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Inside the CDC's Airport Ebola Screening Surge: How the 2026 DRC Bundibugyo Epidemic Tested Global Border Biosecurity

Public Health * Epidemiology * Ebola

As the 2026 Democratic Republic of the Congo (DRC) epidemic of Bundibugyo virus disease (BVD) passes 10,060 confirmed cases and 4,890 deaths across eight Congolese provinces, the U.S. Centers for Disease Control and Prevention (CDC) has issued an urgent call for agency staff to volunteer at major international port-of-entry quarantine stations [1, 2] . Paired with a sweeping federal mandate taking effect November 3, 2026 -- which requires every air passenger boarding a U.S.-bound international flight to sign a legal attestation confirming they have not been in the DRC within the preceding 21 days -- the mobilization highlights a recurring epidemiological tension in global health security: how public health agencies balance the mathematical realities of viral incubation periods against the speed of commercial aviation [1, 3, 4] .

While border temperature checks and travel attestations serve as visible public health barriers, decades of quantitative modeling demonstrate that syndromic airport screening alone catches only a fraction of incubating travelers when a pathogen's latency period spans up to three weeks [5, 6] . Understanding why the CDC is coupling port-of-entry volunteer deployments with a strict 21-day pre-departure exclusion window -- and how these border measures buy time for the newly launched Phase 2/3 Tumaini vaccine trial in Bunia, DRC -- requires examining the virology of Orthoebolavirus bundibugyoense , the statistics of incubation-period right tails, and the operational architecture of post-arrival contact monitoring [2, 3, 7] .

1. Outbreak Telemetry: The 2026 Bundibugyo Epidemic in Context

First identified during a 2007 outbreak in western Uganda's Bundibugyo District, the Bundibugyo virus (BDBV; taxonomically classified as Orthoebolavirus bundibugyoense within the family Filoviridae) is one of four ebolavirus species known to cause severe hemorrhagic and gastrointestinal disease in humans, alongside Zaire ebolavirus (EBOV), Sudan virus (SUDV), and Ta Forest virus (TAFV) [2, 8] . Historically, BDBV outbreaks were smaller and less frequent than Zaire ebolavirus epidemics, exhibiting case fatality ratios (CFR) between 25% and 51% [8] . However, the epidemic declared by the DRC Ministry of Health on May 15, 2026, fundamentally altered that trajectory [2] .

Fueled by high population mobility across the eastern DRC's mining and commercial corridors -- spanning Ituri, North Kivu, South Kivu, and Tshopo provinces -- the 2026 epidemic has grown into the largest Bundibugyo virus outbreak ever recorded and the second-largest filovirus epidemic in history, surpassed only by the 2013-2016 West African Zaire ebolavirus crisis [2, 4] . Although Uganda successfully contained its initial cross-border cluster of 21 cases in June 2026, October 2026 brought renewed regional spillover, including an imported index case and a nine-person

transmission cluster in Kenya as well as an imported case detected in Abuja, Nigeria, involving air travel through regional hubs [2, 4] .

| Epidemiological & Operational Parameter | | 2026 DRC Bundibugyo Outbreak Metrics | | Public Health & Screening Relevance |

| Causative Pathogen | | Orthoebolavirus bundibugyoense (BDBV), negative-sense ssRNA filovirus [2, 8] | | ~35-40% glycoprotein sequence divergence from Zaire ebolavirus limits cross-neutralization by legacy monoclonal antibodies [8, 9] |

| Cumulative Burden (Late Oct 2026) | | >10,060 confirmed cases; ~4,890 deaths (Crude CFR ~ 48.6%) across 8 DRC provinces [2, 4] | | Second-largest Ebola epidemic on record; declared a WHO Public Health Emergency of International Concern (PHEIC) [4] |

| Regional Spillover Events | | Uganda (21 cases, contained June 2026); Kenya (9 cases, 2 deaths, Oct 2026); Nigeria (1 imported case, Oct 2026) [2, 4] | | Demonstrates long-distance air and ground transit connectivity out of the Great Lakes region [2, 4] |

| Incubation Period Distribution | | Mean: 6.3-9.4 days; Observed range: 2-21 days (95th percentile <= 18-21 days) [6, 8] | | Dictates the mandatory 21-day pre-boarding exclusion window and 21-day active post-arrival fever monitoring protocol [3, 6] |

| U.S. Port-of-Entry Intervention | | CDC staff volunteer surge at U.S. gateway airports + universal 21-day passenger attestation (effective Nov 3, 2026) [1, 3] | | Funnels returning U.S. citizens and exempt personnel through designated Port Health Stations (e.g., IAD, ATL, JFK, ORD, IAH) [1, 3] |

| Countermeasure Clinical Trial | | Phase 2/3 Tumaini ("Hope") ring-vaccination trial launched Oct 23, 2026 in Bunia, DRC [7] | | Evaluates cross-protective and BDBV-targeted vaccine candidates to interrupt transmission chains at the source [7] |

2. Inside the CDC Airport Screening Surge and the November 3 Attestation Rule

The CDC's late-October 2026 call for internal volunteers -- soliciting public health advisors, epidemiologists, medical officers, and emergency management specialists from across agency centers -- addresses an acute operational bottleneck at U.S. Port Health Stations [1] . Under layered federal travel orders issued by the Department of Homeland Security (DHS) and the Department of Health and Human Services (HHS), non-U.S. citizens and non-lawful permanent residents who have been physically present in the DRC within the preceding 21 days are temporarily barred from entering the United States [1, 3] . However, U.S. citizens, lawful permanent residents, diplomatic personnel, and credentialed humanitarian responders returning from outbreak zones retain the legal right to return home [3] .

Rather than arriving at dozens of domestic airports, these returning travelers are routed through a small network of designated U.S. international gateway airports -- including Washington-Dulles (IAD), Hartsfield-Jackson Atlanta (ATL), John F. Kennedy International (JFK), Chicago O'Hare (ORD), and Houston George Bush Intercontinental

(IAH) [1, 3] . At these hubs, CDC Port Health personnel and volunteer surge teams work alongside U.S. Customs and Border Protection (CBP) officers to conduct structured entry assessments:

* Exposure Risk Stratification: Responders administer standardized epidemiological interviews assessing whether the traveler provided direct healthcare to BVD patients, handled clinical laboratory specimens, attended traditional burials, or experienced a breach in personal protective equipment (PPE) [3, 10] .

* Non-Contact Infrared Thermometry & Symptom Evaluation: Travelers undergo tympanic or infrared forehead temperature screening (with fever defined clinically as >=38.0 deg C / 100.4 deg F) alongside visual inspection for prodromal symptoms including severe headache, myalgia, asthenia, pharyngitis, or gastrointestinal distress [3, 10] .

* Jurisdictional Handoff to State and Local Health Departments: Every asymptomatic traveler with recent DRC travel history is registered into the CDC's electronic traveller data transfer network, triggering a mandatory 21-day active or direct active monitoring protocol administered by the state or county health department at their final U.S. destination [3, 10] .

To prevent travelers from bypassing designated routing via third-country connections (for example, flying from Kinshasa or Goma to Addis Ababa, Nairobi, or European hubs before booking a separate ticket to the United States), the CDC issued a sweeping global order effective November 3, 2026 [3] . Under this order, every passenger boarding any international flight bound for the United States -- regardless of nationality or departure continent -- must complete a legally binding digital or paper attestation declaring they have not been in the DRC during the prior 21 days, or else hold an official federal exemption routing them to enhanced port screening [3] . Airlines must verify and retain these attestations prior to boarding and deny boarding to non-compliant travelers [3] .

3. The Epidemiology of Border Screening: Why the 21-Day Incubation Window Matters

To appreciate both the utility and the inherent limitations of airport fever screening, epidemiologists model viral importation as a competing-risks process governed by the pathogen's serial interval , incubation period distribution , and infectiousness profile [5, 6] . In filovirus infections, the incubation period -- the time elapsed between initial viral exposure and the onset of clinical symptoms -- follows a right-skewed log-normal or gamma distribution [6] . Across historical Zaire, Sudan, and Bundibugyo ebolavirus outbreaks, the mean incubation period is approximately 6.3 to 9.4 days, with a median near 7 to 8 days [6, 8] . However, the upper tail (the 95th to 99th percentile) extends to 21 days [6] .

Crucially, filovirus transmission dynamics differ fundamentally from respiratory pathogens such as SARS-CoV-2 or influenza A [5, 8] :

* Minimal Pre-Symptomatic Shedding: In Ebola virus disease, peripheral blood viral load (viremia) remains below the threshold required for person-to-person transmission during the asymptomatic incubation phase [8, 10] . Infectious virions appear in blood, vomit, diarrhea, sweat, and mucosal secretions only after systemic

viral replication triggers clinical symptom onset (fever, malaise, and endothelial dysfunction) [8, 10] .

* Exponential Rise in Infectiousness During the Gastrointestinal Phase: During the early febrile prodrome (days 1-3 of illness), viral titers in saliva and skin are relatively low; infectiousness peaks sharply during days 4-10 when severe secretory diarrhea and emesis contain up to 10 [7] to 10 [8] RNA copies per milliliter [8, 10] .

* The Time-of-Flight Blind Spot: Because commercial flights from Central Africa to North America (including layovers) take 16 to 30 hours -- whereas the mean incubation period is roughly 168 to 225 hours -- quantitative stochastic models by Gostic et al. and Read et al. show that 80% to 92% of infected travelers who board a plane while asymptomatic will still be asymptomatic when they land [5, 6] .

Consequently, thermal cameras and forehead thermometers at airport arrival gates can only detect the small minority of infected travelers who either boarded while already febrile (and failed exit screening) or happened to cross the symptom-onset threshold mid-flight [5] . This mathematical reality explains why public health authorities never rely on airport thermometry as a standalone firewall. Instead, the primary epidemiological value of funneling travelers through CDC Port Health Stations is enrollment into 21-day post-arrival monitoring -- ensuring that if an incubating traveler develops a 38.2 deg C fever on day 11 at home, they are immediately transported via bio-containment ambulance to a designated Regional Emerging Special Pathogens Treatment Center (RESPTC) before any secondary household or nosocomial transmission can occur [3, 5, 10] .

4. Molecular Virology of Orthoebolavirus bundibugyoense : Why Existing Vaccines Were Bypassed

Why did the 2026 Bundibugyo outbreak expand to more than 10,000 cases in an era when the global health community possesses stockpiles of licensed Ebola vaccines and monoclonal antibody cocktails? The answer lies in the structural immunogenetics of the filovirus envelope glycoprotein (GP) [8, 9] .

All ebolaviruses encode a ~19-kilobase negative-sense, single-stranded RNA genome comprising seven structural genes: nucleoprotein (NP), viral protein 35 (VP35), matrix protein (VP40), glycoprotein (GP), replication transcription protein (VP30), membrane-associated protein (VP24), and the RNA-dependent RNA polymerase (L) [8] . The trimeric surface glycoprotein (GP 1,2) is the sole viral protein exposed on the lipid envelope and mediates host cell attachment, macropinocytosis, and endosomal membrane fusion via the Niemann-Pick C1 (NPC1) cholesterol transporter [8, 9] . Because GP 1,2 is the primary target of neutralizing antibodies, both the licensed recombinant vesicular stomatitis virus vaccine (rVSV-DeltaG-ZEBOV-GP , marketed as Ervebo) and the FDA-approved monoclonal antibody therapeutics (Inmazeb and Ebanga) were engineered specifically against the Zaire ebolavirus GP sequence [7, 9] .

| Viral Structural Feature / Countermeasure | | Orthoebolavirus zairense (EBOV) | | Orthoebolavirus bundibugyoense (BDBV) |

| Envelope Glycoprotein (GP 1,2) Identity | | Reference canonical Zaire sequence

(100% identity) [8] | | ~60-65% amino acid identity (~35-40% divergence, concentrated in the glycan cap and mucin-like domain) [8, 9] |

| Endosomal Entry Receptor | | Niemann-Pick C1 (NPC1) following cathepsin B/L proteolysis [8] | | Conserved NPC1 domain C binding pocket, but shielded prior to cleavage by divergent surface epitopes [8, 9] |

| Ervebo (rVSV-DeltaG-ZEBOV-GP) Efficacy | | 97.5%-100% ring-vaccination efficacy against Zaire ebolavirus [7] | | Incomplete heterologous neutralization; under clinical evaluation in the 2026 Tumaini trial [7, 9] |

| Licensed mAbs (Inmazeb / Ebanga) | | Potent neutralization of Zaire GP chalice and glycan cap epitopes [9] | | Substantially reduced binding affinity due to epitope substitutions outside conserved internal fusion loop [9] |

| Innate Immune Antagonism | | VP35 dsRNA sequestration + VP24 karyopherin-alpha1 blockade [8] | | Conserved VP35/VP24 interferon suppression, enabling rapid early viremia before adaptive B-cell maturation [8] |

Although Zaire ebolavirus and Bundibugyo virus share conserved inner core proteins and bind the same intracellular NPC1 receptor after cathepsin B/L protease cleavage inside late endosomes, their outer GP 1,2 sequences diverge by roughly 35% to 40% at the amino acid level [8, 9] . The divergence is most pronounced across the heavily glycosylated mucin-like domain (MLD) and the glycan cap -- the exact exterior surfaces exposed to circulating antibodies before the virus enters a host cell [9] . As a result, neutralizing antibodies elicited by monovalent Zaire vaccines or Zaire-specific therapeutic mAbs fail to efficiently recognize intact BDBV virions in the bloodstream, allowing the virus to replicate unchecked in monocytes, macrophages, and dendritic cells while viral proteins VP35 and VP24 shut down host type-I interferon signaling [8, 9] .

5. Closing the Countermeasure Gap: The Phase 2/3 Tumaini Trial and Global Containment

Ultimately, border screening and travel attestations in importing nations can only delay or intercept individual introductions; extinguishing an epidemic of 10,000+ cases requires interrupting transmission chains at the source in Central Africa [4, 7] . On October 23, 2026 , a coalition led by the DRC's Institut National de Recherche Biom dicale (INRB), the World Health Organization (WHO), and the Coalition for Epidemic Preparedness Innovations (CEPI) vaccinated the first participant in Bunia, Ituri Province, launching the pivotal Phase 2/3 Tumaini ("Hope" in Swahili) clinical trial [7] .

Structured on the adaptive ring-vaccination design pioneered during the 2015 Ebola a Suffit! trial in Guinea, the Tumaini protocol identifies newly laboratory-confirmed BVD index patients and rapidly randomizes their primary contacts -- and contacts of contacts -- to receive candidate vaccines [7] . The trial is evaluating both BDBV-specific viral-vectored constructs (expressing the native Bundibugyo glycoprotein) and heterologous regimens to determine whether high-titer cellular and humoral responses targeting conserved internal fusion loop (IFL) and receptor-binding domain (RBD) epitopes can confer rapid ring protection within 10 days of immunization [7, 9] .

6. Clinical Care, Biocontainment Architecture, and Diagnostic Stratification in Receiving Hubs

When a monitored traveler in the United States or another non-endemic country develops a febrile illness within 21 days of departing the DRC, the clinical and laboratory response follows a tiered biocontainment protocol designed to protect healthcare workers while rapidly ruling out more common tropical infections [3, 10] . In practice, empirical data from prior international filovirus responses show that the vast majority of febrile travelers returning from Equatorial Africa are ultimately diagnosed with Plasmodium falciparum malaria, typhoid fever, arboviral infections (such as dengue or chikungunya), or acute respiratory viruses rather than Ebola disease [5, 10] . However, because early Bundibugyo virus disease presents with non-specific prodromal symptoms -- fever, severe frontal headache, myalgia, and profound fatigue -- clinical differentiation is impossible without molecular testing [8, 10] .

Under CDC and National Emerging Special Pathogens Training and Education Center (NETEC) guidelines, any Person Under Investigation (PUI) is immediately placed in a single-patient airborne infection isolation room (AIIR) or negative-pressure biocontainment unit, with clinical staff donning full fluid-resistant personal protective equipment (PPE), powered air-purifying respirators (PAPRs) or N95 respirators with full face shields, and double nitrile gloves [10] . Whole-blood EDTA specimens are collected under strict point-of-care biosafety protocols and tested simultaneously via multiplex real-time reverse transcription polymerase chain reaction (RT-qPCR) -- such as the BioFire FilmArray Warrior/Global Fever Panel -- alongside rapid malaria antigen testing so life-saving antimalarial therapy is never delayed during rule-out diagnostics [8, 10] .

Importantly, because peripheral blood viral RNA levels can remain below the limit of detection during the first 24 to 48 hours of fever, a negative RT-qPCR result obtained less than 72 hours after symptom onset does not definitively exclude Bundibugyo virus infection [8, 10] . Patients testing negative early in their febrile course remain in isolation and undergo a repeat confirmatory RT-qPCR assay at least 72 hours after symptom onset before biocontainment precautions are lifted [10] . For confirmed cases, supportive critical care -- centered on aggressive intravenous crystalloid resuscitation, continuous electrolyte replacement (correcting profound hypokalemia, hyponatremia, and metabolic acidosis driven by secretory gastrointestinal losses), and renal replacement therapy -- can reduce filovirus mortality by more than half even in the absence of strain-specific monoclonal antibodies [8, 10] .

As the Tumaini trial scales across eastern DRC treatment centers and the CDC's November 3 passenger attestation order takes effect alongside volunteer-staffed airport Port Health Stations, the 2026 Bundibugyo response illustrates the interconnected reality of modern biosecurity [1, 3, 7] . Effective containment depends simultaneously on domestic surveillance -- identifying, isolating, and monitoring travelers across the full 21-day incubation distribution -- and international scientific mobilization to deploy pan-filovirus vaccines and broad-spectrum therapeutics capable of neutralizing divergent ebolavirus species before local spillovers become continental emergencies [4, 7, 9] .

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