Why has gene drive research advanced so differently in Anopheles and Aedes?

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Two mosquito vectors, two biological contexts, and different challenges for gene drive research

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Gene drive research has not advanced at the same pace across mosquito vectors of major public health importance. One question has come up repeatedly during the development of this living literature review project: why have gene drive systems, especially homing systems, appeared to progress further and faster in *Anopheles* mosquitoes than in *Aedes aegypti*?

The question matters because both mosquito groups are central to global public health. *Anopheles* mosquitoes include major malaria vectors, while *Aedes aegypti* is a principal vector of dengue and other arboviruses, including Zika, chikungunya and yellow fever viruses. Dengue provides the clearest quantitative marker of this burden. WHO reports that dengue cases increased from 505,430 in 2000 to 14.4 million in 2024, the highest number ever reported in 12 months, with more than 11,000 dengue-related deaths. Other *Aedes*-borne arboviruses add to the public health concern: chikungunya has now been reported with local transmission in 119 countries and territories, Zika transmission persists at low levels in several regions, and yellow fever outbreaks remain prone to under-reporting. From a public health perspective, then, *Aedes* is not a secondary target. It is one of the most important mosquito vectors in the world.

Yet the gene drive research trajectory has been different. In *Anopheles gambiae*, early CRISPR-based homing gene drives produced very high inheritance bias, including reported transmission rates of 91.4% to 99.6% in constructs targeting female reproduction (Hammond et al., 2016). A later gene drive targeting the female-specific transcript of doublesex reached 100% prevalence in caged *An. gambiae* populations within 7–11 generations and caused total population collapse under laboratory conditions (Kyrou et al., 2018). These studies became reference points for the field, shaping expectations about what efficient mosquito gene drives might look like.

Aedes aegypti has presented a more difficult path. Several recent papers explicitly frame their work around the gap between *Aedes* and *Anopheles* homing-drive performance. Attempts in *Ae. aegypti* have generally not matched the highest drive efficiencies observed in *Anopheles* species, and a recent cross-species meta-analysis reinforces this pattern, estimating higher predicted inheritance rates in *An. gambiae* and *An. stephensi* than in *Ae. aegypti* (Anderson et al., 2023; Ang et al., 2025; Verkuijl et al., 2026).

But this should not be read as a simple story of success versus failure. *Aedes* gene drive research has made substantial progress. Recent studies have reported split gene drive systems in *Ae. aegypti* with transmission rates as high as 94%; improved inheritance bias using germline regulatory elements, with one system reaching average inheritance of around 86% from males and 94% from

females; a multiplexed, confinable CRISPR/Cas9 split-drive that increased in caged *Ae. aegypti* populations from 50% to up to 89% carrier frequency; and low-threshold population replacement drives that reached average carrier frequencies of 72% and 82% by generation 16, depending on the system (Li et al., 2020; Anderson et al., 2023; Anderson et al., 2024; Speth et al., 2025).

What emerges from the literature is not a story of *Aedes* falling behind, but a clearer picture of why species biology matters. Gene drive systems operate inside the biology of the mosquito, and their performance depends on how well the system fits that biological context.

In the *Anopheles* vs *Aedes* comparison, several interacting layers appear to matter: germline expression, DNA repair, resistance allele formation, egg biology, urban ecology, target choice and effector biology. None of these factors alone explains the whole difference. Together, however, they show that the comparison is not just a change of vector species or disease target. *Ae. aegypti* brings a different biological context, and that context can shape how gene drive systems perform.

Germline biology and DNA repair: the timing problem

For homing gene drives, timing is everything. These systems depend on biased inheritance: the drive-containing chromosome must be copied, or otherwise preferentially transmitted, so that the genetic element is inherited more often than expected under ordinary Mendelian inheritance. In broad terms, this requires molecular activity in the germline, at a developmental moment when the cell is more likely to repair a DNA break using the homologous chromosome as a template. If the relevant activity happens too early, too late, or in the wrong tissue, the outcome can shift away from efficient copying and toward mutations that block the drive.

This is why germline biology has become such a central theme in the *Aedes* literature. Anderson et al. (2023) explain that efficient homing requires nuclease activity during, or immediately before, meiosis in diploid germline cells. If breaks occur earlier in mitotically active germline cells, or after homologous chromosomes are no longer available, repair is less likely to favour homing. Instead, error-prone repair can generate resistant alleles. Ang et al. (2025) describe this idea as a search for the correct “window” of activity, the period when DNA cleavage is most likely to lead to homing rather than end-joining.

In *Anopheles*, early successful systems seem to have found a relatively favourable alignment between gene drive architecture, germline expression and repair timing. Hammond et al. (2016)

emphasised that homing in *An. gambiae* was temporally and spatially confined to the germline during or before gamete formation. Kyrou et al. (2018) then demonstrated that a carefully chosen reproductive target could support efficient spread in cages without the resistant alleles preventing collapse under those laboratory conditions.

In *Aedes*, the same broad logic has been harder to translate. Li et al. (2017) was an important step because it generated transgenic *Ae. aegypti* lines expressing Cas9 in the germline and showed major improvements in genome engineering efficiency, including a more than order-of-magnitude increase in homology-directed repair in some contexts. But improving genome editing is not the same as achieving a robust gene drive.

Subsequent work showed why the problem is more complex. Reid et al. (2022) assessed single-locus CRISPR/Cas9-based drive variants in *Ae. aegypti* and found that genomic locus, promoter choice, maternal effects and fitness costs all influenced modelled outcomes. Their modelling suggested that some autonomous drives placed in intergenic loci could produce temporary population replacement effects but eventually be blocked by the accumulation of gene drive-blocking indels.

Yang et al. (2024) reported approximately 60% super-Mendelian inheritance for one *Ae. aegypti* homing drive, with resistance allele formation rates that were similar to drive conversion rates in some contexts. The study also observed that many wild-type alleles in the germline remained uncut. This is important because a weak or inconsistent drive is not only slower, but may also create more opportunity for resistance to accumulate.

Recent work has pushed the field further. Anderson et al. (2023) improved *Aedes* inheritance bias by testing germline regulatory elements, concluding that, *Ae. aegypti* is not inherently recalcitrant to Cas9-based homing drives. Anderson et al. (2024) then demonstrated multi-generational spread in cages using a multiplexed split-drive design. Speth et al. (2025) added another layer, showing that two low-threshold CRISPR/Cas9-based population replacement drives could increase in *Ae. aegypti* cage populations over 16 generations, but also that different regulatory choices led to different rates of gene drive-blocking indels.

A major open question concerns DNA repair itself. Romanowski et al. (2026) reported that microhomology-mediated end joining is the predominant repair outcome at CRISPR/Cas9-induced breaks in *Ae. aegypti* embryos, with indel events largely insensitive to disruption of a classical non-homologous end joining component. The study also showed that CRISPR/Cas9 could trigger large deletions through single-strand annealing in *Aedes* embryos. These findings matter because gene drive performance depends not only on whether a break is made, but on how the mosquito repairs it.

This may be one of the clearest biological lessons from the *Aedes–Anopheles* comparison. Homing drives are not just genetic constructs. They are interventions into species-specific developmental biology. The same general design logic can behave differently when placed in a different germline, a different chromatin environment, or a different DNA repair landscape.

Resistance allele formation and the challenge of durable gene drive design

Resistance alleles are a recurring concern in gene drive research. In simple terms, a resistance allele is a target-site variant that can no longer be recognised or acted upon by the drive, while still being inherited by mosquitoes. Such alleles can arise from standing genetic variation in natural populations, or they can be generated when DNA breaks are repaired in ways that alter the target sequence without copying the drive.

The literature suggests that resistance has two different meanings in this discussion. The first is pre-existing resistance in wild populations. Schmidt et al. (2020) addressed this directly by analysing 1,280 genomes from *Anopheles gambiae*, *Anopheles coluzzii* and *Aedes aegypti*. They found that approximately 90% of protein-coding gene drive target genes included at least one potential target site without drive-resistant alleles at a frequency of 1% or higher. More specifically, 97.2% of *Anopheles gambiae sensu lato* coding genes and 92.2% of *Ae. aegypti* coding genes contained at least one such potential target site. Their conclusion was relatively reassuring: standing variation alone may not be an insurmountable barrier, especially for population modification strategies with low fitness costs.

The second meaning is resistance generated by the drive process itself. This is more central to the *Aedes* challenge. Li et al. (2020) noted that drive efficiencies can be affected by target-site availability, expression timing, host-specific factors such as meiosis and recombination, and resistant alleles formed after DNA cleavage is repaired by non-homologous or other end-joining pathways rather than homology-directed repair. Reid et al. (2022) found that gene drive-blocking indels could eventually exhaust some modelled single-locus *Aedes* drives. Yang et al. (2024) reported germline resistance allele formation of 16% for females and 3.4% for males in one *Aedes* drive context.

The design response has been to look for ways to reduce the chances that resistant alleles dominate the outcome. Some studies have explored split systems, which separate drive components and are often discussed in the context of confinement and reversibility (Li et al., 2020). Others have explored multiplexing, where multiple target sites are used to reduce the likelihood that a single resistant change blocks the whole system. Anderson et al. (2024) reported that a multiplexed *Aedes* split-drive could increase in cage populations up to 89% carrier frequency, while deep sequencing suggested that target sites remained available in ways that could help mitigate resistance, at least over the timescale studied.

Still, the lesson is not simply “multiplexing solves resistance.” Anderson et al. (2024) also observed fitness costs and acknowledged that functional mutant alleles could arise in later generations. Speth et al. (2025) showed that gene drive-blocking indel accumulation differed substantially between systems, with one accumulating such indels at more than twice the rate of the other. Verkuijl et al. (2026) add an even broader caution: species identity was the strongest predictor of homing drive

inheritance, but much variation remained unexplained, and no single design factor could account for all outcomes.

That is a useful warning. Gene drive performance cannot be predicted from one variable alone. The same broad category of system may behave differently depending on the mosquito species, insertion context, regulatory architecture, parental sex, fitness effects and repair biology.

Life history and population biology: why ecology changes gene drive expectations

The molecular biology of the germline is only one part of the story. A mosquito gene drive is not expected to operate in a test tube. It is expected to interact with a living population. Here, *Aedes aegypti* differs from many *Anopheles* systems in ways that could matter for spread, persistence and prediction.

One important difference is egg desiccation tolerance. Prasad et al. (2023) compared *Ae. aegypti* and *Anopheles stephensi* and found that *Aedes* eggs can survive drying by acquiring desiccation tolerance at a late developmental stage, whereas *Anopheles* eggs showed poor survival under similar conditions. The *Aedes* embryos underwent metabolic changes, including reduced central carbon metabolism, increased lipid use and polyamine accumulation, which were linked to survival during drying and recovery after rehydration.

This is not a small ecological detail. It means that part of an *Aedes* population can persist as dry eggs outside the active adult population. Eggs laid before a gene drive becomes common could hatch later and reintroduce older genotypes into the breeding population. In a laboratory cage, generations may be more synchronised and observable. In an urban environment with containers, rainfall variation and intermittent breeding sites, the genetic composition of adults at one moment may not fully represent the genetic reservoir capable of producing future adults.

Does this mean egg desiccation will necessarily prevent gene drive spread in *Aedes*? The current note set does not support such a strong conclusion. The more careful interpretation is that desiccation-tolerant eggs add a layer of population memory. They may affect seasonality, recolonisation, genotype turnover and the interpretation of monitoring data. This deserves more explicit modelling and empirical attention.

The second life-history issue is urban fragmentation. *Ae. aegypti* is strongly associated with human habitats and commonly breeds in small artificial containers. Patil et al. (2022) emphasise that *Ae. aegypti* control is complicated by oviposition in small water containers, anthropophilic behaviour and multiple host biting. Anderson et al. (2024) similarly highlight the invasive nature of *Ae. aegypti* and its rapid adaptation to densely populated cities and towns. These features make *Aedes* a highly successful urban vector, but they also create fragmented population structures.

Sanchez et al. (2020) modelled threshold-dependent systems in spatially explicit *Ae. aegypti* populations in an Australian urban setting. Their study showed that local population structure, density dependence, seasonality and spatial heterogeneity can strongly shape expectations about confinement and reversibility. One of their most important messages is that ecological work is essential for improving model predictions.

This is where the *Anopheles* comparison needs to be handled carefully. It would be too simplistic to say that *Anopheles* populations are always continuous and *Aedes* populations are always fragmented. *Anopheles* species differ widely in ecology, breeding habitat and population structure. However, the dominant *Aedes aegypti* use case is often urban, container-breeding and strongly human-associated, whereas many prominent *Anopheles* gene drive studies have been framed around malaria-vector systems with different ecological assumptions. That difference can affect how researchers think about spread, confinement, monitoring and population replacement.

Patil et al. (2022) is useful here even though it is not a gene drive study. It reported the elimination of closed *Ae. aegypti* populations in outdoor containment facilities in India through releases of self-limiting male mosquitoes, with elimination occurring within 10 to 15 weeks in treated cages. The study shows that *Aedes* population suppression can be achievable in relatively isolated conditions. But it also underlines the importance of context: contained or isolated settings are not the same as complex urban landscapes with many breeding sites and possible reintroductions.

Suppression or replacement? How target choice differs between *Anopheles* and *Aedes*

Another biological difference concerns what the intervention is trying to achieve. Much of the most visible *Anopheles* work has focused on population suppression, especially by targeting female reproduction. In this case, the biological goal was clear: reduce the reproductive capacity of the vector population.

For *Aedes aegypti*, a great deal of interest has centred on population replacement: spreading a trait that reduces the mosquito's ability to transmit arboviruses. Reid et al. (2021) review the development of arbovirus-resistant *Ae. aegypti* and emphasise the need to link antiviral effectors to robust gene drives so that the trait can spread through target populations. This is biologically different from population suppression. It requires not only inheritance bias, but also an effector that remains stable, imposes limited fitness costs and works against relevant viruses.

That is a demanding combination. Arboviruses can be diverse and variable. Li et al. (2020) note that available antiviral effectors for *Ae. aegypti* remain limited, and that many rely on small RNA pathway-directed targeting of viral genomes. Reid et al. (2021) similarly describe effectors based on long double-stranded RNAs, microRNAs or ribozymes, while also highlighting concerns around resistant allele formation and the need for recall or overwriting strategies. The biological burden is therefore twofold: the drive must spread, and the effector must remain epidemiologically useful.

Some *Aedes* studies use visible marker or model loci to study drive behaviour rather than directly implementing disease refractoriness. Li et al. (2020), Anderson et al. (2023), Anderson et al. (2024) and Yang et al. (2024) all contribute to understanding drive performance in *Aedes*, but their immediate experimental targets should not be confused with final public health endpoints. Demonstrating biased inheritance in *Aedes* is a milestone. Demonstrating durable, epidemiologically meaningful population replacement is a much larger challenge.

At the same time, the *Aedes* literature is broadening beyond homing systems. Ang et al. (2025) discuss alternatives such as X-shredding and toxin-antidote systems, noting that some of these depend on cutting rather than cutting plus homing. Nevard et al. (2025) explored the barnase-barstar toxin-antidote pair in *Ae. aegypti* as a possible basis for genetic biocontrol systems. This does not answer the *Anopheles–Aedes* homing question directly, but it shows that *Aedes* research may not need to follow exactly the same technological path as *Anopheles*.

The issue is not only whether *Aedes* can “catch up” with *Anopheles* homing drives. It is also whether *Aedes* biology points toward different forms of genetic biocontrol, different deployment concepts and different evidence needs.

What the *Anopheles–Aedes* comparison means for research and decision-making

The comparison between *Anopheles* and *Aedes* is useful because it moves the discussion beyond a simple story of success or delay. The literature does not suggest that *Aedes aegypti* is a poor target for gene drive research. Rather, it shows that each mosquito species brings its own biological conditions, and these conditions shape how gene drive systems behave.

One encouraging message is that the gap between *Anopheles* and *Aedes* now looks less like an absolute barrier and more like a problem of biological fit. In *Anopheles*, some early systems found a productive combination of germline activity, target choice and population-suppression logic. In *Aedes*, that combination has been harder to identify, but recent work shows that performance can improve when systems are better aligned with Aedes biology.

This has an important implication: results from one mosquito genus should inform, but not substitute for, evidence from another. Decision-makers should therefore avoid treating “gene drive in mosquitoes” as a single evidence category. Evidence from *Anopheles* can help frame useful questions for *Aedes*, but it cannot answer all of them. Risk assessment, modelling, monitoring and governance need to be species-specific, disease-specific and ecology-specific.

The literature also points toward a more integrated research agenda. Germline expression, DNA repair, resistance formation, fitness costs, egg biology and population structure cannot be treated as separate problems. They interact. A system that performs well at the molecular level may still be affected by ecological features such as desiccation-tolerant eggs, fragmented urban populations or seasonal breeding patterns.

This is why *Aedes* may become especially informative for the wider field. Its biology is demanding, but that makes it scientifically valuable. By studying why *Aedes* systems perform differently from *Anopheles* systems, researchers can build a more realistic understanding of gene drive biology across species.

Several open questions remain. How predictable are DNA repair outcomes in different mosquito species and tissues? Can the most favourable germline window for homing be identified more reliably in *Aedes*? How much do egg banks and urban population structure affect spread in realistic settings? What level of fitness cost can population replacement systems tolerate? And how should antiviral effectors be evaluated when arboviruses are diverse and changing?

These questions are signs of a maturing field. The early question was whether gene drive inheritance bias could be achieved at all. The current question is more refined: under what biological, ecological and evolutionary conditions can it become stable, predictable and useful?

Gene drive should not be assessed as a uniform technology moving from one mosquito to another. It should be assessed as a set of species-specific approaches whose behaviour depends on the organism, the disease system, the intended purpose and the ecological context in which inheritance unfolds.

Anopheles and *Aedes aegypti* at a glance: biological factors shaping gene drive research

Biological feature	***Anopheles***	***Aedes aegypti***	**Why it matters**
Public health context	Includes major malaria vectors.	Major urban vector of dengue, Zika, chikungunya and yellow fever viruses.	The disease system shapes the research goal.
Main gene drive objective	Prominent studies often focus on population suppression.	Much research focuses on population replacement for arbovirus control.	Suppression and replacement require different biological strategies.
Homing-drive progress	Early systems achieved very high inheritance bias and strong cage outcomes.	Early systems were less consistent, although recent results have improved substantially.	This creates the central question of why progress has differed.
Germline timing	Some systems aligned well with the timing needed for efficient homing.	More optimisation has been needed to identify suitable germline regulation.	Homing depends on activity occurring in the right cells at the right time.

Biological feature	*Anopheles*	*Aedes aegypti*	Why it matters
DNA repair and resistance	Some constrained targets supported strong laboratory outcomes.	Resistance alleles, indels, maternal effects and fitness costs remain recurring concerns.	Repair outcomes can support drive copying or generate resistance.
Life history	Eggs are generally more dependent on suitable aquatic habitats.	Eggs can tolerate dry periods and hatch later when water returns.	Egg survival may affect population turnover and genetic dynamics.
Population ecology	Ecologies vary, but many studies are framed around malaria-vector systems.	Strongly associated with human settlements, containers and fragmented urban habitats.	Spatial structure affects spread, modelling and monitoring.
Main takeaway	Shows that highly efficient mosquito homing drives are biologically possible.	Shows that gene drive performance must be adapted to species-specific biology.	The comparison is not success versus failure, but biological fit.

Frequently asked questions (FAQs)

Why have gene drives advanced faster in *Anopheles* mosquitoes than in *Aedes aegypti*?

Gene drive research in *Anopheles* has advanced faster in some systems because early studies found a favourable alignment between germline activity, DNA repair timing, target choice and population-suppression strategies. In *Aedes aegypti*, this alignment has been harder to achieve, especially for homing gene drives. This does not mean that *Aedes* is unsuitable for gene drive research. It means that its biology has required more optimisation and interpretation.

Can gene drives work in *Aedes aegypti* mosquitoes?

Yes. Recent studies show clear progress in *Aedes aegypti* gene drive research, including improved inheritance bias and multi-generational spread in cage populations. The point is not that gene drives cannot work in *Aedes aegypti*, but that their performance has generally been more difficult to optimise than in some *Anopheles* systems.

What makes *Aedes* gene drives more challenging than *Anopheles* gene drives?

Several biological factors may contribute, including germline expression, DNA repair outcomes, resistance allele formation, fitness costs, egg desiccation tolerance and fragmented urban population structure. These factors interact, so the challenge is not only molecular. It is also ecological and evolutionary.

Why does *Aedes aegypti* egg desiccation matter for gene drive research?

Aedes aegypti eggs can survive dry periods, which means part of the population may persist outside the active adult population. This could affect how genetic traits move through populations over time, especially in urban environments with seasonal or intermittent breeding sites. The significance of this for gene drive spread remains an open question.

What should decision-makers know about gene drives in *Anopheles* and *Aedes* mosquitoes?

Gene drive should not be assessed as a single uniform technology. Evidence from *Anopheles* can help inform *Aedes* research, but it cannot replace species-specific evidence. Risk assessment, modelling, monitoring and governance need to consider the mosquito species, the disease system and the ecological context.

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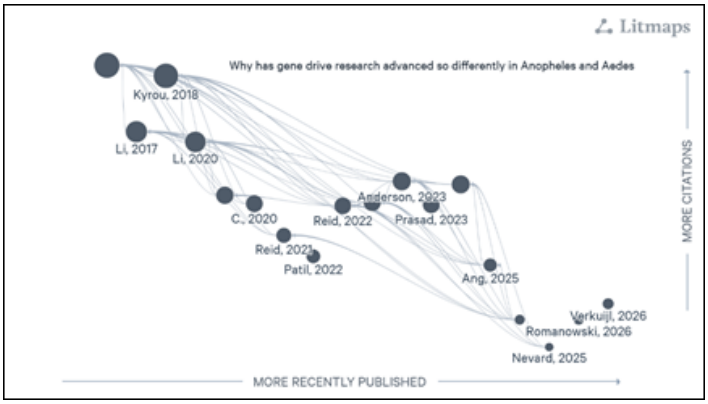

Literature map showing connections between selected studies on Aedes gene drive. Nodes represent publications and are linked through citation relationships.

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