# Rabies in North American Insectivorous Bat Colonies

*Why a single rabid bat does not imply a rabid colony: prevalence data, routes of infection, and the biological brakes on bat-to-bat transmission in summer maternity roosts*

## 1. The Central Question

When one bat from a building roost or maternity colony tests positive for rabies virus (RABV) by the direct fluorescent antibody (DFA) test, a common assumption is that the rest of the colony is likely infected as well. The peer-reviewed literature does not support that assumption. Active RABV infection in apparently healthy, free-ranging insectivorous bats in North America is consistently reported at well under 1%, and the detection of one infected individual is not evidence of elevated prevalence in the group from which it came.

## 2. What the Prevalence Data Actually Show

**Passive surveillance is severely biased upward.** Bats submitted to public health laboratories are, by definition, bats that were grounded, active by day, found indoors, or captured by a pet — all behaviors characteristic of clinical rabies. Roughly 5–10% of such submissions test positive; in 2020 about 6% of ~24,000 bats tested in the United States were positive, and bats accounted for 1,218 of 3,234 rabid wildlife reported in 2022 [1]. These figures describe a self-selected sick population, not colonies.

**Active (unbiased) sampling gives a far lower figure.** Klug et al. tested tree-roosting bats collected as wind-turbine mortalities — a sample not filtered by human contact — and found 0 of 121 hoary bats and 1 of 96 silver-haired bats positive (~1% and 0%), prevalence significantly lower than both passive and prior active estimates. The authors attributed the discrepancy to submission bias and concluded that reported rabies prevalence in bats has been systematically overestimated [2]. Across active surveys of healthy insectivorous bats, prevalence is generally below 0.5–1%.

**Exposure is common; fatal infection is rare.** In a five-year study of marked big brown bats (*Eptesicus fuscus*) occupying summer building roosts in Fort Collins, Colorado, O'Shea et al. found rabies virus neutralizing antibody (RVNA) in 17.9% of adult females, 10.2% of volant juveniles, and 9.4% of adult males, with roost-level seroprevalence ranging from 0% to 47.1%. Cumulative seroprevalence in adult females aged ≥2 years reached 21–48%. Critically, seropositive bats survived and were repeatedly recaptured over subsequent years — seropositivity marks non-lethal exposure, not infection [3]. Steece and Altenbach reported the same decoupling in a large *Tadarida brasiliensis mexicana* cave colony, where brain-antigen positivity was on the order of ~2% or less while antibody prevalence was substantially higher, with maternal antibody detectable in juveniles [4].

## 3. How Bats Actually Contract Rabies

- **Bite inoculation from an infected conspecific.** RABV is present in the saliva of a clinically ill bat and must be introduced through broken skin. Intraspecific bites occur during roost jostling, competition for roost position, mating, and defensive interactions. Wing-membrane puncture wounds the size of big brown bat canine tips are used by researchers as a proxy for recent bat-to-bat biting [3].
- **Bat RABV variants are host-restricted.** Streicker et al., analyzing hundreds of viruses from 23 North American bat species, showed that cross-species transmission and host establishment decline sharply with increasing phylogenetic distance between bat species. Each variant largely circulates within its own reservoir species, which limits amplification in mixed-species roosts [5].
- **Aerosol transmission is an extraordinary event, not a colony norm.** The airborne cases on record derive from caves holding millions of Mexican free-tailed bats (notably Frio Cave, Texas), where extreme bat density, heat, ammonia, and confinement combine. Reviews conclude aerosol transmission is not documented under ordinary natural conditions, and the near-absence of rabies among cavers argues against it [6]. It has no meaningful application to an attic, barn, or bat-house maternity colony of tens to a few hundred bats.
- **Not transmitted by guano, urine, blood, fur, or casual proximity.**

## 4. Mechanisms That Limit Bat-to-Bat Transmission

Five interacting biological constraints keep RABV at low prevalence within a summer colony:

- **There is no carrier state, and the infectious window is very short.** In experimental infection of big brown bats, 16 of 20 inoculated bats developed clinical rabies (mean incubation 24 days, range 13–52). Virus was detected in saliva of only two bats, and only within 24 hours before onset of clinical signs. No bat-to-bat transmission to uninfected cage mates was detected [7]. A bat is infectious for roughly a day or two at the end of a weeks-to-months-long incubation, then dies.
- **Clinical illness removes the bat from the cluster.** Bat rabies is predominantly paralytic. Affected bats lose coordinated flight, become moribund, and fall from or leave the roost — which is precisely why the bats humans encounter are grounded ones. The animal is physically separated from roost mates during the brief period it can transmit.

- **Even direct exposure in confinement often fails to transmit.** Shankar et al. group-housed a captive colony of wild-caught big brown bats. Two bats succumbed to rabies within the first month, and one of the rabid bats was observed biting her cage mates — yet none of the remaining bats developed rabies over the course of the study [8].
- **Repeated low-dose exposure immunizes rather than kills.** Outcome is strongly dose-dependent: lower intramuscular doses produced ~57% mortality with surviving bats seroconverting [7]. Turmelle et al. showed that big brown bats surviving primary RABV infection and re-challenged had reduced mortality after a third challenge despite a high viral dose, demonstrating protective immunological memory from abortive infection [9]. High maternity-colony seroprevalence is therefore evidence of an increasingly immune colony, and maternally derived antibody further protects pups [3, 4].
- **Long, variable incubation desynchronizes infections.** Incubation ranges from ~2 weeks to many months; a captive big brown bat has been documented with a 267-day incubation [10]. Modeling by George et al., parameterized from the Colorado *E. fuscus* population, shows RABV persists precisely because long incubation and low overwinter mortality carry a few infections across the year rather than producing explosive summer epizootics — endemic persistence at low prevalence, not colony-wide outbreak [11]. Roost switching and low within-colony relatedness further dilute transmission chains rather than concentrating them [12].

## 5. Interpretation and Caveats

A single DFA-positive bat from a summer maternity colony or small roost group indicates that RABV is circulating in that regional bat population — which is expected — but provides little information about the infection status of its roost mates. The best available estimate for any given apparently healthy colony mate remains the population base rate of well under 1%, with a substantially larger but non-infectious fraction likely seropositive from survived exposure.

Three caveats apply. First, seroprevalence is not infection prevalence; the two must not be conflated. Second, artificial high-density captive housing can produce transmission dynamics unlike those in wild roosts, as in a documented outbreak in which 5 of 27 captive big brown bats in a white-nose syndrome vaccine trial became infected — all confined to the single cage housing the index case [13]. Third, low colony prevalence does not alter individual human or pet exposure management: any direct contact, bite, or unattended exposure to a bat should be evaluated under current ACIP and NASPHV guidance, and the bat tested when available. Low population prevalence is a statement about bats, not a substitute for post-exposure risk assessment in people.

## References


1. Ma X, Bonaparte S, Corbett P, et al. Rabies surveillance in the United States during 2022. J Am Vet Med Assoc. 2024;262(11):1518–1527. doi:10.2460/javma.24.05.0354
2. Klug BJ, Turmelle AS, Ellison JA, Baerwald EF, Barclay RMR. Rabies prevalence in migratory tree-bats in Alberta and the influence of roosting ecology and sampling method on reported prevalence of rabies in bats. J Wildl Dis. 2011;47(1):64–77. doi:10.7589/0090-3558-47.1.64
3. O'Shea TJ, Bowen RA, Stanley TR, Shankar V, Rupprecht CE. Variability in seroprevalence of rabies virus neutralizing antibodies and associated factors in a Colorado population of big brown bats (Eptesicus fuscus). PLoS One. 2014;9(1):e86261. doi:10.1371/journal.pone.0086261
4. Steece R, Altenbach JS. Prevalence of rabies specific antibodies in the Mexican free-tailed bat (Tadarida brasiliensis mexicana) at Lava Cave, New Mexico. J Wildl Dis. 1989;25(4):490–496. doi:10.7589/0090-3558-25.4.490
5. Streicker DG, Turmelle AS, Vonhof MJ, Kuzmin IV, McCracken GF, Rupprecht CE. Host phylogeny constrains cross-species emergence and establishment of rabies virus in bats. Science. 2010;329(5992):676–679. doi:10.1126/science.1188836
6. Gibbons RV. Cryptogenic rabies, bats, and the question of aerosol transmission. Ann Emerg Med. 2002;39(5):528–536. doi:10.1067/mem.2002.121521 (see also Constantine DG, Rabies transmission by nonbite route, Public Health Rep. 1962;77:287–289; Winkler WG, Airborne rabies virus isolation, Bull Wildl Dis Assoc. 1968;4(2):37–40)
7. Jackson FR, Turmelle AS, Farino DM, Franka R, McCracken GF, Rupprecht CE. Experimental rabies virus infection of big brown bats (Eptesicus fuscus). J Wildl Dis. 2008;44(3):612–621. doi:10.7589/0090-3558-44.3.612
8. Shankar V, Bowen RA, Davis AD, Rupprecht CE, O'Shea TJ. Rabies in a captive colony of big brown bats (Eptesicus fuscus). J Wildl Dis. 2004;40(3):403–413. doi:10.7589/0090-3558-40.3.403
9. Turmelle AS, Jackson FR, Green D, McCracken GF, Rupprecht CE. Host immunity to repeated rabies virus infection in big brown bats. J Gen Virol. 2010;91(9):2360–2366. doi:10.1099/vir.0.020073-0
10. Davis AD, Dupuis M, Rudd RJ. Extended incubation period of rabies virus in a captive big brown bat (Eptesicus fuscus). J Wildl Dis. 2012;48(2):508–511. doi:10.7589/0090-3558-48.2.508
11. George DB, Webb CT, Farnsworth ML, O'Shea TJ, Bowen RA, Smith DL, Stanley TR, Ellison LE, Rupprecht CE. Host and viral ecology determine bat rabies seasonality and maintenance. Proc Natl Acad Sci USA. 2011;108(25):10208–10213. doi:10.1073/pnas.1010875108
12. Walker FM, Sobek CJ, Platts-McPharlin CE, Chambers CL. Relatedness and genetic structure of big brown bat (Eptesicus fuscus) maternity colonies in an urban-wildland interface with periodic rabies virus outbreaks. J Wildl Dis. 2021;57(2):303–312. doi: 10.7589/JWD-D-20-00112
13. Abbott RC, Saindon L, Falendysz EA, Greenberg L, Orciari L, Satheshkumar PS, Rocke TE. Rabies outbreak in captive big brown bats (Eptesicus fuscus) used in a white-nose syndrome vaccine trial. J Wildl Dis. 2020;56(1):197–202. doi:10.7589/2018-10-258



**Prepared by Discover Bats Ohio**

Discover Bats Ohio — "Discover the Amazing World of Bats." DiscoverBatsOhio.com


### Independence & Disclaimer

*Compiled August 3, 2026 by Todd Cartner from peer-reviewed literature and federal surveillance data, with AI-assisted research and drafting; all sources are cited for verification. Not commissioned or funded by any outside party. This describes rabies prevalence among bats generally and is not medical or veterinary advice — specific human or pet exposures should be evaluated under current ACIP and NASPHV guidance.*