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Prey Substitution in the Atlantic Forest: Why Mosquitoes Are Swapping Wildlife for Urban Biomass

Environment * Climate Change * Public Health

Introduction: The Anthropocene and the Generalist Vector

Human restructuring of the biosphere has systematically simplified complex ecosystems, replacing specialized flora and fauna with agricultural monocultures and peri-urban settlements. While stratigraphers define this epoch as the Anthropocene, its biological signature is the ascendancy of opportunistic generalists -- foremost among them hematophagic mosquitoes (Diptera: Culicidae). Nowhere is this clearer than in Brazil's Atlantic Forest (Mata Atl ntica). Once a continuous 1.3-million-square-kilometer coastal biome rivaling the Amazon, centuries of urbanization and clearing have reduced it to a fragmented archipelago with less than 30% of its original cover intact.[1]

A 2026 study published in Frontiers in Ecology and Evolution by Alves, Machado, Alencar, and colleagues demonstrates how this fragmentation dismantles barriers against zoonotic spillover.[1, 6] As biodiversity declines and wild vertebrates vanish from forest remnants, sylvatic mosquitoes redirect their host-seeking apparatus toward the most abundant remaining biomass: Homo sapiens.[2, 3] Integrating field molecular biology, neurobiological olfaction models, and comparative epidemiology across malaria and arboviral zones, this report examines how defaunation drives prey substitution and amplifies zoonotic risk.

Fragmentation Geometry and Molecular Blood-Meal Forensics

Landscape fragmentation alters forest remnants through microclimatic edge effects and vertebrate defaunation. Shrinking patch size elevates perimeter-to-interior ratios, exposing margins to higher temperatures, lower humidity, and wind shear that collapse vertical stratification and draw canopy vectors toward the ground.[4, 5] Simultaneously, hunting and habitat loss eliminate tapirs, agoutis, and howler monkeys -- creating an "empty forest" blood famine.[2]

Alves, Alencar, and colleagues sampled two reserves in Rio de Janeiro state: the 7,400-hectare Guapia u River Ecological Reserve (REGUA), supporting >455 bird and 61 mammal species, and S tio Recanto Preservar (SRP), a smaller secondary fragment surrounded by agriculture.[1, 6] Using CDC light traps at 2 meters during crepuscular periods, the team captured >1,700 mosquitoes and isolated engorged females before digestive enzymes degraded host DNA.[6] Amplifying vertebrate mitochondrial DNA via polymerase chain reaction (PCR) targeting the cytochrome b (Cytb) gene and querying BLAST reconstructed species-level trophic networks.[3, 6]

The results revealed an overwhelming anthropophilic signal: 75% of identified blood meals derived from humans (18 distinct individuals), with birds (six individuals), amphibians, rodents, and canids comprising the remainder.[3, 6] Human blood was detected across sylvatic species including Aedes scapularis, Psorophora ferox, and

Coquillettidia taxa. Moreover, mixed meals -- Coquillettidia fasciolata containing human and bird blood, and Coquillettidia venezuelensis containing human and amphibian blood -- confirmed active cross-species and cross-class bridge biting.[1, 6]

Disease Ecology: Dilution Failure and the Bridge Vector Guild

Under the Dilution Effect Hypothesis, intact vertebrate diversity suppresses disease transmission because vectors distribute bites across "incompetent" sink hosts while predators regulate mosquito densities.[7, 8] In Atlantic Forest fragments, deforestation triggers a trophic cascade that replaces dilution with a Concentration Effect, focusing bites on humans.[2, 9]

| Species | | Primary Niche | | Host Plasticity | | Anthropogenic Risk Factor | | Virological Competence |

| Aedes scapularis | | Ground pools, edge | | High: Mammals & humans | | Invades dwellings; desiccation-resistant | | Yellow Fever, Rocio, VEE[6, 12] |

| Psorophora ferox | | Flooded woods | | Medium-High: Generalist | | Persistent diurnal/crepuscular biter | | Rocio, Ilheus, WNV[11, 13] |

| Cq. venezuelensis | | Pistia roots | | Extreme: Amphibian-human | | Submerged larvae evade surface control | | Oropouche (OROV), EEE[1, 6, 14] |

| Aedes albopictus | | Peri-urban edge | | High: Opportunistic | | Invasive container breeder | | Dengue, Zika, YFV[6, 24] |

| Hg. janthinomys | | Forest canopy | | Conditional: Primatophile | | Descends when canopy fragments | | Sylvatic Yellow Fever[5] |

Key guild members act as direct viral bridges: desiccation-resistant Aedes scapularis commutes into homes and carried natural Yellow Fever virus (YFV) infections during Brazil's 2016-2018 outbreaks;[10, 12] Psorophora ferox transmits neurotropic Rocio virus;[13] and root-piercing Coquillettidia venezuelensis bridges amphibians and humans.[1, 14]

| Mechanism | | Dilution Effect (Intact Forest) | | Fragmentation Reality | | Consequence |

| Host Diversity | | High (primates, birds, marsupials) | | Defaunated; humans dominate | | Loss of sink hosts concentrates bites.[2, 8] |

| Stratification | | Canopy vectors isolated | | Edge collapses vertical gradient | | Arboreal vectors contact humans.[5] |

| Olfactory Learning | | Diverse vertebrate odors | | Dominant human lactic/carboxylic plume | | Dopaminergic anthropophily.[16, 18] |

Olfactory Neurobiology: Dopaminergic Learning and Host Switching

Mosquito host localization integrates long-range (>10 m) CO2 plume anemotaxis, mid-range visual contrast, and short-range (<1 m) detection of human L-(+)-lactic acid and carboxylic acids via the ionotropic co-receptor Ir8a.[15, 17] Crucially, associative learning mediated by the neurotransmitter dopamine in the antennal lobe pairs blood-meal rewards with concurrent host odors.[16] In defaunated remnants,

females orienting toward abundant human settlements gain immediate blood rewards while zoophilic foragers starve. Dopaminergic conditioning within a single generation, reinforced across generations by natural selection on olfactory receptors, domesticates sylvatic vectors alongside invasive Aedes albopictus.[15, 18, 24]

Arboviral Amplification and Global Defaunation Parallels

In the vectorial capacity equation, C = (m * a2 * pn) / ln(p), the human-biting rate a is squared because transmission requires sequential bites.[19] Shifting 75% of blood meals onto humans therefore drives an exponential surge in vectorial capacity for circulating arboviruses including Yellow Fever, Oropouche (OROV), and Rocio virus.[6, 14, 19] Identical edge dynamics drive zoonotic spillover globally: Malaysian Borneo oil-palm fragmentation forces macaques and Anopheles balabacensis into shared edges to spread Plasmodium knowlesi;[20] Western Ghats clearing in India concentrates Haemaphysalis ticks and rodents to amplify Kyasanur Forest Disease;[21, 22] and Amazon pasture conversion pushes canopy Haemagogus into settlements.[23]

Conclusion: Re-engineering Surveillance at the Forest Interface

The predominance of human blood meals inside Atlantic Forest reserves proves that biodiversity loss actively selects for anthropophilic vectors. Preventing future arboviral epidemics requires expanding vector control to include crepuscular forest-edge trapping, routine PCR blood-meal barcoding, and multi-trophic ecological restoration that rebuilds vertebrate dilution buffers.[6, 25]

Updates... (Nov 22, 2026): Corrected publication year, volume, and page range on Tchouassi et al. (2021, Transboundary and Emerging Diseases, 68(4), 2503-2513), corrected author initials and journal attribution on Wu et al. (2025, Frontiers in Cellular and Infection Microbiology, PMC12623374), and added exact publication dates across all cited sources. (Earlier update, Nov 18, 2026: Corrected author initials and volume metadata on Alves et al., 2026 and Kaushal et al., 2025.)

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