Pseudorabies

Aujeszky's Disease
Mad Itch
Infectious Bulbar Paralysis

Last Updated: January 2024

Importance

Pseudorabies (Aujeszky's disease) is a highly contagious, economically significant viral disease of pigs that can occasionally cause fatal illnesses in other animals. It is generally a severe disease with central nervous system (CNS) signs in suckling piglets without maternal antibodies; however, older pigs mainly have self-limited respiratory signs, and reproductive losses are the primary syndrome in adults. Recovered swine can carry the virus latently, and may resume shedding it at a later time. Pseudorabies became a particular concern in the second half of the 20th century, when industrialization of the swine industry facilitated virus transmission, and a number of countries subsequently implemented eradication programs. As a result, this virus has been eliminated, or mostly eliminated, from domestic pigs in many areas, though it often still circulates in feral pigs and/or wild boar. It remains a concern for commercial pigs in some countries including China, where clinical signs are usually controlled by vaccination. New viral variants were found in vaccinated herds in China around 2010 and have since recombined with other viral strains, complicating disease control.

Other animals exposed to infected suids occasionally develop a neurological illness that resembles rabies but is often accompanied by severe pruritus. Most cases are fatal. While these animals are not usually contagious, large outbreaks have been seen in farmed mink and foxes fed contaminated pig tissues. Occasional cases still occur even in pseudorabies-free countries when animals, especially hunting dogs and wildlife, are exposed to wild suids. Recent reports from China have also proposed a causative role for pseudorabies virus in viral encephalitis and ocular disease in humans. To date, these reports have been based mainly on the detection of viral nucleic acids with a highly sensitive technique that can sometimes reveal novel agents but requires confirmation by standard diagnostic methods when the agent is not an established pathogen.

Etiology

Pseudorabies is caused by a herpesvirus informally known as pseudorabies virus or Aujeszky's disease virus, which belongs to the genus *Varicellovirus* in the family Orthoherpesviridae (subfamily Alphaherpesvirinae). It was recently given the formal name *Varicellovirus suidalpha1*, replacing its previous name of suid herpesvirus 1 (SuHV-1). Pseudorabies virus has only one serotype but 4 main genotypes, I to IV, which can reassort with each other and with live attenuated vaccine strains. Isolates can differ in virulence, and some of the viruses maintained in wild boar and feral swine appear to be relatively attenuated for pigs, though not necessarily for other species.

Species Affected

Members of the species *Sus scrofa*, including domestic pigs, wild boar and their crosses, are the natural hosts for pseudorabies virus. At least one textbook suggests that African suids in the genera *Phacochoerus* (warthogs), *Potamochoerus* (bush pigs), *Hylochoerus* (giant forest hogs) and *Porcula* (pygmy hogs) are also susceptible, but there do not seem to be any published studies on these animals. The susceptibility of Asian wild Suidae, such as babirusa (*Babyrousa* spp.), has not been described. Information about peccaries (family Tayassuidae) is also limited; however, viral nucleic acids were detected in the spleens of white-lipped peccaries (*Tayassu pecari*) and collared peccaries (*Pecari tajacu*) that had died of other causes, and one study found antibodies to the virus in white-lipped peccaries.

Other mammals sometimes act as incidental hosts. Clinical cases have been reported in domestic animals including cattle, sheep, goats, cats and dogs; farmed carnivores such as mink and foxes; and various captive or free-living wildlife, including African wild dogs (*Lycaon pictus*), wolves (*Canis lupus*), coyotes (*C. latrans*), red foxes (*Vulpes vulpes*), panthers (*Puma concolor*), Iberian lynx (*Lynx pardinus*), bears, skunks and raccoons (*Procyon lotor*). Naturally acquired cases have not been documented in any nonhuman primates, and early reports found that rhesus monkeys (*Macaca mulatta*) and Barbary macaques (*M. sylvanus)* were not susceptible to intramuscular, intravenous or intradermal inoculation, though they sometimes became ill if the virus was injected directly into the brain or sciatic nerve. However, a later study reported successful intranasal inoculation of grivet monkeys (*Cercopithecus*

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aethiops) and squirrel monkeys (*Saimiri sciureus*) with a laboratory-adapted strain of pseudorabies virus. Rats, mice and guinea pigs, as well as rabbits, can serve as laboratory models. In rabbits, the virus is reported to replicate in a wide variety of tissues, similarly to pigs, though it is generally found only in the CNS of most incidental hosts.

The virus's ability to infect birds is unclear. One fatal outbreak in 2-day old chickens was attributed to a Marek's disease vaccine contaminated with a laboratory-grown pseudorabies virus adapted to chicken cells. The illness was not contagious, and it could be reproduced by intramuscular injection but not oral inoculation of young chicks. Reports of experimental infections in other birds, such as pigeons, do not confirm their susceptibility as they described animals injected directly into the brain.

Zoonotic potential

Until recently, pseudorabies virus was not considered to be zoonotic. Although a few, mostly older, reports proposed a causative role for this virus in various self-limited illnesses, these cases were not confirmed by virus detection, and they were generally dismissed as coincidences. Since 2017, however, Chinese researchers have published a number of case reports that attribute viral encephalitis and/or ocular lesions to pseudorabies virus, in particular the variant viruses circulating in China. All of these cases have been diagnosed with a novel, very sensitive technique that can detect small fragments of nucleic acids, with limited supportive evidence from other diagnostic methods. Additional information about these and other proposed human cases is available in the Public Health section, below.

Geographic Distribution

Pseudorabies virus is completely absent from a few countries, such as Australia and Greenland, but it has been eliminated or mostly eliminated from domestic pigs in a number of other locations including New Zealand, parts of the Americas including most of North America, some countries in Europe, and some Asian nations. It often continues to circulate in wild suids in many of these locations. Pseudorabies can still be found in domestic pigs in some countries, especially in Asia, Latin America and Africa.

Transmission

Domestic pigs are usually thought to become infected via inhalation or ingestion, but venereal transmission is also possible, and fetuses can be infected *in utero*. In addition to respiratory secretions and saliva, pseudorabies virus has been found in milk, urine, vaginal secretions and semen. Infected tissues from suids or incidental hosts can transmit the virus to pigs if they are eaten. Aerosols are thought to account for some short-distance transmission indoors and might also spread the virus over longer distances outdoors, though the latter is controversial. Similar mechanisms are probably responsible for virus transmission in feral pigs and wild boars, though the relative importance of some routes may differ. Both domestic pigs and wild boars can become inapparent carriers, with the virus becoming latent in nerve ganglia near the site of virus entry. Latent viruses can be shed if they are reactivated by stressors such as transport, crowding, farrowing or corticosteroid administration.

Incidental hosts usually become infected during close contact with pigs or by eating contaminated raw porcine tissues. Although there are rare exceptions (e.g., some experimentally infected lambs), incidental hosts do not usually spread the virus to other animals even when small amounts of virus can be found in nasal or oral secretions.

Pseudorabies virus has been reported to remain viable for as long as several days to 2 weeks on various fomites at 20-26°C (68-79°F), and for a month in pig slurry, with some reports of longer survival if the fomites were spiked with large amounts of virus or placed in a sealed, humid environment. The virus can persist longer when temperatures are very cold, and survives freeze-thaw cycles. At 26°C, it survived for 20-35 days in a liquid medium in the laboratory, but exposure to direct sunlight (38-39°C/ 100-102°F) destroyed the infectivity of this solution within 15 minutes. One study, which used an environmental chamber programmed to simulate conditions during a winter shipment of 37 days, detected infectious virus in a number of sterilized feed or feed ingredients at the end of this time. However, the presence of environmental microorganisms can sometimes greatly reduce pathogen survival, and its persistence in unsterilized feed ingredients under transport conditions remains to be determined.

Disinfection

Pseudorabies virus can be inactivated by a number of disinfectants including sodium hypochlorite, sodium or calcium hydroxide, chlorhexidine, quaternary ammonium compounds, phenolics, ethanol, iodine and potassium permanganate. Powdered laundry detergent was also reported to be effective. Although this virus is only stable between at pH 5 and 9, inactivation by acids or alkali is reported to be variable and prolonged treatment may be necessary. Viruses in cell culture medium can be inactivated in a few seconds by heating the solution to 70℃ (158°F), while viruses in pig swill required 5-10 minutes at 70-80℃ (158-176°F). UVC light is reported to be effective against pseudorabies virus, though UVA and UVB were not.

Incubation Period

Infections in pigs often become apparent in about 2-6 days. The incubation period is thought to be less than 9-10 days in most incidental hosts, with dogs and cats commonly developing the first signs within a few days.

Clinical Signs

Suids

The clinical signs of pseudorabies in pigs are influenced by their age. Suckling pigs without maternal antibodies often develop neurological signs, as well as fever and other nonspecific signs of illness (e.g., anorexia, lethargy), and in some cases, vomiting and diarrhea or constipation. In very young animals (i.e., < 1-2 weeks of age), the initial signs of illness can be quickly followed by tremors, seizures, opisthotonos, paresis, hindleg paralysis or other signs of CNS involvement, with affected animals usually dying within 24-36 hours. Sudden deaths are also possible, and some individuals may die with only nonspecific signs of illness. The incidence of neurological signs and mortality rate are lower in slightly older piglets, which can also have respiratory signs.

In weaned (grower-finisher) pigs, pseudorabies is mainly a respiratory illness characterized by fever, anorexia, weight loss, nasal discharge, sneezing and coughing, which can sometimes progress to dyspnea. Some pigs may also have conjunctivitis, vomiting, diarrhea and /or constipation, and neurological signs are seen occasionally. Affected animals tend to recover within a week or two, though secondary bacterial infections, including pneumonia, can be a complication. Adult pigs usually have inapparent infections or mild illnesses, with respiratory signs predominating, though there have been reports of neurological signs ranging from mild muscle tremors to convulsions. Pregnant sows may reabsorb infected fetuses, abort or give birth to weak, trembling neonates. Affected litters can contain a mixture of normal piglets, stillborn piglets and weak piglets.

Infections in feral swine and wild boar seem to be asymptomatic or mild in many cases. Mild respiratory signs are the most commonly reported syndrome, though neurological signs have been seen, especially in very young animals.

Other animals

Pseudorabies in incidental hosts is characterized by neurological signs that can include behavioral changes, ataxia, paralysis/ paresis, laryngeal and pharyngeal spasms, proprioceptive deficits and convulsions. The CNS signs are often accompanied by localized pruritus, which manifests as severe licking, rubbing or gnawing, and often leads to self–mutilation. Pruritus might be less common in some species than others. A prodromal syndrome, with nonspecific signs such as fever, anorexia and lethargy, may be seen in some animals before the neurological signs develop. Excessive salivation is common after the CNS is affected, and ruminal atony may be noted in livestock. There have also been reports of vomiting, diarrhea or respiratory signs (nasal discharge, coughing, wheezing, dyspnea) in some animals, and a few dogs developed bloody diarrhea and/or hematemesis. Reports of survival are rare, with most affected animals dying within a few days.

The only known outbreak in birds occurred when a virus adapted to growth in chicken cells was apparently inoculated into day-old chicks via a contaminated vaccine. In this outbreak, the chicks developed progressive ascending paralysis with trembling and a high case fatality rate.

Post Mortem Lesions

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Suids

Gross lesions are often subtle, absent or difficult to find in pigs, and the lesions that occur may be nonspecific. Many animals have serous or fibrinonecrotic rhinitis, but this may be apparent only if the head is split and the nasal cavity opened. In some cases, there may also be congested meninges; pulmonary edema, congestion or consolidation; necrotic tonsillitis or pharyngitis; or congested lymph nodes with small hemorrhages. Necrotic foci are sometimes found in the internal organs, particularly in very young piglets or fetuses, and are most likely to be detected in the liver and spleen.

Other animals

The CNS lesions in the brain and/or spinal cord of incidental hosts are mainly characterized by edema, congestion of the meningeal vessels and/or multifocal hemorrhages. Traumatic lesions as a result of the intense pruritus are also common. Many animals have few or no other gross lesions; however, there are some reports of pulmonary edema, hemorrhages or congestion, and endocardial, epicardial and/or thymic petechiae and ecchymoses. More extensive hemorrhagic signs in various internal organs were described in some farmed mink, dogs and experimentally infected cats in China.

Diagnostic Tests

Suids

Pseudorabies virus, its nucleic acids and antigens may be found in nasal swabs, oropharyngeal fluid, and swabs or biopsies of the tonsils in sick pigs, and in various tissues, especially the brain, spleen, lung and tonsil, at necropsy. Latently infected pigs are most likely to be identified by serology, or by examining the trigeminal ganglia (or the sacral and/ or trigeminal ganglia in wild suids) by PCR. Live virus generally cannot be recovered from these animals with ordinary culture techniques.

Pseudorabies virus can be isolated in a number of cell lines or primary cell cultures, though porcine kidney cells (e.g., the PK-15 cell line) are used most often. Recovered viruses can be identified by immunofluorescence or immunochemical staining, virus neutralization, and genetic assays such as PCR. PCR can also identify viral nucleic acids directly in clinical samples. Most PCR tests can detect the viral variants circulating in China, but tests that can distinguish these strains from classical pseudorabies virus isolates have also been published. Viral antigens can be found in clinical samples by immunostaining.

Various serological tests including ELISAs, latex agglutination and virus neutralization can detect antibodies to pseudorabies virus in serum. Some of these tests can distinguish vaccinated from infected pigs, if gene-deleted vaccines are used. Paired titers can be used to confirm a recent infection. Some serological tests can also be employed with whole blood, milk, muscle exudates (meat juice) or oral fluids in surveillance.

Other animals

Pseudorabies in incidental hosts can be diagnosed by virus isolation, PCR and/or antigen detection tests on CNS samples. The brain, particularly the brainstem, is used most often, but affected sections of the spinal cord were found to be valuable in some cattle, and the virus can also be found sometimes in peripheral ganglia, such as the trigeminal ganglia. It has also been detected occasionally in other tissues such as the nasal or oropharyngeal mucosa, tonsil, salivary gland, oral fluid, pruritic areas of skin, and internal organs (e.g., lung, heart, stomach, adrenal gland) in some species, though this is not reliable. Serology is not expected to be helpful in incidental hosts, as these animals usually die before mounting an antibody response

Treatment

Treatment of affected animals is generally limited to supportive care and treatment for secondary infections.

Control

Disease reporting

Veterinarians who encounter or suspect pseudorabies should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Attenuated, inactivated or gene-deleted marker vaccines can be used to protect pigs from clinical signs in endemic areas, but do not provide sterile immunity or prevent latent infections. The gene-deleted vaccines allow vaccinated pigs to be distinguished from pigs infected with field viruses.

Pseudorabies can be eradicated from a herd by depopulation, followed by cleaning, disinfection, and restocking with virus-free animals, but other eradication strategies such as test-and-removal or offspring segregation have also been published. Preventive measures for pseudorabies-free herds in an endemic area include isolation and testing of new animals before they are added to the herd, and biosecurity measures to prevent entry via contaminated fomites, people and incidental hosts such as infected rodents. In areas where the virus has been eliminated from domestic pigs but not wild suids, biosecurity measures are primarily focused on protecting the herd from contact with the latter animals (e.g., with a double fence system), together with strict sanitation. Because pseudorabies infections acquired from wild suids can be inapparent, it may be necessary to periodically monitor higher risk herds with laboratory tests.

Preventive measures in incidental hosts are based on avoiding contact with potentially infected swine, including wild suids, and their tissues. In particular, raw tissues from suids that might be infected should not be fed to carnivores. Vaccines are not available for animals other than pigs, and the attenuated viruses in swine vaccines are reported to cause pseudorabies in some species.

Morbidity and Mortality

Pseudorabies viruses differ in virulence, and the importance of this disease can vary between regions. The variants that emerged in China in 2011 have been reported to be more virulent than some classical strains, while at least some viruses circulating in wild suids seem to be relatively attenuated. Mortality is strongly age-dependent. A naive herd infected by a virulent virus can have a mortality rate as low as 1-2% in grower/ finisher pigs and 5-10% in weaner pigs, but up to 50% or more in nursing piglets, and as high as 100% in animals < 2 weeks of age, while infections in adult pigs are not usually fatal. However, particularly virulent viruses can cause deaths even among adult swine, while some mild variants are characterized by mild signs and asymptomatic infections except in very young animals. Losses are also influenced by previous exposure, and piglets born to immune sows may be protected by maternal antibodies up to 4 months of age. The abortion rate in pregnant sows is generally 20% or less. Severe illnesses seem to be rare in wild suids, but whether they are inherently more resistant to pseudorabies than domestic pigs is still unclear.

Cases in incidental hosts are often sporadic, but historical outbreaks in cattle sometimes affected 3-60% of the herd, and large outbreaks with 80-90% mortality have been seen in farmed mink and foxes fed contaminated pig tissues. How often incidental hosts become infected after exposure to infected pigs is unclear. Some authors indicate that relatively large amounts of virus may be necessary to infect most animals, and calves in direct contact with experimentally infected pigs for several weeks sometimes remained healthy even when the virus spread to contact pigs. Clinical cases in incidental hosts, including those caused by viruses from wild suids, are usually fatal. There are, however, rare reports of survival, and a few studies have documented antibodies to pseudorabies in wildlife and hunting dogs, at rates ranging from 1% (hunting dogs) to 10% (wild foxes).

Public Health

Until recently, it was generally accepted that humans are not susceptible to pseudorabies virus. A very small number of case reports published between 1914 and 1992 described possible pseudorabies-associated illnesses in people exposed to infected animals, such as laboratory workers injured when handling experimentally infected dogs or cats. However, none of these cases resembled the illnesses seen in other incidental hosts, and most were characterized by various combinations of nonspecific, self-limited signs such as fever, headache, sore throat, lethargy/ weakness, myalgia, arthralgia, tinnitus, diarrhea and/or aphthous stomatitis (canker sores), which can be caused by a number of common human illnesses and could have been coincidental. Pruritus accompanied other signs of a wound infection in one case, which is not unusual. In another proposed incident, six people had self-limited pruritus of the arm and shoulder after handling a herd of pruritic cattle with pseudorabies. None of the cases were confirmed by detecting the virus, its antigens or nucleic acids. While one report described low antibody titers to pseudorabies virus in three people with nonspecific clinical signs, this could have been caused by cross-reactivity to human herpes simplex virus 1. Parenteral inoculation of pseudorabies virus into two human volunteers (apparently self-inoculation) resulted in no illness.

In 2017, a case report from China described possible pseudorabies in a swine worker who had pig sewage spilled on her body, including in her eyes. She developed a fever and headache the day after the incident, followed by visual impairment. Antibiotics and an antiviral agent (acyclovir) were administered, but the clinical signs persisted and endophthalmitis with retinal necrosis was diagnosed 2 weeks later. Culture of the vitreous humor, collected during vitrectomy, did not reveal any organisms at this time; however, multigenomic next generation sequencing (mNGS) found a strong signal for pseudorabies virus in the vitreous humor, as well as weak signals for bovine herpesvirus 5, several environmental bacteria, *Cryptococcus gattii*, *Corynebacterium urealyticum* and the common intestinal organism *Brachyspira pilosicoli*. The patient's fever and headache resolved after the vitrectomy and treatment with a different antiviral drug (valacyclovir), and she was later found to have antibodies to pseudorabies virus in blood and CSF. Whether she was seropositive before the illness was not known, and the authors indicated that antibodies to this virus have been detected in 5-90% of some swine-exposed populations tested in China,

This report was followed by the publication of at least 26 case reports of encephalitis and/or ocular signs attributed to the pseudorabies viruses circulating in China. Almost all of these patients were known to have direct contact with pigs or their tissues. Most cases began with febrile cold- or flu-like symptoms before progressing to neurological signs (most often seizures, headache and/or disorders of consciousness), and many were preceded or accompanied by respiratory signs, including severe pneumonia. Some patients also developed various ocular lesions such as endophthalmitis, retinal vessel occlusion, retinal detachment and/or retinal necrosis, during the course of the illness. The neurological signs varied from mild to severe, and a number of patients recovered, though some cases were fatal and the survivors sometimes had serious residual CNS or ocular deficits. The course of the illness was prolonged in a number of cases, with improvement then recurrence of the neurological signs. Most patients were given antiviral drugs, in addition to symptomatic and supportive treatment. Most also received other agents including steroids, antibiotics or unspecified anti-infective agents, and intravenous immunoglobulin."

All of these cases were initially diagnosed with a novel genetic technique known as multigenomic next generation sequencing (mNGS). mNGS uses very high throughput sequencing to generate short sequences from all of the DNA in a sample, including that of host cells and any microorganisms that may be present. The number of unique reads (short sequences attributed to pseudorabies virus) found in the reported clinical cases ranged from a single read to tens of thousands, and the percentage of the pseudorabies genome covered by the detected fragments varied from 0.02% to 84%. However, the majority of the papers detected < 5% of the pseudorabies genome, including some that found < 1%. The authors of one report with limited (2.5%) coverage noted that the reads were nevertheless distributed over the entire pseudorabies genome, suggesting that more than a fragment was present, but most papers did not provide this information. A few reported evidence for nucleic acids in the eye but not the brain in cases of encephalitis with ocular complications.

Metagenomic NGS is a promising technique for identifying unexpected causes of an illness, and might be particularly valuable in diseases such as encephalitis, where a specific etiology is never identified in many cases. However, mNGS is also very sensitive and subject to false positives from a variety of sources including the reagents used, environmental contaminants (e.g., agents on the skin during sample collection) or even rare coincidental contamination by nucleic acids in body fluids. In addition, the readout is based on matching the sequences in a sample to those that have been uploaded to genome databases, and it can be influenced by any errors in those databases. Accidental matches of low-complexity sequences to low-quality reads from the sample are a particular concern. For these reasons, experts recommend that reports of unusual pathogens based on mNGS be confirmed by conventional means.

A few of the case reports from China reported confirming the mNGS results by PCR, but the presence of nucleic acids alone cannot determine whether live virus is present. A few authors found antibodies to pseudorabies virus in the blood, and sometimes the CSF, of patients; however, the absence of paired titers or determination of the antibody class (i.e., IgM), together with reports of antibodies in some healthy swine workers in China, makes this finding difficult to interpret in nearly all of the cases. One report did describe seroconversion in a person who had been seronegative 2 weeks after the onset of clinical signs, which may be suggestive, and another reported a slight rise in serum antibody levels between days 14 and 21/ 28. None of the authors reported the blood/CSF antibody ratio, which can help distinguish a CNS infection from the entry of antibodies into CSF after disruption of the blood/brain barrier by inflammation. One additional concern with the serological results is that many papers did not specify the assay employed, and some apparently used a commercial ELISA from IDEXX Laboratories, which is designed to detect pseudorabies antibodies in pigs and does not appear to have been validated for human samples. Cross-reactivity with human herpesviruses may also be a concern, as in an older report.

Whether postmortem clinical samples were ever tested for viral antigens, nucleic acids or live virus is unclear. Only one paper has described isolating pseudorabies virus from a patient. These authors found the virus in a sample of CSF in one of 4 patients. This sample was taken more than 40 days after the onset of neurological signs, during a period when mNGS found low levels of nucleic acids in the CSF on 2 days and in throat swabs on 3 days. However, all other CSF samples (which were apparently collected every 2-4 days before and after this time) were negative by mNGS, including samples taken during the first week of neurological signs. At that time, mNGS did detect pseudorabies nucleic acids in the blood, but virus could not be isolated from the blood. No virus could be isolated from the throat swabs with positive mNGS readings or from any samples in the other three patients. These and other irregularities in this report, including very limited information on the course of the illness, suggest that additional studies should be pursued for definitive confirmation of pseudorabies as a human pathogen.

Internet Resources

The Merck Veterinary Manual

The Pig Site

United Kingdom. Department for Environment, Rural and Food Affairs (DEFRA). Aujeszky's disease in Europe

United States Department of Agriculture (USDA). Pseudorabies

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet. Spickler, Anna Rovid. 2024. *Aujeszky's Disease*. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

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