which implies that rapid contact-tracing and preemptive testing are likely required to efficiently control future outbreaks.

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Herpes Simplex Virus 1 in Trigeminal Ganglia of Trafficked Neotropical Primates, Peru, 2024

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We detected herpes simplex virus 1 in the trigeminal ganglia of trafficked Neotropical primates (1 *Aotus azarai*; 3 *Sapajus macrocephalus*) in Peru. Tests also revealed *Saimiriine alphaherpesvirus* 1 in the trigeminal ganglia of 2 *Saimiri* sp. monkeys. Our findings suggest latency and raise concerns about diagnostic standards, viral reactivation, and spillover risks.

Illegal trafficking operations have lead to the extraction of thousands of Neotropical primates (NP) from the Peruvian Amazon, exposing them to humans and other domestic and wild species, creating opportunities for bidirectional zoonotic disease transmission and spillover (*1*). Among humans, herpes simplex virus 1 (HSV-1) infects around 67% of adults globally (*2*) and establishes lifelong latency in the trigeminal ganglia (TG) (*3*). HSV-1 infections in NP can cause mild or severe disease, sometimes leading to ulcerative lesions and neurologic impairment (*4*). Questions remain, however, regarding the ability of HSV-1 to establish latency in NP after natural infection.

During latency in humans, HSV-1 lytic gene expression is suppressed, but the latency-associated transcripts and associated microRNAs remain transcriptionally active (*5*). As a result, production of infectious viral particles ceases (*6*), and diagnostic approaches based on PCR testing of peripheral tissues, blood, or oral swab samples fail to detect latently infected persons. Trafficked NP with undetected HSV-1 infections might be placed into

Table. Herpesvirus detection in various organs and sample sites from trafficked Neotropical primates with *Alphaherpesvirus*-positive trigeminal ganglia, Peru, 2024

		Sample type						
Animal ID	Species	Trigeminal ganglia	Oral swab	Heart	Liver	Spleen	Kidney	Salivary glands
NE-004-24	*Sapajus*	HSV-1	SapLCV1	SapLCV1	NR	NR	NR	SapCMV1
NE-015-24	*Sapajus*	HSV-1	NR	NR	NR	NR	NR	NR
NE-020-24	*Aotus*	HSV-1	NR	NR	NR	HSV-1	HSV-1	NR
NE-033-24	*Sapajus*	HSV-1	NR	NR	NR	HSV-1	NR	NR
NE-011-24	*Saimiri*	SaHV-1	SsciLCV2	SsciLCV2	NR	NR	NR	SsciCMV1
NE-021-24	*Saimiri*	SaHV-1	SbolCMV1	SsciLCV2	NR	SsciLCV2	NR	SbolCMV1

*ID, identification; HSV-1, human simplexvirus 1; NR, negative results; SaHV-1, *Saimiriine* herpesvirus 1; SapCMV1, *Sapajus apella* cytomegalovirus 1; SapLCV1, *Sapajus apella* lymphocryptovirus 1; SbolCMV1, *Saimiri boliviensis* cytomegalovirus 1; SsciCMV1, *Saimiri sciureus* cytomegalovirus 1; SsciLCV2, *Saimiri sciureus* lymphocryptovirus 2.

rehabilitation centers and possibly released into wild populations, posing a threat to primate health and conservation (*7*,*8*).

To assess whether HSV-1 establishes latency in NP, we investigated the presence of viral DNA in the TG (and its absence in other tissues) in 37 trafficked NP carcasses representing 7 species (Appendix Table 1, https://wwwnc.cdc.gov/EID/article/32/5/25-1408-App1.pdf) in Peru. On gross examination, we observed no lesions suggestive of active HSV-1 infection, such as oral mucosal ulcers, and we classified all animals as asymptomatic on the basis of macroscopic findings alone.

We aseptically collected oral swab samples as well as TG, heart, liver, spleen, kidney, and salivary gland samples and preserved all samples in RNAlater (Thermo Fisher Scientific, https://www.thermofisher.com). When fresh carcasses were available, we preserved TG in 10% formalin for histologic confirmation (Appendix Figures 1, 2). In extracting DNA, we used a nested panherpesvirus PCR, targeting a 215–315 bp region of the DNA polymerase gene, as previously described (*9*) (Appendix). TG samples were positive for herpesvirus DNA (Appendix Table 2) in 13 (35%) NP. Sequencing results revealed *Alphaherpesvirinae* DNA from 6 of the samples and *Gammaherpesvirinae* DNA from the other 7 samples.

We identified HSV-1 in the TG of 4 (11%) NP sampled: 1 *Aotus azarai* monkey and 3 *Sapajus macrocephalus* monkey. We noted no HSV-1 in any of the 35 oral swab samples we analyzed, including those from TG-positive primates. In 2 of the 4 NP with HSV-1–positive TG, testing also revealed HSV-1 in the spleen, kidney, or both (Table). We observed *Saimiriine herpesvirus* 1, another alphaherpesvirus species, in the TG of 2 *Saimiri* monkeys.

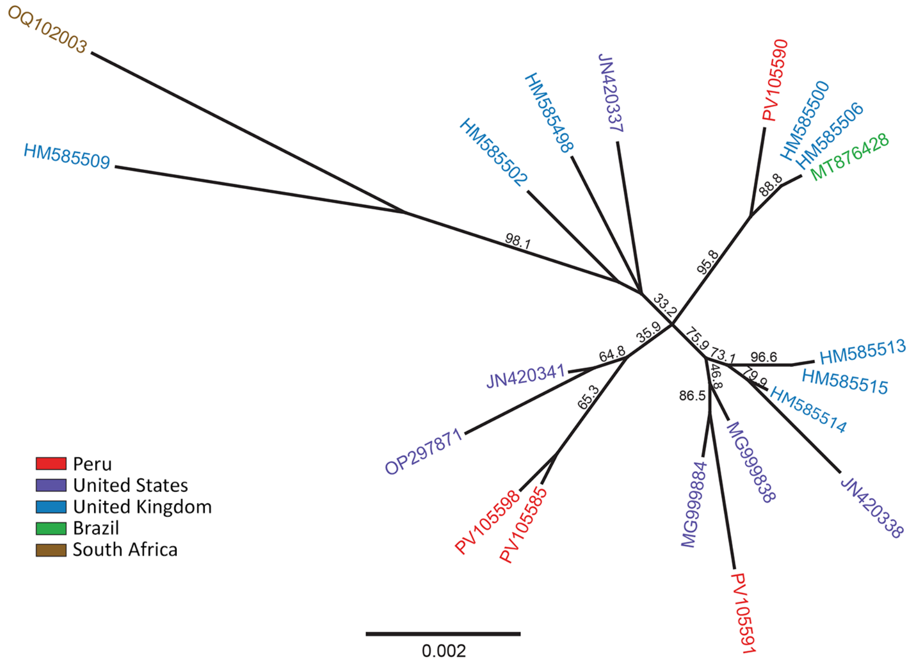

Figure. Maximum-likelihood phylogeny of herpes simplex virus 1 (HSV-1) created for study of HSV-1 in trigeminal ganglia of trafficked neotropical primates, Peru, 2024. Tree constructed from ≈4 kb of the UL30 DNA polymerase gene, applying the general time reversible substitution model with 1,000 bootstrap replicates. Red text indicates full-length HSV-1 DNA polymerase gene sequences detected in trigeminal ganglia of 4 primates from Peru. For comparison, we selected 16 additional reference sequences from GenBank (accession numbers provided) to represent diverse geographic regions worldwide. Phylogenetic analysis suggests a genetically diverse origin of HSV-1 infections in the primates evaluated, likely derived from genetically distinct introductions (spillovers). Sequences deposited in GenBank (accession nos. PV105585 [primate NE-004-24], PV105590 [primate NE-015-24], PV105591 [primate NE-020-24], and PV105598 [primate NE-033-24]).

Phylogenetic analysis of the full HSV-1 DNA polymerase gene (≈4 kb) revealed that 2 sequences (from primates NE-004-24 and NE-033-24) share a recent common ancestor. The remaining sequences are distributed across different branches of the phylogenetic tree, suggesting the infections originated from genetically distinct introductions (spillovers) rather than from a localized outbreak (Figure).

Multiorgan analysis revealed co-infections with 3 distinct herpesviruses, representing all herpesvirus subfamilies (*Alphaherpesvirinae*, *Betaherpesvirinae*, and *Gammaherpesvirinae*), in 3 TG-positive NP (Table). We noted cytomegaloviruses in the salivary glands of 3 of the TG-positive NP, as well as lymphocryptoviruses in the heart tissue, spleen, or both of those same 3 primates. We did not assess the herpesvirus status of organs from TG-negative NP.

Our findings suggest that, as in the case of human infections, HSV-1 may naturally establish latency in the TG of some NP. The detection of HSV-1 DNA in the spleen and kidney does not rule out latency, because latency also has been documented in nonneuronal cells, including neutrophils and B and T lymphocytes (*10*). Confirming true latency in NP would require herpesvirus reactivation studies and RNA sequencing from positive TG.

None of the NP with HSV-1–positive TG showed macroscopic lesions compatible with HSV-1 disease, and we detected no viral DNA in oral swabs. Those findings highlight a critical diagnostic challenge in detecting latent HSV-1 infections in live NP. Oral swab samples can be collected from living primates with minimal distress, but TG can only be obtained postmortem, precluding their use in health evaluations before releasing animals into the wild. Whether latently infected NP can undergo viral reactivation under natural stress conditions and transmit HSV-1 to humans or naive NP remains unknown.

In conclusion, we detected HSV-1 and *Saimiriine herpesvirus* 1 in the TG of NP, consistent with latency. Our findings underscore the relevance of TG as a target tissue for future research and broaden our understanding of the diversity and latency of alphaherpesviruses in NP. Our study also highlights the need for less invasive methods, such as specific antibody profiles or T-cell–specific biomarkers of latency, to identify latent herpesvirus infections in live NP. Identifying such infections can help mitigate potential spillover to other primates, including humans.

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Probable *Bartonella clarridgeiae* Prosthetic Valve Endocarditis and Aortic Root Abscess, Australia, 2020

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We describe a case of endocarditis and aortic root abscess caused by *Bartonella clarridgeiae* bacteria in a patient in Australia. The patient initially sought care for leg pain and was found to have bilateral tibioperoneal trunk mycotic aneurysms. 16S rRNA PCR on excised aneurysm tissue identified the cause as *B. clarridgeiae*.

Bartonella species are gram-negative, fastidious, facultative intracellular bacteria (*1*). They are a cause of blood culture–negative endocarditis, infective endocarditis with negative blood cultures resulting from antibiotic exposure or fastidious pathogens (*2*). *B. clarridgeiae* is an emerging pathogen in the genus, first identified in 1995 and attributed as a human pathogen in 1997 (*3*,*4*). Tibioperoneal trunk (TPT) aneurysms are a rare clinical phenomenon; causes include trauma, vasculitis, and infective endocarditis or mycotic aneurysm (*5*). We describe a case of bilateral TPT aneurysms, prosthetic valve infective endocarditis, and aortic root abscess in a patient in Australia in 2020 that was caused by *B. clarridgeiae*, identified on 16S ribosomal RNA of aneurysm tissue samples.

A man in his 80s sought care at a local emergency department for pain in his left calf for 1 month. He had aortic stenosis requiring a transcatheter aortic valve implantation 2 years earlier. He had sought care several times over the previous 8 months with calf pain, chest pain, and lethargy. On examination, he was noted to have a swollen and tender left calf, a pansystolic murmur, 2 splinter hemorrhages, and a temperature of 37.9°C. He initially received intravenous cefazolin for possible cellulitis, later changed to amoxicillin/clavulanate.

Computed tomography (CT) angiogram demonstrated a hematoma in the left calf associated with a TPT pseudoaneurysm and a right TPT thrombus. The left TPT aneurysm was surgically repaired. Transesophageal echocardiography (TOE) showed no valvular incompetence or vegetations.

CT positron emission tomography scan demonstrated focal intense fluorodeoxyglucose activity at the transcatheter aortic valve implantation (SUVmax = 5.9) (Figure, panel A), postsurgical changes involving the left lower leg (Figure, panel B), and focal moderate activity at the right tibioperoneal trunk, indicating another mycotic aneurysm. We reviewed TOE findings again and identified an echolucent space at the sinus of Valsalva, consistent with an aortic root abscess. Therapy was switched to intravenous ceftriaxone and vancomycin. The patient was deemed not to be a cardiac surgery candidate.

Four sets of blood cultures were negative for bacteremia. Operative tissue culture tested negative for bacterial growth using standard media; we sent the tissue samples for 16S rRNA PCR. We conducted serologic testing for *Brucella* spp., *Coxiella burnettii*, syphilis, and HIV. We tested for *Bartonella* spp. using FOCUS Diagnostics Indirect Immunofluorescence Assay IgG kit for *B. henslae* and *B. quintana* IgG (http://focusdiagnostics.in). 16S rRNA PCR detected *B. clarridgeiae* DNA in operative tissue samples. Serology results for *B. henslae* bacteria were strongly positive (IgG ≥1:2,048 [<128]); all other serology results, including *B. quintana* testing, were negative. We performed PCR testing of blood with primers and probe targeting a conserved portion of the citrate synthase gene; results for *Bartonella* spp. DNA were negative.

We switched treatment to intravenous gentamicin with oral doxycycline (100 mg 2×/d). The patient experienced ongoing fevers and elevated C-reactive protein levels. Because of concern for treatment failure, we added oral ciprofloxacin for 6 weeks; repeated TOE showed stable changes. After 6 weeks, the patient continued oral doxycycline (100 mg 2×/d) for suppression; he remained well at a 2-year follow-up visit.