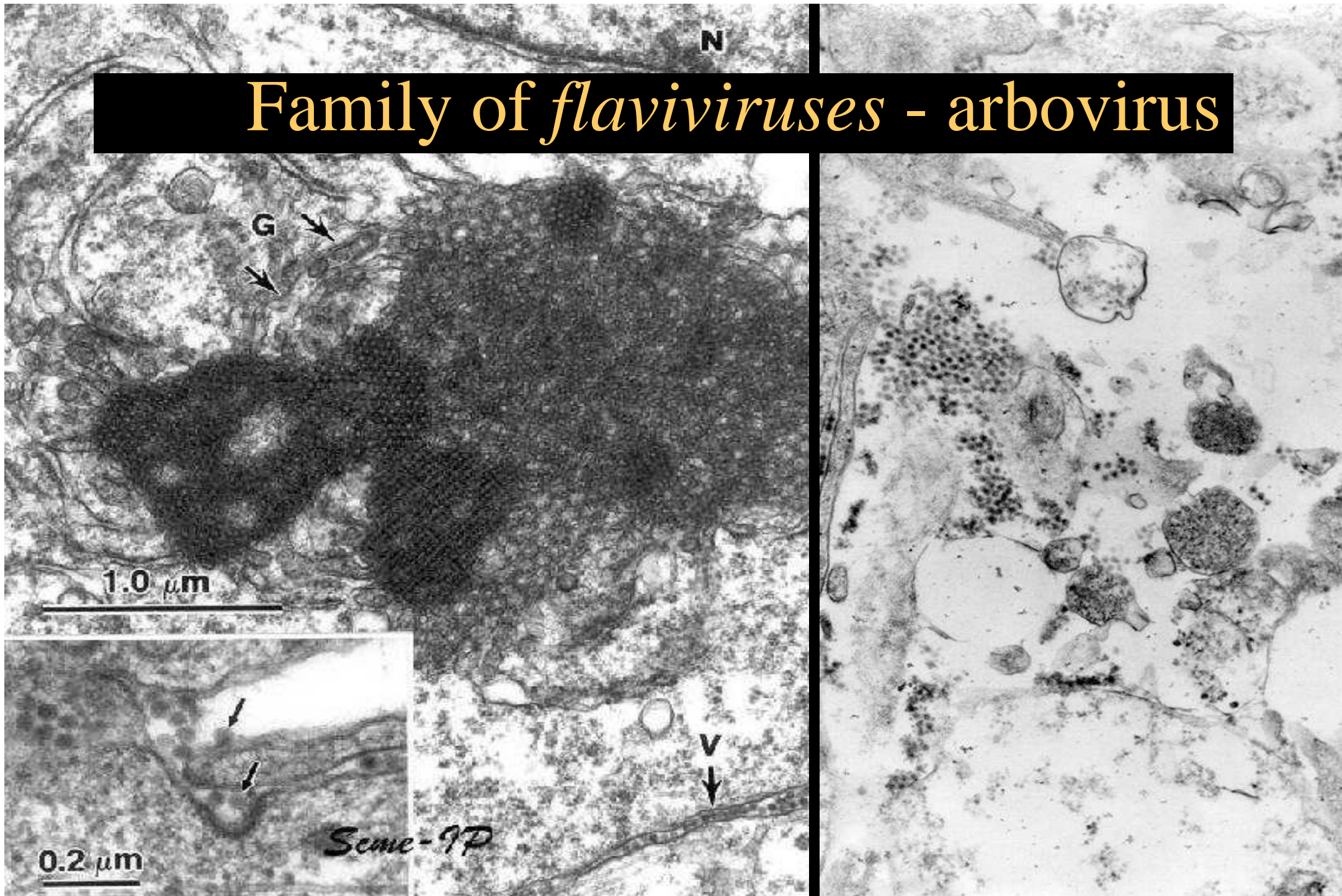
Mosquito-borne viral infection of people

- **West Nile Fever (WNF)**

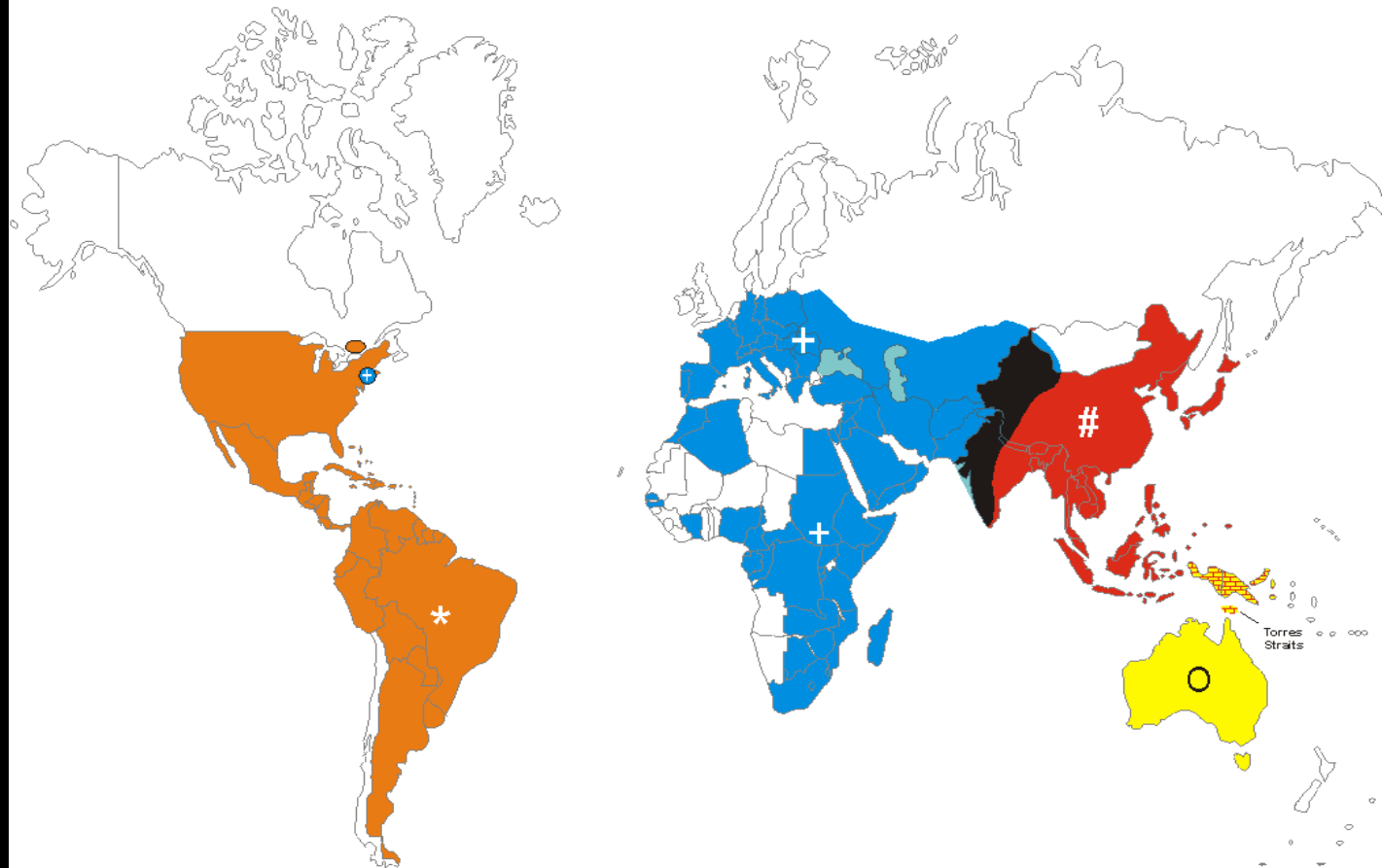
Viral disease (encephalitis) in people caused by the West Nile Virus

- **Equine West Nile Virus Encephalomyelitis**

Family of *flaviviruses* - arbovirus



The Geographic Distribution of the Japanese Encephalitis Serocomplex of the Family Flaviridae, 2000.



- St. Louis encephalitis
- * Rocio and St. Louis (Brazil)
- + West Nile virus
- # Japanese encephalitis
- West Nile and Japanese encephalitis
- Japanese and Murray Valley encephalitis
- Murray Valley and Kunjin

West Nile Virus

1999 - 2007

- Human Cases

1999 – 62

2000 – 18

2001 – 56

2002 – 4,008

2003 – 9,122

2004 – 2,470

2005 – 2,949

2006 – 4,219

2007 – 3,576

- Horse cases

1999 – 25 horses

2000 – 60 horses

2001 – 738 horses

2002 – 15,257 horses

2003 – 4,636 horses

2004 - 1,341 horses

2005 – 1,088 horses

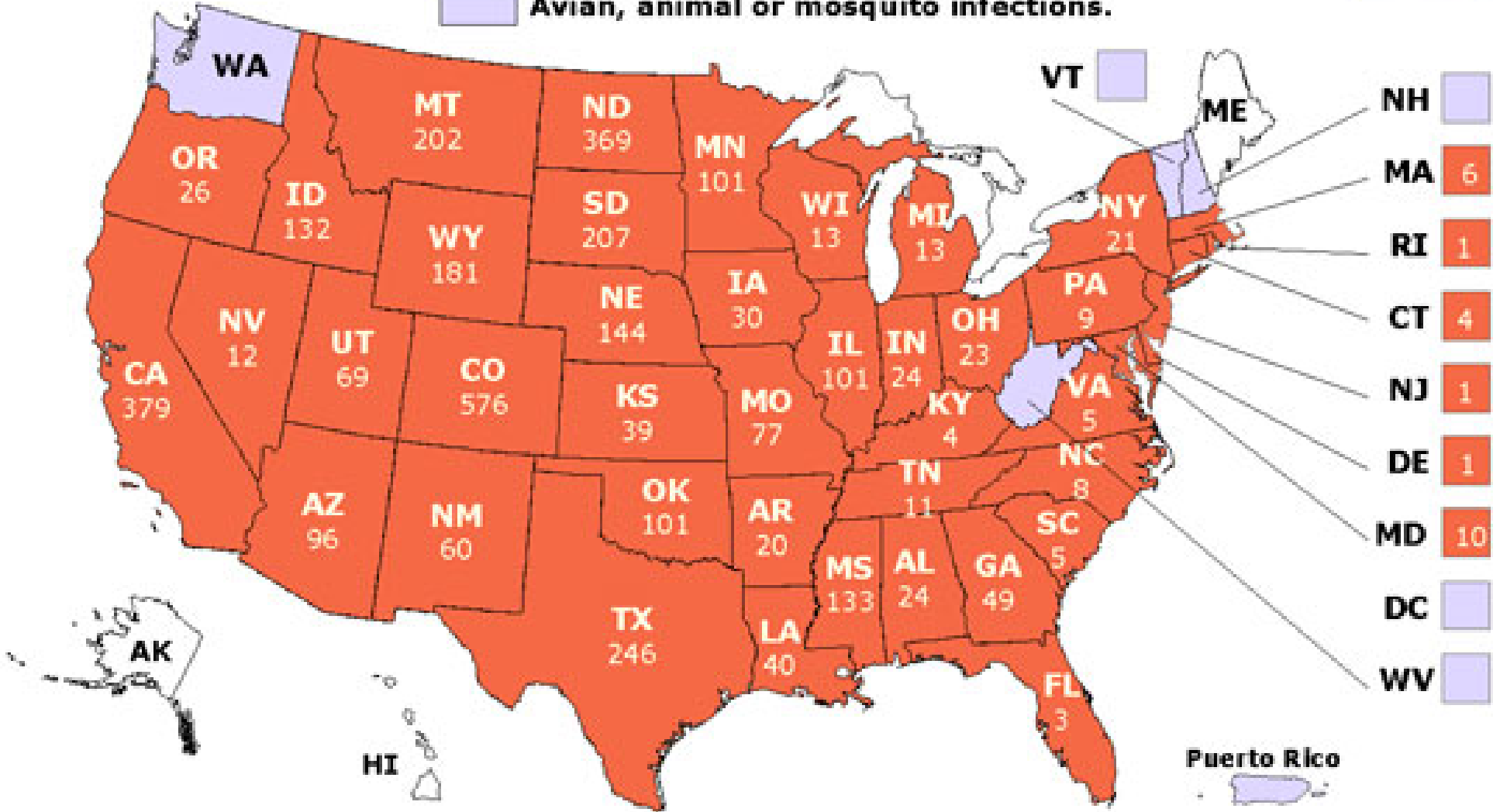
2006 – 1,061 horses

2007 – 484 horses



 Indicates human disease case(s).

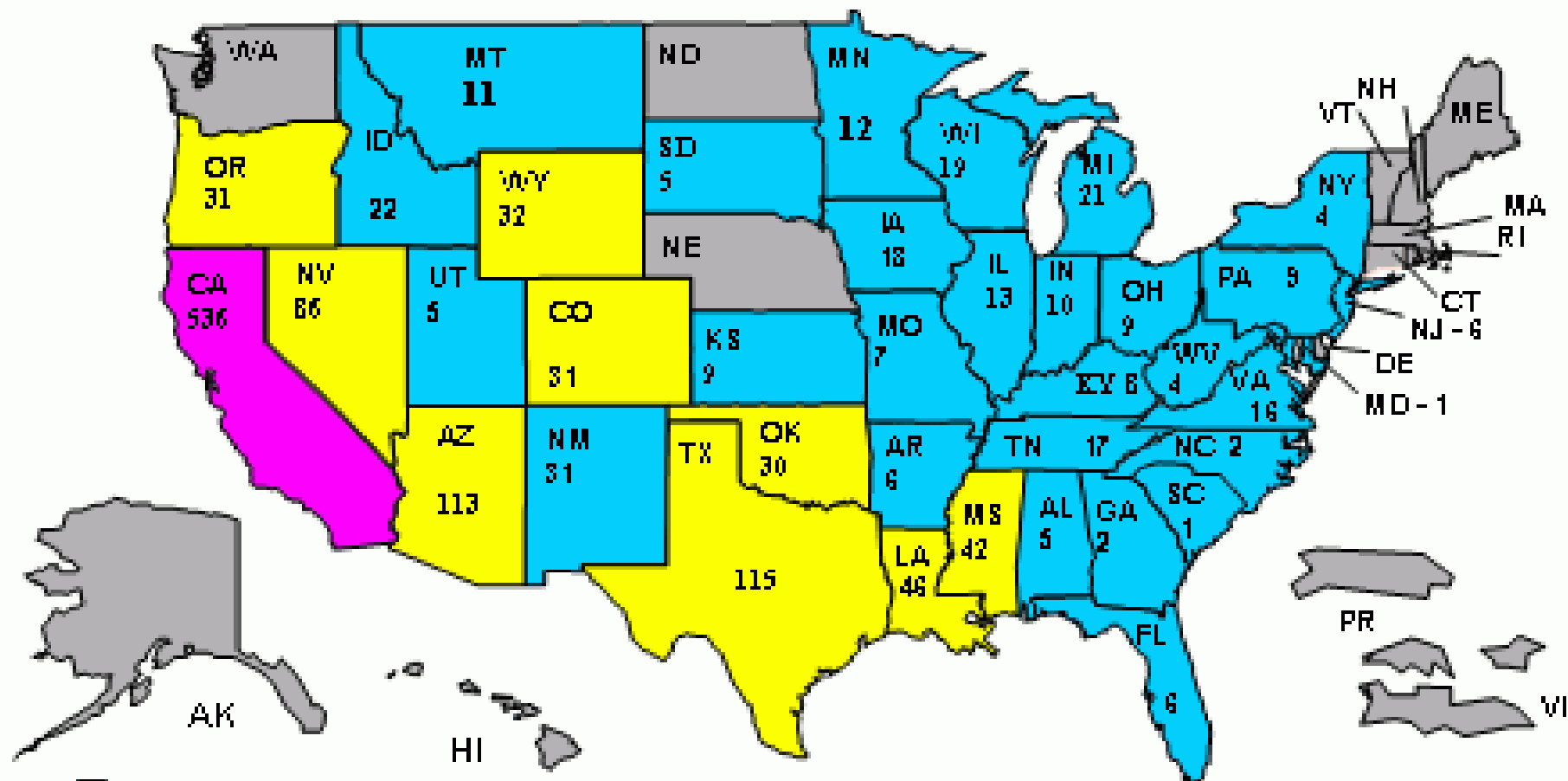
 Avian, animal or mosquito infections.



West Nile Virus in 2004

States with an Equine Case(s)

Total Cases 1,341



- 1 – 24 Case(s)
- 25 – 199 Case(s)
- 200 – 399 Case(s)
- 400 – 799 Case(s)
- 800 – or More Case(s)
- Not Detected

Updated: December 10, 2004

2004

NY

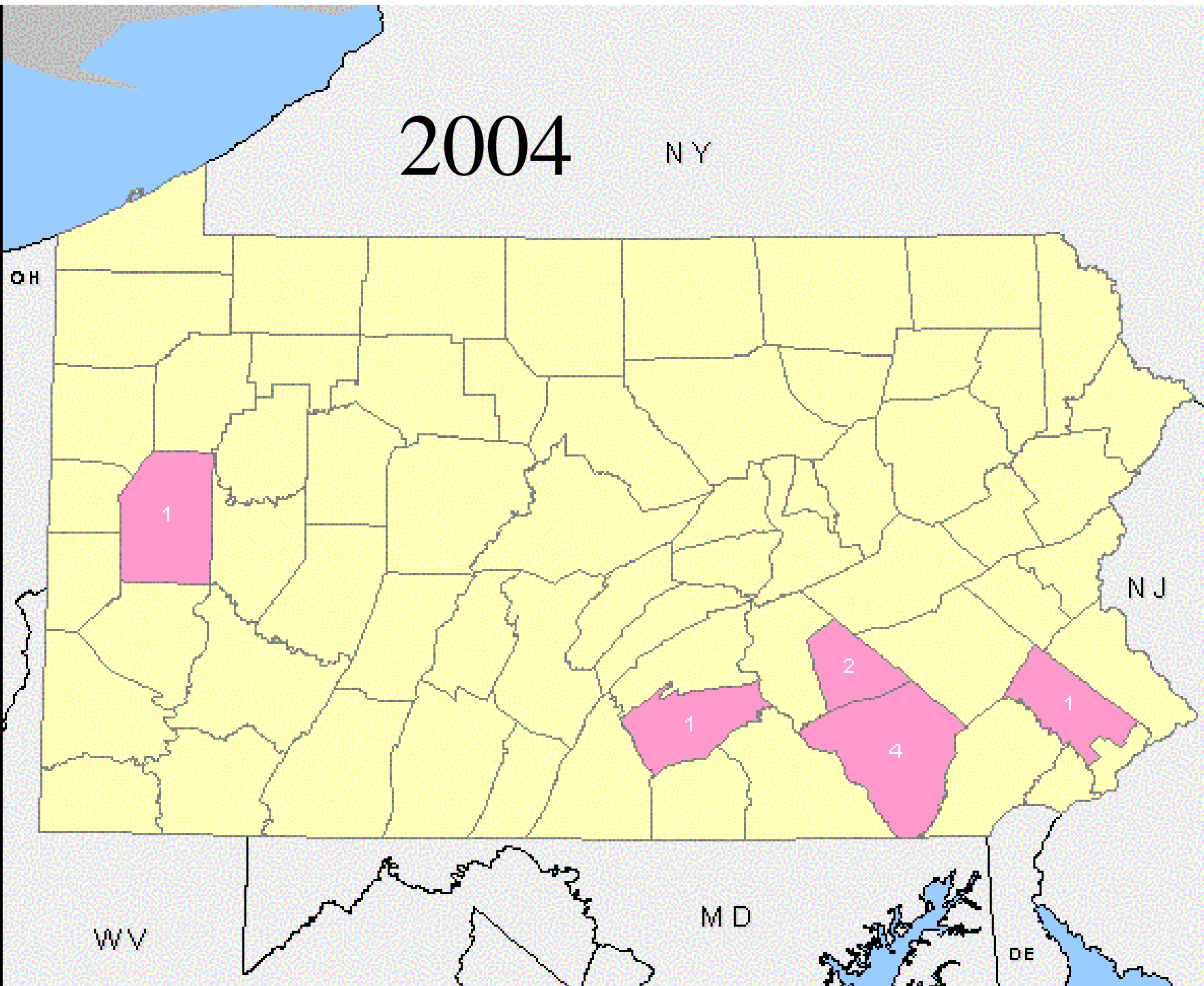
OH

NJ

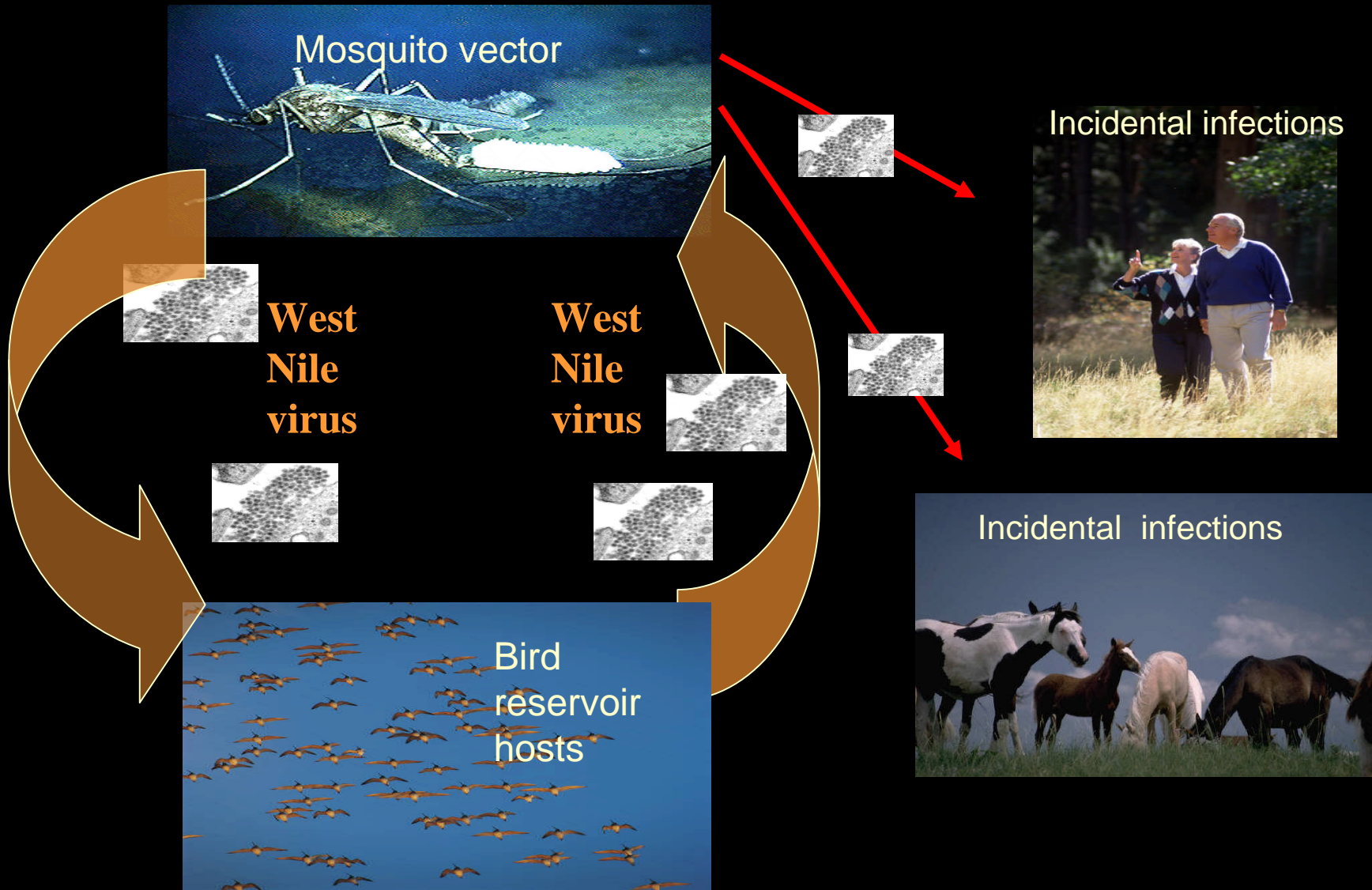
WV

MD

DE



West Nile Virus Transmission Cycle



West Nile Virus Disease in birds

- Europe – only subclinical infections
- North American outbreak
 - First indication – dead crows
 - Neurologic disease – circling, ataxia, seizures
 - Disease of other organ systems
 - Sudden death
 - Fatal in many species of birds

West Nile Virus

Clinical signs in birds



- Weakness, sternal recumbency
- Ataxia
- Tremors
- Anisocoria
- Abnormal head posture
- Circling
- Convulsions

Found in at least 138 bird species

American crow

Ring-billed gull

Yellow-billed cuckoo

Rock dove

American robin

Blue jay

Fish crow

Red-tailed hawk

Broad-winged hawk

Cooper's hawk

Belted kingfisher

American kestrel

Herring gull

Laughing gull*

Bald eagle*

Black-crowned night-heron*

Mallard*

Sandhill crane*

* Captive bird

Other Domestic Animal Hosts



- Horses
- Humans
- Sheep
- Chickens
- Pigs
- Cows
- Dogs
- Cats

West Nile Virus

Other WNV-Positive Mammals



2002 Outbreak

- 3 dogs
- 8 squirrels
- 2 unspecified species

2003 Outbreak

- Camelids

Can West Nile Virus Cause Illness in Dogs or Cats?

Dogs - South Africa

- 37% of dogs seropositive (138/377)
- Virus isolated from 1 dog
- Experimental induction – mild recurrent myopathy
- Low level viremia

Cats

- No published reports
- WNV isolated from dead cats (3) New York

Serosurvey NY 1999 epidemic low infection rate

Equine West Nile Virus Encephalomyelitis World Wide



- 1962-1965 France
- 1963 Egypt
- 1990 Portugal
- 1996 Morocco
- 1998 Italy, Israel
- 1999 Long Island (NY)
- 2000 Northeastern USA, France, Israel
- 2001 Eastern USA
- 2002 – 2006 USA

West Nile Virus

1999 equine outbreak

- Serology from 83/146 resident horses
 - Convalescening horses
 - Contact horses
 - Commingled with a case
 - Co-owned or managed by an owner of a case animal
- 83 horses sampled
 - 15 had a recent clinical illness – all positive
 - 68 horses no history of clinical illness
 - 21 (31%) were serologically positive

West Nile Virus Serology

- **Nigeria** – 71% of horses seropositive
- **Greece**
 - 20% of horses
 - 9% of sheep, 9% of goats, 4% of cattle, 1% of pigs
 - 25% of birds, 29% of humans
- **Russia**
 - 67% of horses, 24% of wild animals
 - 7% of camels, 9% of sheep, 35% of cattle
- **France**
 - 5% of cattle, 2% of sheep, 3% pigs, 5% of humans
- **USA – 1999**
 - 31% of horses on outbreak farms

West Nile Virus

Equine disease in the old world

14 horses from Tuscany, Italy

- Ataxia, weakness, paresis of the hind limbs
- Paraparesis progressing to tetraplegia, recumbency (2 - 9 days).

94 equids (horses, mules, donkeys) in Morocco

- Influenza-like syndrome
- Hind limb paresis progressing to paralysis
- Death in 5 – 10 days in 45% of cases

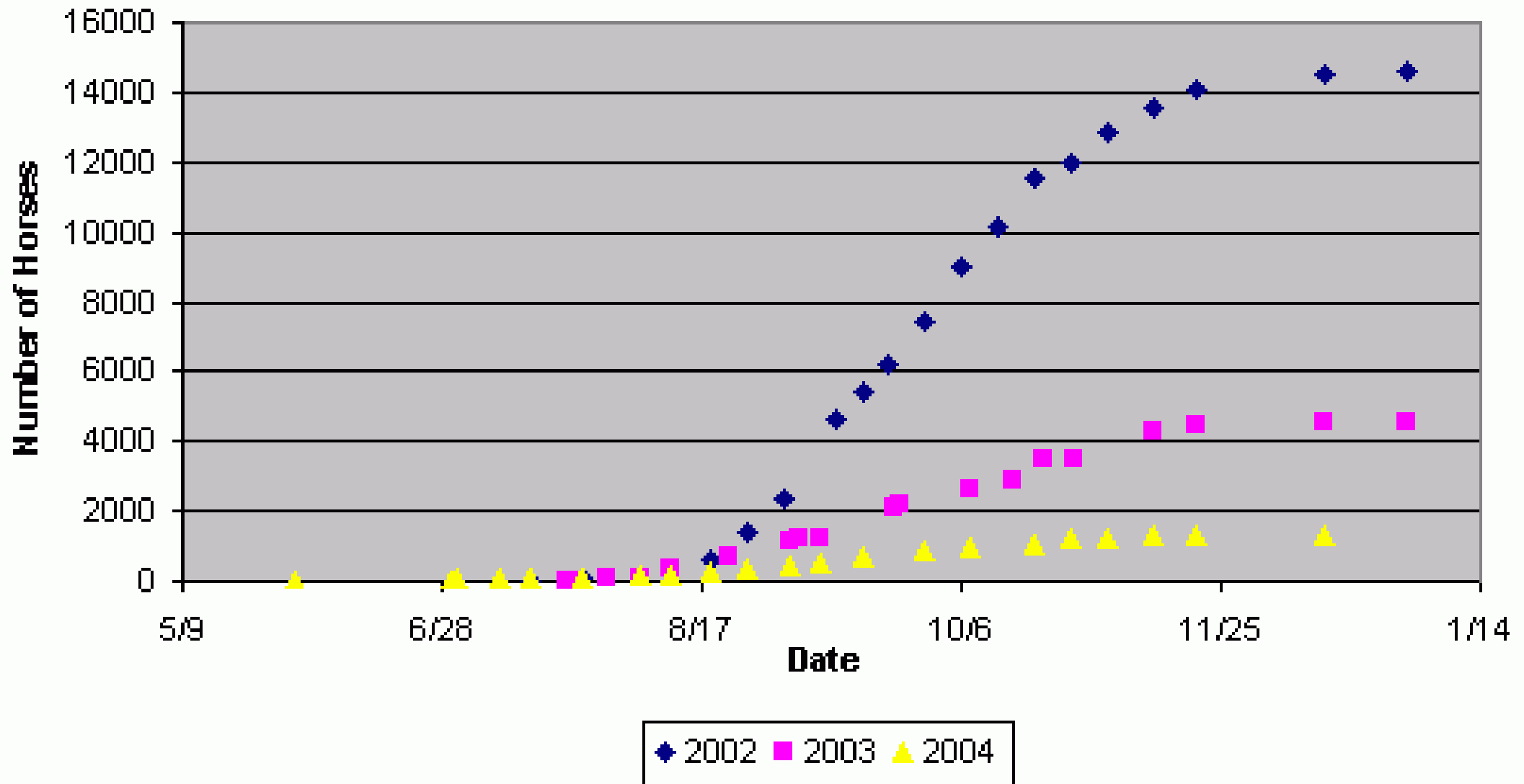
France

- Incubation period about 6 days
- Biphasic fever (6-10 days then 18 days with CNS signs)
- Ascending meningoencephalitis
- Staggering gait progressing to hind leg paralysis
- Death in 14 – 21 days

West Nile Virus Epidemiology

- Exposure geographically clustered
 - Exposure chance event
- Blackbird roosts/waterfowl within 0.5 mile
- Pleasure horses at higher risk
 - More likely exposed to vector? - trail riding activities
- Insect control methods - farm or horse level
 - Not associative with likelihood of infection
 - Primarily aimed at fly control
- Increased risk when not housed in stalls at night
- Mosquito species
 - *Culex spp* primarily identified
 - Transmission to horses ??

West Nile Equine Encephalitis Cases: Trend Comparisons for 2002, 2003, and 2004



Equine West Nile Virus Encephalomyelitis

Clinical Signs



- Fever (< 50%)
- Acute onset of ataxia of all limbs
- Marked hypermetria
- Recumbency
- Single foreleg lameness
 - Progressing to bilateral forelimb lameness/ataxia
 - Monoparesis, paraparesis,
tetraparesis progressing to recumbency
- Radial nerve paralysis

Equine West Nile Virus Encephalomyelitis

Clinical Signs

- Hypersensitivity to touch and sound
- Somnolent - periodically falling to knees
- Anisocoria and a slow pupillary light response
- Tremors and lip twitching
- Muscle fasciculations
 - Most pronounced in the neck and triceps region
- Difficulty swallowing
- Facial nerve paralysis
- Central blindness
- Seizure activity

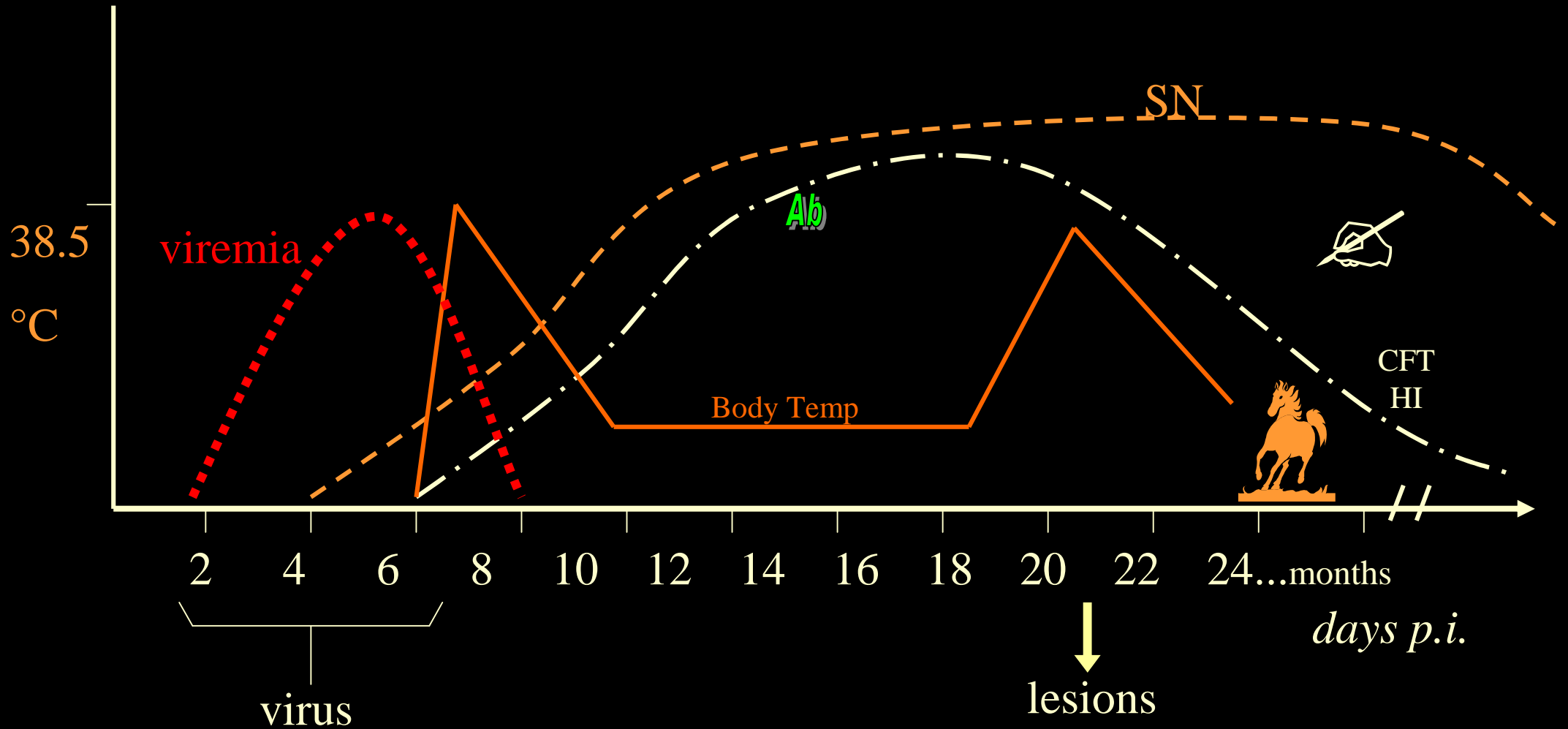
Equine West Nile Virus Encephalomyelitis Laboratory Findings

- CBC – normal
- Fibrinogen – normal
- CSF – usually normal
 - Increase CSF protein and xanthochromia
- Blood chemistry normal

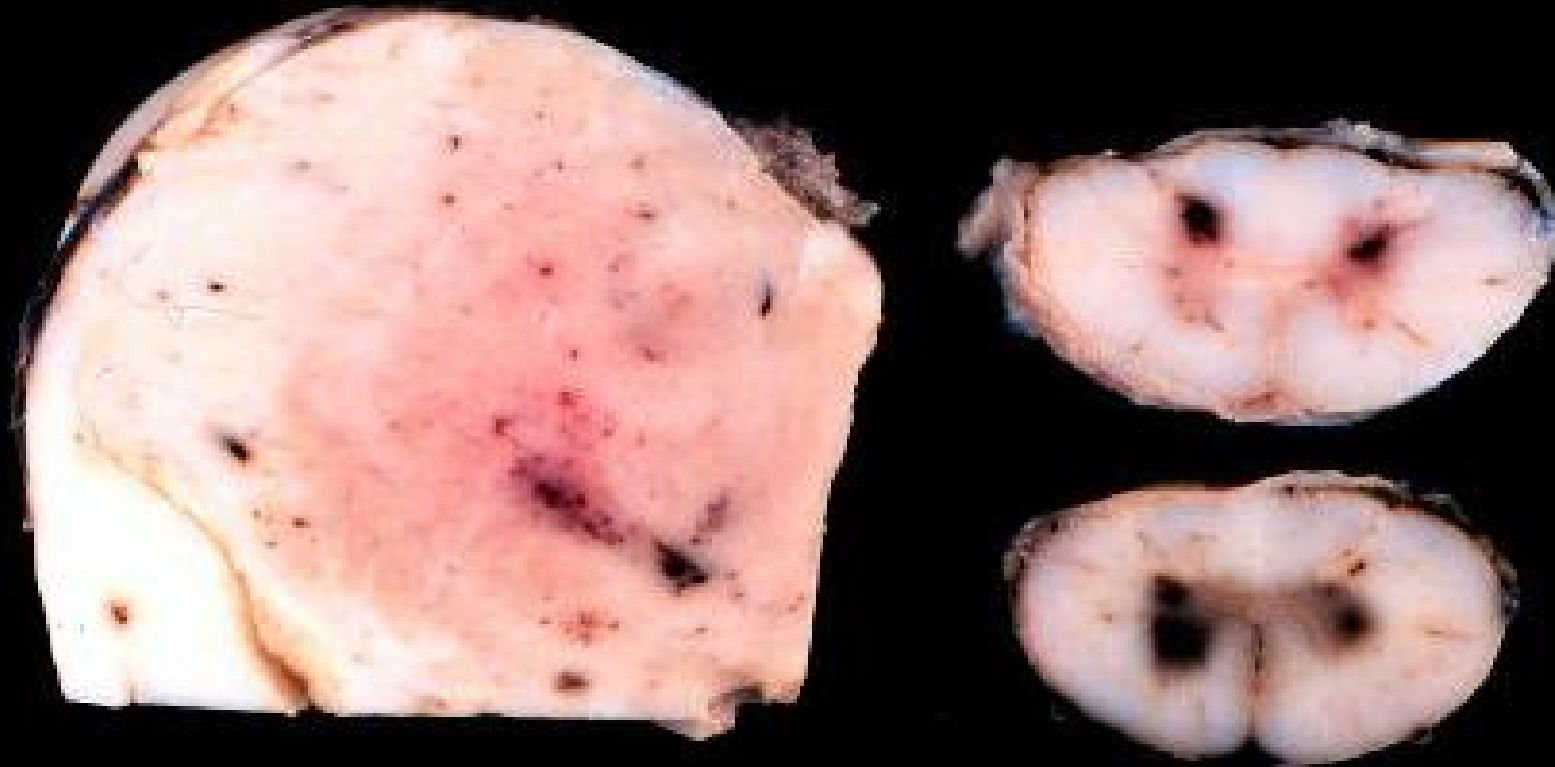
Equine West Nile Virus Encephalomyelitis Diagnosis

- Serology
 - Fluorescent antibody
 - Virus neutralizing antibodies (3 wks PI to years)
 - IgM capture ELISA (10-12 days PI to weeks?)
- RT-PCR
 - Blood – negative
 - CSF – negative
 - Brain/cord - positive
- Virus isolation
 - Blood – negative
 - Brain/cord

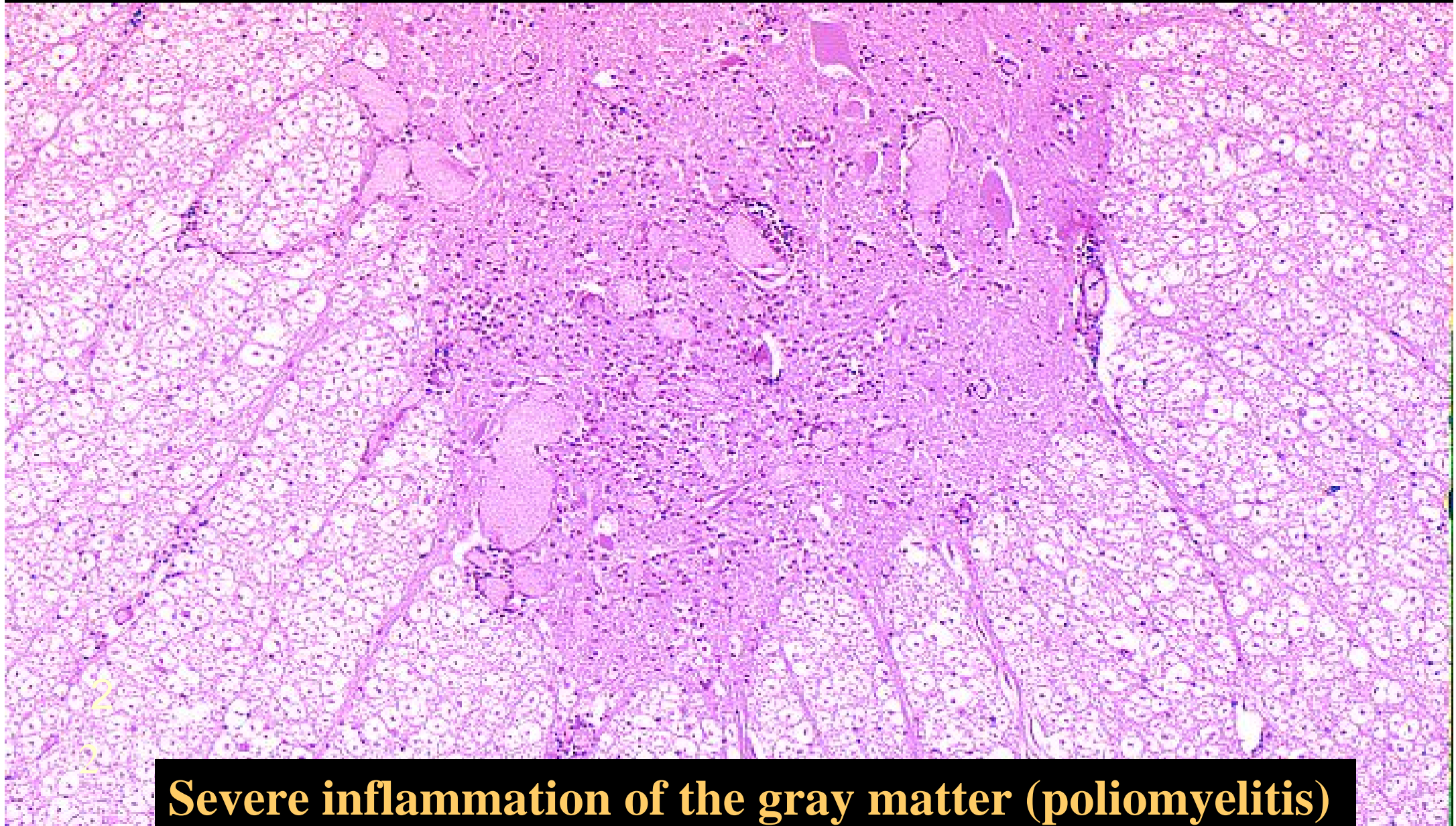
Experimental infection WNV in horses



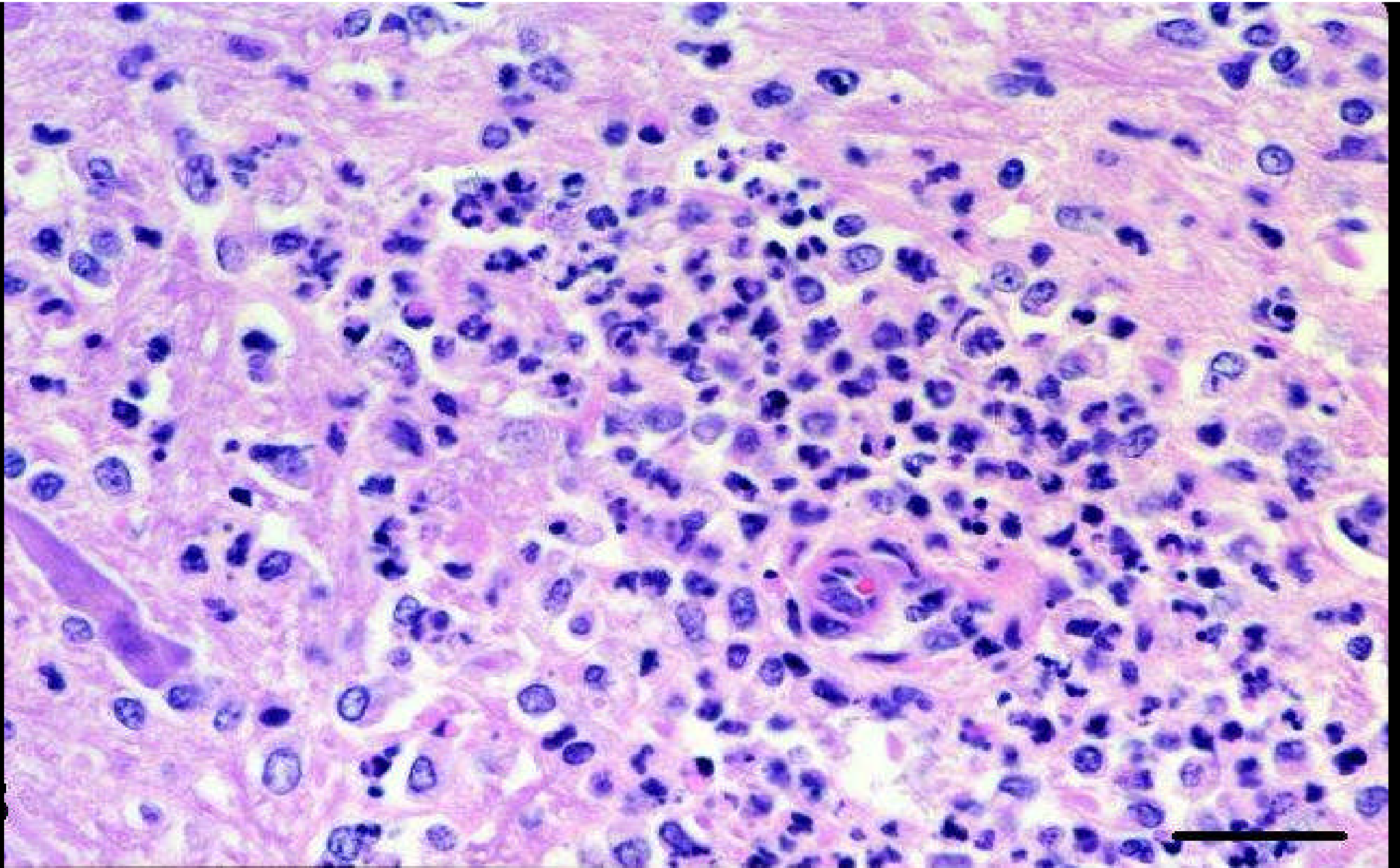
Joubert *et al.* Bull Acad Vét Fr, 1971



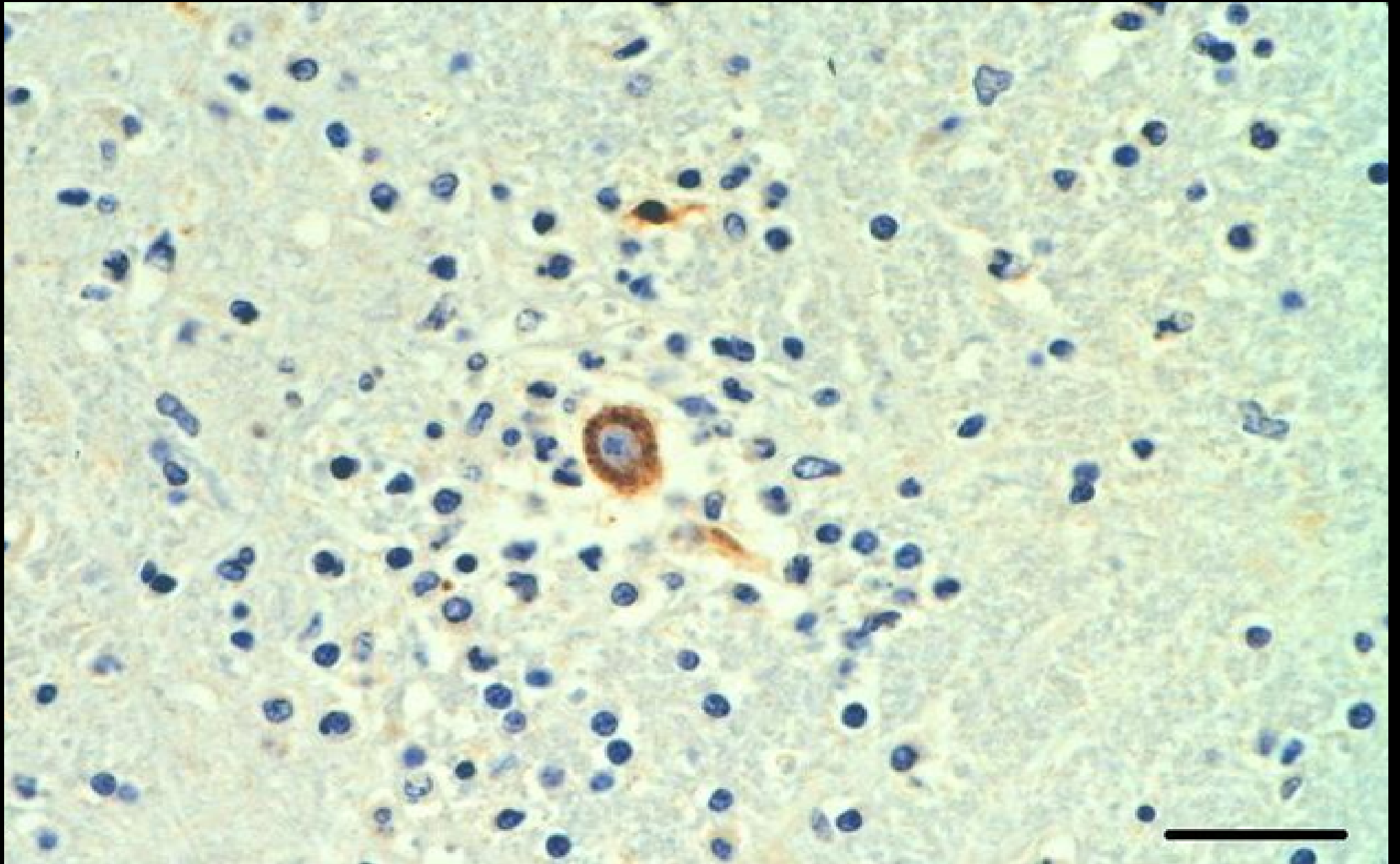
Multifocal Hemorrhages



Severe inflammation of the gray matter (poliomyelitis)



Inflammation and necrosis of the neural cells



West Nile virus antigen – cytoplasm of neuron

Equine West Nile Virus Encephalomyelitis Differential Diagnosis

- Eastern, Western, Venezuelan Equine Encephalomyelitis
- Equine herpes virus 1
- Rabies
- Equine protozoal myelitis
- Leukoencephalomacia
- Stenotic cervical myelopathy
- Hepatic, intestinal or renal encephalopathies

Equine West Nile Virus Encephalomyelitis Treatment

- Supportive, Preventing self-inflicted injury
- Fluids
- Treat for other differential diagnoses
 - Herpes virus suspect status
 - Rabies suspect status
- Hyperimmune plasma?
- Interferon therapy?

Equine West Nile Virus Encephalomyelitis Clinical Course and Outcome

Recovery

- 80% patients recover fully
- Course averages 22 days

Reported fatality rates

- World wide 30 – 64%
- USA 50 - 33%
- Presented with the most severe signs (recumbency)
- Rapid progression of neurologic signs leading to recumbency
- Recumbent horses 78 times more likely to die
- Female 2.9 times more likely to die
- > 3 years old more likely to die

Prognosis good if continued ability to rise

Course before death average 2 days (0 – 6 days)

Equine West Nile Virus Encephalomyelitis

Fort Dodge vaccine

- Fully licensed

Vaccine tested with challenge studies

- No vaccinates or controls developed signs
- Vaccinates did not develop viremia

- Millions of doses sold

Few adverse reactions

Lay horsemen fear reproductive problems

- Internet web site

Equine West Nile Virus Encephalomyelitis

Fort Dodge vaccine

- Efficacy unknown

Weeks after 2nd dose before a measurable antibody response.

Horses may develop WNV soon after 1st dose of vaccine

- Suggesting that one dose is not protective

- The manufacturer's recommendations

2 doses 3-6 weeks apart

Yearly booster

- Epidemiology

Unvaccinated horses > 2 X more likely to die

Equine West Nile Virus Encephalomyelitis

Merial vaccine

- Recombitek
 - Recombinant canarypox vaccine
 - Vectored recombinant DNA vaccine
- Stimulates both arms of the immune system
- Challenge studies
 - Prevents viremia up to 1 year later
 - Need 2 vaccinations
- Appears safe in small field studies
 - Not labeled for use in pregnant mares
- Efficacy unknown

Equine West Nile Virus Encephalomyelitis

Prevention



- Other arbovirus encephalitis vaccines

EEE, WEE, VEE

Do not cross protect

- Mosquito control very important

Equine West Nile Virus Encephalomyelitis Prevention

- Stop the bird – mosquito infection cycle

- Mosquito control

Primary *Culex spp*

Equine vector?

Range < 1000 yards

Local problem – local control



Larval habitat destruction

Culex spp

- Any puddle that lasts more than 4 days - habitat
- Reduce the amount of standing water available

Water troughs, water buckets

Swimming pools, plastic wading pools

Bird baths, wheelbarrows

Clogged roof gutters

Recycling containers

Discarded tires

Tin cans, plastic containers, ceramic pots

Any water-holding container

Prevention Mosquito Control

- Practice mosquito control on farms
 - Identify and destroy larval habitats
 - Larvicides - BTI - *Bacillus thuringiensis* var. *israelensis*
- Stable horses during peak mosquito feeding times
 - Dusk, Dawn
 - Stable at night
- Adult mosquito control – last resort
 - Use insect repellents

*What is the risk of transmission
from a mosquito bite?*



Risk of Transmission by a Mosquito Bite in an Endemic Area

- Area where birds have West Nile virus
- Area where mosquitoes carry the virus
- Very few mosquitoes carry the virus
1 less than out of 500
Chance that one mosquito bite will be from an infected mosquito is very small
- If bitten by an infected mosquito
Chance of developing illness are roughly 1 in 300

Can I Get Infected with West Nile Virus by Caring for an Infected Horse?



- West Nile virus is mosquito transmitted
- No evidence of animal-to-person transmission

Can a Horse Infected with West Nile Virus Infect Other Horses?

- No documented evidence that West Nile virus is transmitted from horse-to-horse.
- New evidence – possible transplacental infection

Abortion

Are Horses a Source of West Nile Virus?

- Are horses terminal hosts?
- Historic evidence
 - No virus in blood when clinically ill
 - 2 experimental induction studies
- USDA & CSU/CDC
 - 3 exp inoculation/transmission studies since 1999
 - 16 horses max viremia 10^3 /ml (most $< 10^2$ /ml)
 - 8 horses infected via mosquito (*Aedes albopictus*)
 - 1 hrs developed clinical signs
 - > 600 mosquitoes fed on viremic horses – all negative
- Virus in nervous tissue
- Very little virus in tissues

Should a Horse Infected with West Nile Virus be Destroyed?



- There is no reason to destroy an infected horse
- At least half of the horses recover from the infection
- Treatment is supportive and not specific

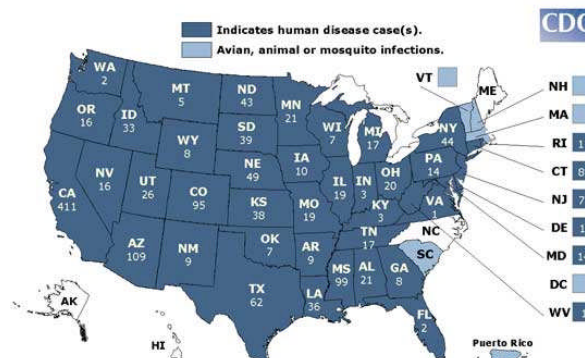
Equine West Nile Virus Encephalomyelitis

I. Definitions

- A) West Nile Virus (WNV) - Flavivirus in the Japanese Encephalitis Antigenic Complex; arbovirus.
- B) West Nile Fever (WNF)– viral encephalitis in people caused by the West Nile Virus

II. Background

- A) West Nile virus has emerged in regions of Europe and North America. WNV causes fatal encephalitis in humans and horses, as well as causing mortality in certain domestic and wild birds.
- B) WNV was first isolated from a febrile woman in the West Nile District of Uganda in 1937. Equine disease was first noted in Egypt and France in the early 1960s.
- C) WNV has been identified in Africa, Europe, the Middle East, west and central Asia, Oceania and North America. Outbreaks in humans have occurred in Algeria in 1994, Romania in 1996-1997, the Czech Republic in 1997, the Democratic Republic of the Congo in 1998, Russia in 1999, the United States in 1999-2008, and Israel in 2000. Epizootics of disease in horses occurred in Morocco in 1996, Italy in 1998, the United States in 1999-2008, and France in 2000.
- D) Human cases US 2002 – 4156 cases with 284 deaths; US 2003 – 8567 cases with 199 deaths; US 2004 – 2470 cases with 88 deaths; US 2005 – 2949 cases with 116 deaths; US 2006 – 4219 cases with 161 deaths; US 2006 – 3359 cases with 98 deaths; US 2007 – 3630 cases with 124 deaths; US 2008 – 1370 cases with 37 deaths



III. Transmission

- A) Arbovirus transmitted primarily by *Culex spp* mosquitoes
 - 1) The virus has been isolated from 43 mosquito species, predominantly of the genus *Culex*.
 - 2) Transovarial transmission has been demonstrated though at low rates.
 - 3) Requires 6-10 days incubation after feeding on infected bird before mosquito is infectious
 - 4) Virus isolations have occasionally been reported from other hematophagous arthropods (e.g., bird-feeding argasid [soft] or amblyommine [hard] ticks), and experimental transmission has been successful.

- B) Not thought to be transmitted by direct contact with birds
 - 1) In North America, infected birds have been found to have an abundance of virus in many tissues and to excrete virus in feces.
 - 2) It is possible, when handling dead birds or when carnivores eat birds infected with WNV, that exposure may occur.
 - 3) Preliminary reports suggest possible direct bird to bird transmission.
 - (a) Raptors can acquire the virus by eating infected prey
 - (b) Birds may transmit virus in feces
 - (c) Transovarial transmission may occur
 - 4) Caution should be taken when handling live or dead birds in a WNV endemic area.

- C) All mammals, with the possible exception of Lemurs, are thought to be dead end hosts.
 - 1) WNV has been isolated from mice and hamsters, camels, camalids, cattle, horses, dogs, humans, lemurs, and frogs.

- D) Transmission does not occur by direct mammal-to-mammal contact.

IV. Disease in birds

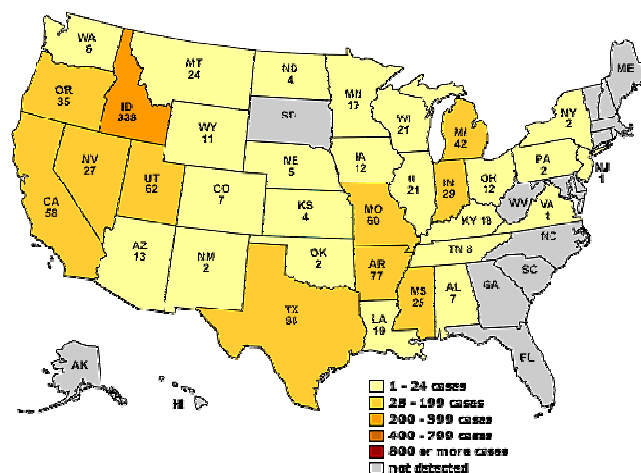
- A) In Europe – only subclinical infections
- B) North American outbreak
 - 1) First indication that the virus is in an area is often the finding of dead corvids, especially crows.
 - 2) The birds show signs of neurologic disease
 - (a) Weakness, sternal recumbency
 - (b) Ataxia
 - (c) Tremors
 - (d) Anisocoria
 - (e) Abnormal head posture
 - (f) Circling
 - (g) Convulsions

- 3) Disease of other organ systems - virus can be found in the heart, kidney, liver and gastrointestinal tract with abundant virus in the lumen of the GI tract.
- 4) Sudden death from systemic infection is common.
- 5) Has been identified in at least 138 species of birds in North America.

V. Equine West Nile Virus Encephalomyelitis World Wide

- A) 1962-1965 - Camargue (France) – 50 cases
- B) 1963 – Egypt
- C) 1990 – Portugal
- D) 1996 – Morocco - 42 of 94 affected horses died
- E) 1998 – Italy - 14 cases in 1998, 6 died or were euthanized
- F) 1998 – Israel
- G) 1999 – Long Island (NY) – 13 of 25 cases fatal
- H) 2000 – Northeastern USA - 23 of 65 cases fatal
 - 1) New Jersey (27 horses), New York (24 horses), Connecticut (7 horses), Delaware (4 horses), RI (1 horses), Massachusetts (1 horses), Pennsylvania (1 horses).
- I) 2000 – France – highly fatal outbreak
- J) 2001 – Eastern USA – 738 equine cases in 20 states.
 - 1) 33 % fatality rate (156/470).
- K) 2002 – USA - 14,717 equine cases in 40 states.
- L) 2003 – USA – 4,426 equine cases in 48 states.
- M) USA - 2004 – 1,341 equine cases; 2005 – 1,075 equine cases; 2006 – 1061 equine cases; 2007 – 466 equine cases ; 2008 – 218 equine cases
- N) Seroepidemiology (high rate subclinical infections): 31% in the Long Island outbreak, 20-70% of normal horses seropositive in endemic areas Europe. France 8% of horses in outbreak IgG titers; 4% had IgM titers.

2006 – 1061 EQUINE CASES



VI. **Epidemiology** -- USDA case control study 2000

- A) 1487 equids on 49 case farms and 101 control farms
 - 1) Farms located in Connecticut, Delaware, Massachusetts, New Jersey, New York, Pennsylvania and Rhode Island.
- B) Findings
 - 1) Marginally significant association between case farms and presence of Blackbird roosts and waterfowl congregations within 1/2 mile.
 - 2) Insect control methods -- farm or horse level
 - (a) Not associative with likelihood of infection
 - (b) lack of association may be due to
 - (1) Heterogeneous approach from farm to farm
 - (2) Control methods targeted flies
 - 3) Pleasure horses at higher risk
 - (a) More likely expose the vector? -- trail riding activities
 - 4) Increased risk when not housed in stalls at night
 - 5) Occurrence of cases associated with horse demographics and geographic distribution of dead birds
 - (a) Endemic focus in bird roosts results in virus amplification
 - (b) Proceed spillover to horses
 - 6) Exposure of horses is geographically clustered
 - (a) But within regions of virus activity, exposure of horses appears to be a chance event
 - (b) This type of pattern may respond to effective vaccination
 - 7) Mosquito species responsible for transmission to horses
 - (a) Has not been identified
 - (b) Control measures should be directed towards appropriate mosquito species

VII. **Clinical disease - North American experience**

- A) Clinical signs
 - 1) Fever – in less than half the cases
 - 2) Acute onset of ataxia of all limbs
 - 3) Marked hypermetria
 - 4) Early recumbency
 - 5) Single foreleg lameness progressing to bilateral forelimb lameness/ataxia, monoparesis, paraparesis, tetraparesis progressing to recumbency
 - 6) Radial nerve paralysis
 - 7) Hypersensitivity to touch and sound – frequently present
 - 8) Somnolent – periodically falling to knees
 - 9) Anisocoria and a slow pupillary light response

- 10) Tremors and lip twitching
- 11) Muscle fasciculations
- 12) Most pronounced in the neck and triceps region
- 13) Difficulty swallowing
- 14) Facial nerve paralysis
- 15) Central blindness
- 16) Seizure activity
- B) Estimated incubation period 5 – 15 days
- C) Fatality rate – 40 - 33%
- D) Course before death average 2 days (0 – 6 days)

VIII. **Laboratory Findings**

- A) CBC – normal
- B) Fibrinogen – normal
- C) CSF – normal 3 of 4 cases
 - 1) Abnormal case had increase CSF protein and xanthochromia (N=1)
- D) Blood chemistry normal

IX. **Diagnosis**

- A) Serology
 - 1) Fluorescent antibody
 - 2) Virus neutralizing antibodies
 - 3) IgM capture ELISA
- B) RT-PCR
 - 1) Blood – negative?
 - 2) CSF – negative?
 - 3) Brain/cord - positive
- C) Virus isolation
 - 1) Blood – negative?
 - 2) Brain/cord – positive

X. **Differential Diagnosis**

- A) Eastern, Western, Venezuelan Equine Encephalomyelitis
- B) Equine herpes virus 1
- C) Rabies
- D) Equine Protozoal myelitis
- E) Leukoencephalomalacia
- F) Stenotic cervical myelopathy
- G) Hepatic, intestinal or renal encephalopathies

XI. Outcome

- A) 1999 outbreak Long Island (NY) – 50% fatal
- B) 2000 outbreak Northeastern USA – 40% fatal
- C) 2001 outbreak Northeastern USA – 33% fatal
- D) Reported mortality rates 30 – 64%

XII. Post Mortem

- A) Only nervous tissue involvement
 - 1) Few gross lesions within the brain – occasional hemorrhages
- B) Prominent rhombencephalic lesions
 - 1) Multifocal perivascular lymphocytic rhombencephalitis
 - 2) Ring hemorrhages, neutrophils and multifocal microgliosis
- C) Peroxidase immunohistochemical staining
 - 1) West Nile virus antigen in cytoplasm
 - 2) Few neurons, nerve fibers, glial cells, neutrophils
- D) Virus isolation
- E) RT-PCR

XIII. Treatment

- A) Supportive
- B) Preventing self-inflicted injury
- C) Fluids
- D) Treat for other differential diagnoses
- E) Herpes virus suspect status
- F) Rabies suspect status
- G) Plasma transfusion – passive antibody transfer
- H) Interferon therapy

XIV. Prevention

- A) Available vaccine
 - 1) Fort Dodge
 - (a) Vaccine tested with challenge studies
 - (1) No vaccinates or controls developed signs
 - (2) Vaccinates did not develop viremia
 - 2) Millions of doses sold
 - 3) Few adverse reactions
 - (a) Internet stories of abortion with congenital malformations
 - (b) Appear to have no basis – for numbers vaccinated, few reported adverse reactions
 - (c) Killed vaccine unlikely source of congenital malformations

- (d) Isolation of wild virus from some aborted fetuses in Kentucky
 - (1) Wild virus
 - (2) Undefined role in abortion
 - (3) Isolated in small percent of aborted fetuses
- 4) Efficacy unknown
 - (a) It may take several weeks after the second dose before a measurable antibody response.
 - (b) Horses may develop WNV soon after receiving the first dose of vaccine
 - (1) Suggesting that one dose is not protective
 - (c) The manufacturer's recommendations
 - (1) 2 doses 3-6 weeks apart
 - (2) Yearly booster
 - (d) Vaccinated horses may develop disease
 - (1) Frequency this occurs is under investigation
 - (2) The percent of vaccinates which are not fully protected unknown
 - (e) Unvaccinated horses > 2X more likely to die
- B) Recombitek[®] - Merial vaccine
 - 1) Recombinant canarypox vaccine
 - 2) Prevents viremia up to 1 year
- C) Other arbovirus encephalitis vaccines
 - 1) EEE, WEE, VEE
 - 2) Do not cross protect
- D) Mosquito control very important
 - 1) Stop the bird – mosquito infection cycle
 - (a) Mosquito control
 - (1) Primary infected mosquito is *Culex spp*
 - (2) Control tailored to this species
 - (3) Vector for horse could be another, less commonly infected, species
 - 2) *Culex spp* control
 - (a) Range < 1000 yards
 - (b) Local problem – local control
 - 3) Larval habitat destruction for *Culex spp*
 - (a) Any puddle that lasts more than 4 days - habitat
 - (b) Reduce the amount of standing water available
 - (1) Water troughs, water buckets
 - (2) Swimming pools, plastic wading pools
 - (3) Bird baths, wheelbarrows
 - (4) Clogged roof gutters
 - (5) Recycling containers
 - (6) Discarded tires
 - (7) Tin cans, plastic containers, ceramic pots
 - (8) Any water-holding container
 - (c) Larvicides - BTI - *Bacillus thuringiensis var. israelensis*

- 4) Stable horses during peak mosquito feeding times
 - (a) *Culex spp* - dusk, dawn
- 5) Adult mosquito control – last resort
 - (a) Use insect repellents
 - (1) Pyrethroid-based
 - (2) Containing DEET? – only equine approved products

XV. Control

- A) What is the threat to care givers?
 - 1) No evidence of animal-to-person transmission
- B) Can a horse infected with West Nile virus infect other horses?
 - 1) No documented evidence that WNV is transmitted from horse-to-horse.
- C) Are horses a source of West Nile virus? Are horses terminal hosts?
 - 1) Historic evidence
 - (a) No virus in blood when clinically ill – dead-end host
 - 2) 2 historic experimental induction studies
 - (a) Showed low level or no viremia – dead-end host
 - 3) 3 experimental inoculation/transmission studies since 1999 in US
 - (a) USDA & CSU/CDC
 - (b) 16 horses studied - maximum viremia 10^3 /ml (most $< 10^2$ /ml)
 - (c) Below number needed to infect feeding mosquitoes – dead-end host
 - 4) 8 horses infected via mosquitoes (*Aedes albopictus*)
 - (a) 1 hrs developed clinical signs
 - (b) 600 mosquitoes fed on horses during viremia – all negative
 - 5) Virus
 - (a) In nervous tissue – very low numbers
 - (b) No virus seen in other tissues – dead-end host
- D) Should a horse infected with West Nile virus be destroyed?
 - 1) There is no reason to destroy an infected horse
 - 2) Half of the horses recover from the infection