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PSEUDORABIES VIRUS INFECTION IN RACCOONS: A REVIEW

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INTRODUCTION

Pseudorabies (Aujeszky's Disease) is an infectious viral disease. It primarily attacks swine, but a wide range of domestic and wild animal species also are susceptible to infection.^{5,6,12} The clinical signs of raccoons infected with pseudorabies virus (PRV) are similar to those of rabies and distemper. Wildlife authorities should be familiar with these diseases to insure accurate diagnosis and protect public health. A November, 1980 episode of distemper in raccoons in eastern Missouri was marked by confusion between the raccoons' clinical signs and those of PRV infection. Considerable controversy arose then over the importance of pseudorabies in raccoons. The purpose of this review is to summarize the current information on this subject.

SUSCEPTIBILITY TO INFECTION

Involvement of raccoons in outbreaks of pseudorabies has been suggested by researchers.^{2,8} Sick, dead and dying raccoons have been found on or near swine-rearing premises that have experienced pseudorabies in swine. Pseudorabies in raccoons never has been reported without a concurrent infection in swine nearby. The disease may be spread through the common practice of tem-

porarily disposing of baby pig carcasses or placentas in a manner which allows their consumption by wild animals. All reported cases of pseudorabies in raccoons has occurred following outbreaks in swine.

Raccoons often live in close association with man and livestock. Investigators have studied the raccoon's possible role in the transmission of pseudorabies among swine herds.^{8,13} Laboratory investigations have revealed raccoons are susceptible to high doses of PRV.¹³ These studies found that raccoons infected by oral inoculation of PRV consistently showed clinical signs and died when dosed with levels of 10^5 TCID₅₀ (Tissue Culture Infective Doses) and above of virus (Table 1). All five raccoons given 10^3 TCID₅₀ of PRV survived and did not develop signs of PRV infection. Six of nine raccoons exposed with 10^4 TCID₅₀ of PRV also survived.

In studies of the potential for PRV transfer between raccoons, investigators placed animals showing clinical signs of pseudorabies in cages with healthy raccoons. The in-contact raccoons did not develop clinical signs of PRV. This indicated that PRV is unlikely to transfer from raccoon to raccoon. Wright and Thawley¹³ found that infected raccoons excrete PRV in oral discharges. Since

TABLE 1. Death rate data of raccoons orally exposed with pseudorabies virus (PRV).

Item	Virus Dose in TCID ₅₀			
	10 ³	10 ⁴	10 ⁵	10 ⁶
Raccoons inoculated	5	9	13	4
Survivors	5	6	0	0
Dead	0	3	13	4
Death rate %	0	33	100	100

direct transfer of virus does not readily occur, it may be assumed that viral excretion from raccoons is not sufficient to provide infectious levels for other raccoons, and the virus "dead ends" with the infected raccoon.

CLINICAL SIGNS

A wide-range of clinical signs may be shown by raccoons infected with PRV.^{8,12,13} Depression, with an intense localized pruritis, is the most frequently observed clinical sign in experimentally infected raccoons. Signs of central nervous system dysfunction, which include convulsions, disorientation and incoordination, also have been observed consistently. Respiratory signs such as coughing and wheezing, together with profuse oral and nasal discharge also may occur. In studies performed by Wright and Thawley,¹³ 26 raccoons were experimentally infected with oral doses of PRV; 20 showed clinical signs and died. Pruritis localized about the head, muzzle or extremities was observed in nine of these raccoons (45%), respiratory distress was observed in seven of the 20 raccoons (25%). About 50% of the raccoons in this study, and nine of 10 raccoons studied by Trainer and Karstad,¹² died showing signs only of depression.

Since a wide range of clinical signs are shown by raccoons infected with PRV, other diseases easily may be mistaken for pseudorabies. Clinical signs of canine distemper (CD) most often are confused

with those of PRV infection. Information concerning the epidemiology of CD virus among wildlife populations is limited. Serologic surveys of raccoons in New York and Maryland found the prevalence of CD neutralizing antibody titers ranged from 22% to 84%.^{4,10} This would indicate CD infection is common among raccoon populations. Experimental infection of raccoons with CD virus has indicated an incubation period of 11 to 30 days.³ Clinical cases of CD observed in the field, and following experimental infection, showed similar signs of dyspnea, ataxia, clonic convulsions, ascending paralysis, progressive blindness, loss of appetite, emaciation, diarrhea and coma.^{3,7} These signs also are consistent with PRV or rabies virus infection.

PATHOLOGY AND DIAGNOSIS

Pseudorabies infection does not produce distinct pathology in the raccoon.^{8,13} However, the virus may be isolated from the tissues following death. Wright and Thawley¹³ found the virus was isolated most consistently from the brain and tonsils (Table 2). Lung and salivary glands were less reliable for PRV isolation. The isolated virus could be kept at room temperature (24C) for five days following death.¹³

In contrast, the pathology of CD infection in raccoons shows gross lesions lacking or confined to lung congestion.³ Microscopic lesions of interstitial pneumonia may be observed with

TABLE 2. Virus isolation attempts from tissues of raccoons that died after exposure to PRV.

Tissue	Virus dose in TCID ₅₀		
	10 ⁴	10 ⁵	10 ⁶
Brain	3*/3+	11/12	4/4
Tonsils	3/3	8/11	3/4
Salivary glands	2/3	2/6	0/1
Lungs	2/3	7/12	1/4

* No. of raccoons from which PRV was isolated.

+ No. of raccoons from which isolation was attempted.

characteristic eosinophilic cytoplasmic inclusion bodies present in the epithelial cells of the bronchi, bile duct, renal pelvis and urinary bladder. Diffuse focal areas of demyelination and malacia may be observed in sections of the cerebrum and midbrain. A diagnosis of CD may be based on the characteristic histopathologic changes or on isolation of the CD virus.

Rabies may be differentiated from pseudorabies and CD by the pathognomonic brain changes associated with rabies infection.¹¹

FIELD EPIDEMIOLOGY

A feature that characterizes infection with PRV is the high mortality of affected raccoons. In experimental studies, all raccoons that have been successfully infected have died within seven days of showing clinical signs. In field serologic surveys performed in PRV endemic areas, no evidence of previous PRV infection could be found in live healthy raccoons.¹³ This would indicate that the raccoon most likely acts as a "dead end" host and plays no important role in the maintenance of PRV in nature. Serologic evidence suggests CD and rabies viruses are maintained in raccoon populations.^{1,3,9,10} The only reported field cases of raccoon pseudorabies have been from areas that simultaneously were reporting clinical

pseudorabies in swine. The authors consider this the most likely source of infection for the reported field raccoon cases.

SUMMARY

Pseudorabies is a rarely reported disease of raccoons. Laboratory and field evidence of PRV infection suggests the raccoon is a "dead end" host with little opportunity for raccoon-to-raccoon spread of virus. All reported field cases have been associated closely with infected swine and swine have been considered the source of the raccoon infection.

The clinical signs of PRV in raccoons closely resembles those of canine distemper and rabies virus infections. Infection with the latter viruses are considered more prevalent and likely to be mistaken for PRV infection. Both CD and rabies virus may be maintained in raccoon populations with raccoon-to-raccoon transfer while PRV may not. Differentiation of PRV, CD and rabies infections is best achieved by histopathologic analysis of lung and brain tissue, together with virus isolation.

It is of utmost public health importance that wildlife authorities recognize the similarities between these diseases, together with the different epidemiologic behavior of the viruses and the means to differentiate clinical cases.

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