Nipah virus (NiV) is a zoonotic pathogen that causes high case-fatality rates (CFRs) in humans. Two NiV strains have caused outbreaks: the Malaysia strain (NiV<sub>M</sub>), discovered in 1998–1999 in Malaysia and Singapore (≈40% CFR); and the Bangladesh strain (NiV<sub>B</sub>), discovered in Bangladesh and India in 2001 (≈80% CFR). Recently, NiV<sub>B</sub> in African green monkeys resulted in a more severe and lethal disease than NiV<sub>M</sub>. No NiV vaccines or treatments are licensed for human use. We assessed replication-restricted single-injection recombinant vesicular stomatitis vaccine NiV vaccine vectors expressing the NiV glycoproteins against NiV<sub>B</sub> challenge in African green monkeys. All vaccinated animals survived to the study endpoint without signs of NiV disease; all showed development of NiV F Ig, NiV G IgG, or both, as well as neutralizing antibody titers. These data show protective efficacy against a stringent and relevant NiV<sub>B</sub> model of human infection.

Bats of the genus Pteropus are the primary reservoir in nature for NiV (3), but several other mammal species can be infected by NiV (4–7). Analysis of NiV genomes has identified 2 NiV strains responsible for outbreaks: Malaysia strain NiV<sub>M</sub> and Bangladesh strain (NiV<sub>B</sub>). NiV<sub>M</sub> caused the first identified outbreak of NiV during 1998–1999 in Malaysia and Singapore (≈270 persons infected; CFR ≈40%) (8,9) and perhaps was responsible for a 2014 outbreak in the Philippines (CFR ≈52%); however, this speculation is based on short genomic reads, so the NiV strain that caused this outbreak is not known (10). NiV<sub>B</sub> has caused repeated outbreaks in Bangladesh and northeastern India; outbreaks occurred almost every year during 2001–2015 (11–15). These NiV<sub>B</sub> outbreaks had higher CFRs, averaging ≈80% (14), and showed documented human-to-human transmission (11,16).

Eight experimental preventive candidate vaccines against henipaviruses have been evaluated in NiV<sub>M</sub> animal models: 1) canarypox and 2) vaccinia viruses encoding the NiV<sub>M</sub> fusion protein (F) or the NiV<sub>M</sub> attachment protein (G) that have shown protection against NiV<sub>M</sub> in hamsters and pigs (17,18); 3) a recombinant adeno-associated virus expressing the NiV<sub>M</sub> G protein that completely protected hamsters against homologous NiV<sub>M</sub> challenge (19); 4) recombinant vesicular stomatitis viruses (rVSVs) expressing the NiV<sub>M</sub> F protein or the NiV<sub>M</sub> G protein that had 100% efficacy in hamsters against NiV<sub>M</sub> (20); 5) rVSVs expressing the NiV<sub>B</sub> F protein or the NiV<sub>B</sub> G protein that completely protected ferrets from NiV<sub>M</sub> disease (21); 6) an rVSV expressing the Zaire ebolavirus (EBOV) glycoprotein (GP) and the NiV<sub>M</sub> G protein (rVSV-EBOV-GP-NiVG) that demonstrated efficacy in NiV<sub>M</sub> hamster (22) and African green monkey (Chlorocebus aethiops) (23) models; 7) a recombinant measles virus virus vector expressing the NiV<sub>M</sub> G

Author affiliations: Galveston National Laboratory, Galveston, Texas, USA; University of Texas Medical Branch, Galveston

DOI: https://doi.org/10.3201/eid2506.181620

Current affiliation: Mayo Clinic, Rochester, Minnesota, USA.
protein that had efficacy in the NiV, African green monkey model (24); and 8) a recombinant subunit vaccine based on the HeV G protein (sG\text{HeV}) that completely protected small animals against lethal HeV and NiV\textsubscript{M} infections (25–27) and was efficacious in the robust African green monkey model of HeV (28) and NiV\textsubscript{M} infection (29). Of 8 vaccines, the sG\text{HeV} vaccine is furthest along in evaluation; it has received licensure as a veterinary vaccine for HeV in horses (Equivac HeV, Zoetis, https://www.zoetis.com) in Australia and is being considered as a human vaccine against NiV. When tested against NiV, these 8 vaccine vectors have been tested only against NiV\textsubscript{M} infection in animal models, and although the antigenicity of these vaccines should not be a concern given that HeV G is an immunogen against NiV\textsubscript{M} infection, there are new data on the NiV\textsubscript{B} African green monkey model to consider as far as dose/regimen of vaccines.

NiV\textsubscript{B} infection in African green monkeys is more pathogenic than NiV\textsubscript{M} infection (30). This difference resulted in significantly reduced efficacy of antibody therapy because of temporal differences in viral load. Specifically, the human monoclonal antibody m102.4 that had been shown to completely protect African green monkeys against lethal NiV\textsubscript{M} disease when treatment was delayed until day 5 after virus exposure provided no protection when African green monkeys were challenged with NiV\textsubscript{B} and treated beginning at day 5 after virus challenge (30,31). Considering these new data, the current vaccines against NiV need to be evaluated for possible differences in dose/regimen against the more pathogenic NiV\textsubscript{B} infection in the robust African green monkey model. To assess single-dose vaccine efficacy, we evaluated the rVSV vaccine vectors expressing either the NiV\textsubscript{B} F or NiV\textsubscript{B} G proteins 28 days after a single-dose vaccination in the NiV\textsubscript{B} African green monkey model, which most faithfully recapitulates human disease (5,30).

Animal Ethics Considerations and Experiments
Healthy adult African green monkeys were handled in the animal BSL-4 containment space at the Galveston National Laboratory (Galveston, TX, USA). Research was approved under animal protocol 1310040 by the University of Texas Medical Branch Institutional Animal Care and Use Committee (Appendix, https://wwwnc.cdc.gov/EID/article/25/6/16-1620-App1.pdf).

We used 10 adult African green monkeys weighing 3.5–6.0 kg in this study. One animal served as control (received G\textsubscript{B} rVSV-ΔG-GFP, and 3 animals per vaccine group received G\textsubscript{B} rVSV-ΔG-NiV\textsubscript{B}/F-GFP, G\textsubscript{B} rVSV-ΔG-NiV\textsubscript{B}/G-GFP, or rVSVΔG-NiV\textsubscript{B}/F/G. For vaccination, animals were anesthetized with ketamine and vaccinated with ≈10\textsuperscript{7} PFU by intramuscular injection (day –28). Twenty-eight days after vaccination, the animals were exposed to ≈5 × 10\textsuperscript{4} PFU of NiV\textsubscript{B}; the dose was equally divided between the intratracheal and the intranasal routes for each animal. Animals were monitored for clinical signs of illness (i.e., temperature, respiration quality, blood count, and clinical pathologic findings) at 0, 3, 6, 8, 10, 15, 21, and 28 days postchallenge (dpc).

NiV\textsubscript{B} Serum Neutralization Assays
We determined neutralization titers against NiV\textsubscript{B} using a conventional serum neutralization assay. In brief, we serially diluted serum 5-fold or 2-fold depending on magnitude of neutralization titers and incubated with ≈100 PFU of NiV\textsubscript{B} for 1 h at 37°C, as previously described (30).

RNA Isolation from NiV\textsubscript{B}-Infected African Green Monkeys
We isolated RNA from NiV\textsubscript{B}-infected animals as described previously (30). For viremia, we added 100 μL of blood to 600 μL of AVL viral lysis buffer (QIAGEN, https://www.qiagen.com) for RNA extraction. For virus load in tissue, we stored ≈100 mg in 1 mL RNAlater (QIAGEN) for 7 d to stabilize RNA, removed the RNA later completely, and homogenized tissues in 600 μL RLT buffer (QIAGEN) in a 2-mL cryovial using a tissue lyser (QIAGEN) and ceramic beads.

Detection of NiV\textsubscript{B} Load
We isolated RNA from blood or tissues and assessed it using primers and probe targeting the N gene and the intergenic region between N and P genes of NiV\textsubscript{B} for quantitative reverse transcription PCR (qRT-PCR). The probe used was 6FAM-5′CGT CAC ACA TCA GCT CTG ACA A 3′-6TAMRA (Life Technologies, https://www.thermofisher.com), as described previously (30).

Hematology and Serum Biochemistry
We assessed clinical pathology of NiV\textsubscript{B}-infected African green monkeys by hematology and serum biochemistry.
analysis as described previously (30). We performed the hematology assays using a laser-based hematologic analyzer (Beckman Coulter, https://www.beckmancoulter.com) and serum biochemistry analysis using a Piccolo point-of-care analyzer and Biochemistry Panel Plus analyzer discs (Abaxis, https://www.abaxis.com).

**Histopathology and Immunohistochemistry**

We performed necropsies on all animals and collected tissue samples of all major organs. We performed histopathologic and immunohistochemical examination and analyses as described previously (30).

**Results**

**Immunization of African Green Monkeys and Measuring the Humoral Immune Response**

Previously, single-injection, single-round replication rVSV vaccine vectors expressing the NiV F or NiV G proteins were described, characterized, and shown to be efficacious against NiV challenge in ferrets (21). To assess the efficacy of these vectors in the NiV African green monkey model, 4 groups of African green monkeys received a single intramuscular vaccination of rVSV vectors on day –28 (Figure 2). To analyze the antibody response to rVSV-ΔG-NiV vaccinations, we assessed circulating antibodies for neutralization activity against NiV before and after vaccination by using a 50% plaque-reduction neutralization titer (PRNT<sub>50</sub>) assay. All 4 groups had no detectable neutralizing antibody titers before vaccination (Table 1, day –28). On the day of challenge, the control animal (C-1) did not have detectable neutralizing antibody titers against NiV, whereas all animals from the specific NiV protein vaccination groups (F, G, and F/G) had detectable neutralizing antibodies against NiV (Table 1, day 0). Overall, the detectable neutralizing antibody response against NiV reached a 1:640 dilution titer in the G and F/G groups and from 1:160 to 1:640 in the F group.

**NiV Challenge and Viral Load of Vaccinated African Green Monkeys**

To determine the efficacy of the rVSV-ΔG-NiV vectors against NiV disease in African green monkeys, we challenged these animals by combined intratracheal and intranasal routes with a lethal challenge dose of NiV on day 0 (Figure 1). All African green monkeys were closely monitored for up to 28 dpc for clinical signs of illness. The NiV antigen vaccinated animals in the F (F-1–3), G (G-1–3), and F/G (F/G-1–3) groups showed no signs of clinical illness (Table 2) and were 100% protected against NiV challenge (Figure 1, panel A), whereas the animal in the nonspecific vaccinated control group (C-1) exhibited clinical signs of disease (Table 2). In addition, the control animal was the only NiV-infected animal to have lymphopenia and serosanguinous nasal discharge during the course of disease (Table 2).

To determine the level of NiV replication in animals after challenge, we assessed viral load by qRT-PCR on...
from 6 dpc (Figure 1, panel D). The lack of systemic and circulating detection of NiVβ RNA correlated with survival (Table 2; Figure 1, panel A).

### Gross Pathologic, Histopathologic, and Immunohistochemical Analyses of NiVβ-Infected African Green Monkeys

In the F, G, and F/G groups, we observed no gross pathologic findings at study endpoint. However, in the control animal that died of NiVβ infection, gross pathologic findings included serosanguinous pleural effusion, failure of all lung lobes to collapse with severe pulmonary hemorrhage and congestion, and multifocal to coalescing hemorrhage of the mucosal surface of the urinary bladder.

Lung sections examined from the control animal had moderate lymphoplasmacytic interstitial pneumonia characterized by a diffuse thickening of alveolar septae by moderate numbers of lymphocytes, plasma cells, polymerized fibrin, and edema fluid. The alveolar spaces were flooded by edema fluid, polymerized fibrin, foamy alveolar macrophages, and cellular debris. Endothelial syncytial cells were most apparent in medium- to small-caliber vessels (Figure 3, panel A). The animals in the F, G, and F/G groups had no major histologic findings in the lung sections (Figure 3, panels C, E, G). Immunohistochemical analysis revealed strong NiV antigen immunoreactivity within scattered alveolar macrophages and the endothelium of the alveolar septae and syncytial cells within medium- to small-caliber vessels in up to 75% of the examined pulmonary tissues (Figure 3, panel B). The lung sections of the F, G, and F/G groups were devoid of detectable NiV antigen (Figure 3, panels D, F, H).

Spleen sections from the control animal were depleted of lymphocytes in the multifocal follicular germinal centers within the splenic white pulp and were effaced by hemorrhage, fibrin, syncytiotial cell formation (Figure 4, panel A). Spleens from the F, G, and F/G groups were devoid of detectable NiV antigen (Figure 4, panels C, E, G). Immunohistochemical analysis of the spleen from the

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**Table 1. NiVβ serum neutralization titers in vaccinated African green monkeys (Chlorocebus aethiops)**

<table>
<thead>
<tr>
<th>Vaccine</th>
<th>Animal no.</th>
<th>Day –28†</th>
<th>Day 0†</th>
<th>Day 28†</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>C-1</td>
<td>&lt;20</td>
<td>&lt;20</td>
<td>40‡</td>
</tr>
<tr>
<td>F only vaccine</td>
<td>F-1</td>
<td>&lt;20</td>
<td>640</td>
<td>1,280</td>
</tr>
<tr>
<td></td>
<td>F-2</td>
<td>&lt;20</td>
<td>160</td>
<td>2,560</td>
</tr>
<tr>
<td></td>
<td>F-3</td>
<td>&lt;20</td>
<td>320</td>
<td>5,120</td>
</tr>
<tr>
<td>G only vaccine</td>
<td>G-1</td>
<td>&lt;20</td>
<td>640</td>
<td>5,120</td>
</tr>
<tr>
<td></td>
<td>G-2</td>
<td>&lt;20</td>
<td>640</td>
<td>5,120</td>
</tr>
<tr>
<td></td>
<td>G-3</td>
<td>&lt;20</td>
<td>640</td>
<td>5,120</td>
</tr>
<tr>
<td>F+G vaccine</td>
<td>F/G-1</td>
<td>&lt;20</td>
<td>640</td>
<td>2,560</td>
</tr>
<tr>
<td></td>
<td>F/G-2</td>
<td>&lt;20</td>
<td>640</td>
<td>5,120</td>
</tr>
<tr>
<td></td>
<td>F/G-3</td>
<td>&lt;20</td>
<td>640</td>
<td>2,560</td>
</tr>
</tbody>
</table>

†Titers are reciprocal serum dilution at which 50% of virus was neutralized.

### Table 2. Clinical findings and outcome of NiVβ-infected African green monkeys

<table>
<thead>
<tr>
<th>Animal no.</th>
<th>Sex</th>
<th>Group</th>
<th>Clinical illness</th>
<th>Clinical and gross pathology findings†</th>
</tr>
</thead>
<tbody>
<tr>
<td>C-1</td>
<td>F</td>
<td>Control ΔG vaccine</td>
<td>Loss of appetite (d 6–8); labored breathing (d 6–8). Died on d 8.</td>
<td>Lymphopenia (d 6); serosanguinous nasal and oral discharge (d 8), serosanguinous pleural fluid, severely inflated, enlarged lungs with severe congestion and hemorrhage of all lobes, multifocal to coalescing hemorrhage of the mucosal surface of the urinary bladder.</td>
</tr>
<tr>
<td>F-1</td>
<td>F</td>
<td>F vaccine</td>
<td>None</td>
<td>Thrombocytopenia (d 15); &gt;3-fold increase in ALT (d 6), &gt;3-fold increase in AST</td>
</tr>
<tr>
<td>F-2</td>
<td>M</td>
<td>F vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>F-3</td>
<td>M</td>
<td>F vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>G-1</td>
<td>F</td>
<td>G vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>G-2</td>
<td>M</td>
<td>G vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>G-3</td>
<td>M</td>
<td>G vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>F/G-1</td>
<td>F</td>
<td>F + G vaccine</td>
<td>None</td>
<td>Increase in CRP (d 8)</td>
</tr>
<tr>
<td>F/G-2</td>
<td>M</td>
<td>F + G vaccine</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>F/G-3</td>
<td>M</td>
<td>F + G vaccine</td>
<td>None</td>
<td>Thrombocytopenia (d 21, d 28); increase in CRP (d 8, d 10, d 15)</td>
</tr>
</tbody>
</table>

†ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein.

†Lymphopenia is defined as a ≥30% decrease in number of lymphocytes; thrombocytopenia is defined as a ≥30% decrease in number of platelets.
control animal revealed strong immunoreactivity for NiV antigen within the endothelium, syncytial cells, and scattered mononuclear cells in up to ≈50% of the examined splenic tissue (Figure 4, panel B), whereas the spleen sections of groups F, G, and F/G were devoid of detectable NiV antigen (Figure 4, panels D, F, H).

**Discussion**

An important step in the preclinical development of a vaccine is efficacy testing in standards of animal models of disease. For NiV, the standard is the African green monkey model. Although the initial studies on the NiV model in African green monkeys were reported as near uniformly lethal, data from several groups have revealed the model is not 100% lethal, depending on dose and route of infection (5,24,29–31,33,34). Combining the control animals from these studies, in which African green monkeys were challenged with various combinations of routes (e.g., intratracheal, intranasal, intraperitoneal, oral, small particle aerosol) at various doses, revealed that 18 (72%) of 25 animals died; however, most of the control animals were positive for circulating NiV RNA and had signs of clinical disease to varying degrees. Historically, our previous studies with the NiV model has resulted in the deaths of all 14 control African green monkeys; the mean time to death was 7.14 days (Figure 1, panel A). We recently compared the pathogenesis of NiV and NiV strains in African green monkeys and observed that NiV caused more pulmonary and splenic pathologic findings (30). We also observed the efficacy of time to treatment post-NiV challenge with a human monoclonal antibody m102.4 was shorter for NiV-infected animals than for NiV-infected animals (30). With these animal data in mind and the fact that NiV has been responsible for most NiV outbreaks since 2002, we wanted to test our rVSV NiV vaccine vectors expressing NiV F and G proteins as immunogens, which had 100% efficacy against NiV challenge in ferrets (21), against NiV challenge in African green monkeys.

In this study, we vaccinated 1 control African green monkey with a nonglycoprotein rVSV vector control, G/Ind* rVSV-ΔG-GFP, and 3 groups of 3 African green monkeys with NiV antigen vectors: G/Ind* rVSV-ΔG-NiV/F-GFP, G/Ind* rVSV-ΔG-NiV/F-GFP, or G/Ind* rVSV-ΔG-NiV/F-GFP. The control animal, C-1, did not develop NiV neutralizing antibodies by the day of challenge; had detectable circulating NiV RNA at 6 dpc; had clinical signs of NiV-mediated disease; and ultimately died of infection, showing typical NiV gross pathology and histopathologic findings. Conversely, the 3 rVSV NiV vaccine groups had animals in which detectable circulating NiV F, G, or F and G IgG developed, and circulating neutralizing antibody titers developed in all 3 groups by 28 days postvaccination. Each vaccine cohort had detectable NiV RNA in nasal swab samples and only the F and F/G groups in oral swab samples, but none of the cohorts had any detectable circulating NiV RNA throughout the course of the study. Consistent with the vaccine response from each cohort and the control of systemic spread of NiV infection and control of NiV-mediated disease, all of the specifically vaccinated African green monkeys survived NiV challenge.

The results of this study are similar to what we observed with these rVSV NiV constructs in the ferret model, which showed 100% protection regardless of the vaccine construct (21). Differences were that we found higher PRNT results for neutralizing antibody titers on day of challenge in this study and detected no circulating NiV RNA in the African green monkeys but did have detectable viral RNA at 6 dpc in the ferret study. Although we
did not detect circulating viral RNA in the African green monkeys, the increase of neutralizing antibody titers at the study endpoint suggests sterilizing immunity was not achieved, and dosing or regimen will require further testing to reach sterilizing immunity with this single-round replication vaccine vector.

The single-round replication rVSV NiV vectors in this study and the replication-competent rVSV-EBOV-GP-NiVG (23) are the only vaccine vectors to show 100% single-dose vaccine efficacy against NiV in the African green monkey model. Although both studies used this model, they differed in several ways. Our study used NiV\textsubscript{B} and challenged through the intratracheal and intranasal routes, whereas the other study used NiV\textsubscript{M} by the intratracheal route only (intratracheal challenge route used in initial model [5]). Here, we report detectable levels of NiV RNA in nasal swab samples at early times postchallenge, whereas the rVSV-EBOV-GP-NiVG study did not report any detectable NiV RNA in nasal swab samples. Whether these differences resulted from use of the intranasal route as part of the challenge cannot be determined here; however, neither study reported circulating levels of NiV RNA in nasal swab samples at early times postchallenge, whereas the rVSV-EBOV-GP-NiVG study did not report any detectable NiV RNA in nasal swab samples. Whether these differences resulted from use of the intranasal route as part of the challenge cannot be determined here; however, neither study reported circulating levels of NiV RNA in nasal swab samples at early times postchallenge, whereas the rVSV-EBOV-GP-NiVG study did not report any detectable NiV RNA in nasal swab samples. Whether these differences resulted from use of the intranasal route as part of the challenge cannot be determined here; however, neither study reported circulating levels of NiV RNA in nasal swab samples at early times postchallenge, whereas the rVSV-EBOV-GP-NiVG study did not report any detectable NiV RNA in nasal swab samples.

The PRNT\textsubscript{50} titers we reported can be directly compared with the recombinant subunit sG\textsubscript{HeV} vaccine NiV study in African green monkeys that also was 100% efficacious (29), whereas we detected higher PRNT\textsubscript{50} titers against NiV from the single injection of single-round replication vectors (from 160 to 640; Table 1) versus the PRNT\textsubscript{50} titers 2 weeks after boost vaccination (from 28 to 379) for the recombinant subunit sG\textsubscript{HeV} vaccine. However, these lower titers most likely are due to the sG\textsubscript{HeV} vaccine being heterotypic because the PRNT\textsubscript{50} titers against HeV in a similar African green monkey study were 640–1,280 on day of challenge (28). The development of neutralizing antibodies to the NiV glycoproteins after vaccination are important for protection, as highlighted by a single monoclonal antibody against the henipavirus G protein, m102.4, that is 100% protective against HeV, NiV\textsubscript{M}, and NiV\textsubscript{B} when administered at least 3 dpc (30,31,35).

In our study, the F cohort did not produce as consistent a neutralizing antibody titer response as did the G and F/G cohorts. Further analysis also revealed that, although no major changes occurred in hematologic and blood chemistry results for any of the vaccine cohorts, minor changes occurred in the F and F/G cohorts (Table 1). These data, taken together with the lack of detectable NiV\textsubscript{B} RNA in the oral swab samples of the G group, suggest the rVSV NiV G vector might be the better option among the 3 vaccine vectors.
In summary, we found that single-round replication rVSV vectors against NiV \( B \) provided 100% efficacy against NiV \( B \) challenge using a single-dose regimen. The rVSV vaccine platform has received attention recently because the replication-competent rVSV-ZEBOV GP vaccine vector against EBOV has now been given to >16,000 humans in clinical trials ranging from phase 1 to phase 3 and has been safe and efficacious (36); however, data for pregnant women and immunocompromised persons are not yet available. A single-round replication rVSV vaccine vector that is immunogenic and efficacious would have an attractive safety profile. Whether these single-round replication rVSV NiV vaccine vectors are as safe as the recombinant subunit sG \( H e V \) vaccine has yet to be determined, and the subunit vaccine has yet to be tested with a single-dose vaccine regimen. Although multidose vaccine regimens would be a potential strategy for laboratory and healthcare workers and for first responders in stable settings with defined risk for an NiV outbreak, an outbreak setting or a case of deliberate release of NiV would require rapid protection with a single administration of vaccine. The single-dose strategy was successfully enacted using a close-contact ring vaccination strategy with the rVSV-ZEBOV-GP vaccine at the end of the 2013–2016 EBOV epidemic (37–39). The strategy was so successful that it became the World Health Organization recommendation for future EBOV outbreaks and has recently been set into motion in the ongoing outbreak in the Democratic Republic of the Congo (40). Recent studies also suggest that the ring vaccination strategy for viruses such as EBOV (depending on transmissibility) that are endemic to countries that might not be able to afford a mass herd-immunity vaccination strategy might be more effective than mass vaccinations at controlling outbreaks (41).

Further studies should examine the time to immunity of the \( G_{\text{ind}}^* \text{rVSV-}\Delta G-\text{NiV}_B/G \) in the NiV \( B \) African green monkey model because these data will be instrumental in providing information about whether this vaccine vector could be implemented in a ring vaccination strategy during future NiV outbreaks, such as the current one in India (2).

**Acknowledgments**

We thank the staff of the University of Texas Medical Branch Animal Resources Center for animal husbandry, Robert W. Cross for assistance with the animal study, and Natalie Dobias for assistance with histologic processing. We thank Thomas G. Ksiazek for kindly providing the NiV \( B \) isolate used in this study.

This study was supported in part by National Institutes of Health (U01 AI082121 for research and UC7AI094660 for BSL-4 operations) and by funds provided to T.W.G. by the UTMB Department of Microbiology and Immunology.

**About the Author**

Dr. Mire is an associate professor in the Department of Microbiology and Immunology at the University of Texas Medical Branch–Galveston and the Galveston National Laboratory. His research focuses on understanding host–pathogen interactions of highly pathogenic RNA viruses.
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Address for correspondence: Thomas W. Geisbert, University of Texas Medical Branch, Microbiology and Immunology, 301 University Blvd, Galveston, TX 77550-0610, USA; email: twgeisbe@utmb.edu