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Appendix Figure 5. Partial rank correlation coefficients (PRCCs) of model parameters.

References

* This supplementary material was provided by the authors to give readers further details on their article. The material was reviewed but not copyedited.
Transmission Model

We developed a homogeneous mixing disease transmission model of the introduction and subsequent spread of Zika virus within a population that was not previously exposed to the virus, as is the case with the 2015 Zika outbreak in the Americas. We considered a vector-human cycle transmission of Zika, where the human population is stratified by sex (males and females), age (pre-reproductive age, reproductive age, and post-reproductive), and pregnancy status. The equations for pregnant females were further stratified according to early and later stages to allow us to conservatively assume that only early pregnancy Zika infection can lead to microcephaly and other fetal complications including fetal death, placental insufficiency, fetal growth restriction, and central nervous system abnormalities (57). As a homogeneous mixing model, our model does not explicitly account for human and vector mixing pattern or geographic variation in human and mosquito population densities.

We represented the natural history of Zika infection among humans using a compartmental susceptible-exposed-infectious-recovered (SEIR) structure and assuming a lifelong immunity following infection (Figure S1). This assumption is consistent with closely related flaviviruses such as dengue virus (19, 20, 58) and yellow fever virus (21), as well as the presence of neutralizing Zika virus antibodies observed in previously infected individuals (55). The Ae. Aegypti vector population is divided into three epidemiological classes: susceptible \((S_\nu)\), exposed \((E_\nu)\), and infected \((I_\nu)\).

**Mosquitoes:**

\[
\frac{dS_\nu}{dt} = b_\nu(S_\nu + E_\nu + I_\nu) - \left( c_\beta \left( \sum_u (I_u^+ + \eta_u I_u^-) / N_u + d_\nu \right) \right) S_\nu
\]

\[
\frac{dE_\nu}{dt} = \frac{dS_\nu}{dt}.
\]

Change in susceptible mosquitoes over time = \( \frac{dS_\nu}{dt} \).

Change in exposed mosquitoes over time = \( \frac{dE_\nu}{dt} \).
\[
\frac{dE_v}{dt} = \left( c\beta_v \left( \sum_u (I_u^w + \eta^u I_u^w) / N_u \right) \right) S_v - (\tau_v + d_o) E_v
\]

\[
\frac{dI_v}{dt} = \tau_v E_v - d_o I_v
\]

**Humans of pre-reproductive age:**

\[
\frac{dS_i}{dt} = \frac{dE_i}{dt} = \frac{dI_i}{dt} = \frac{dR_i}{dt}
\]

\[
\frac{dS_i}{dt} = b_i \theta_i \left( S_i^2 + I_i^2 + R_i^2 \right) - \rho_i S_i - \left( c\beta_i I_v / N_h \right) S_i
\]

\[
\frac{dE_i}{dt} = -\rho_i E_i + \left( c\beta_i I_v / N_h \right) S_i - \alpha E_i
\]

\[
\frac{dI_i}{dt} = -\rho_i I_i + \alpha E_i - \gamma I_i
\]

\[
\frac{dR_i}{dt} = -\rho_i R_i + \gamma I_i
\]

**Males of reproductive age:**

\[
\frac{dS_m}{dt} = \rho_m S_m - \rho_m S_m - \left( c\beta_v I_v / N_h \right) S_m
\]

Change in infected mosquitoes over time = \( \frac{dS_i}{dt} \).
Change in exposed reproductive age males over time = \( \frac{dE^2_m}{dt} \).

\[
\frac{dE^2_m}{dt} = \rho_m^1 E^1_m - \rho_m^2 E^2_m + (c \beta_m I_m / N_m) S^2_m - \alpha E^2_m
\]

Change in infected reproductive age males over time = \( \frac{dI^2_m}{dt} \).

\[
\frac{dI^2_m}{dt} = \rho_m^1 I^1_m - \rho_m^2 I^2_m + \alpha E^2_m - \gamma I^2_m
\]

Change in recovered reproductive age males over time = \( \frac{dR^2_m}{dt} \).

\[
\frac{dR^2_m}{dt} = \rho_m^1 R^1_m - \rho_m^2 R^2_m + \gamma I^2_m
\]

Females of reproductive age not pregnant:

Change in susceptible reproductive age not pregnant females over time = \( \frac{dS^2_f}{dt} \).

\[
\frac{dS^2_f}{dt} = \rho_f^1 S^1_f - \rho_f^2 S^2_f - (c \eta_f^1 \beta_f I_f / N_f) S^2_f - p_{hf} S^2_f + b_{hf} S^2_{pf}
\]

Change in exposed reproductive age not pregnant females over time = \( \frac{dE^2_f}{dt} \).

\[
\frac{dE^2_f}{dt} = \rho_f^1 E^1_f - \rho_f^2 E^2_f + (c \eta_f^1 \beta_f I_f / N_f) S^2_f - \alpha E^2_f - p_{hf} E^2_f + b_{hf} E^2_{pf}
\]

Change in infected reproductive age not pregnant females over time = \( \frac{dI^2_f}{dt} \).

\[
\frac{dI^2_f}{dt} = \rho_f^1 I^1_f - \rho_f^2 I^2_f + \alpha E^2_f - \gamma I^2_f - p_{hf} I^2_f + b_{hf} I^2_{pf}
\]

Change in recovered reproductive age not pregnant females over time = \( \frac{dR^2_f}{dt} \).

\[
\frac{dR^2_f}{dt} = \rho_f^1 R^1_f - \rho_f^2 R^2_f + \gamma I^2_f - p_{hf} R^2_f + b_{hf} R^2_{pf}
\]
Females of reproductive age in early pregnancy:

\[ \frac{dS^2_{p_f}}{dt} = p_h S^2_f - \left( c n^2_1 \beta I^1 / N^1_h \right) S^2_{p_f} - \delta S^2_{p_f} \]

Change in susceptible reproductive age in early pregnancy females over time = \[ \frac{dE^2_{p_f}}{dt} \]

\[ \frac{dE^2_{p_f}}{dt} = p_h E^2_f + \left( c n^2_1 \beta I^1 / N^1_h \right) S^2_{p_f} - \alpha E^2_{p_f} - \delta E^2_{p_f} \]

Change in exposed reproductive age in early pregnancy females over time = \[ \frac{dI^2_{p_f}}{dt} \]

\[ \frac{dI^2_{p_f}}{dt} = p_h I^2_f + \alpha E^2_{p_f} - \gamma I^2_{p_f} - \delta I^2_{p_f} \]

Change in infected reproductive age in early pregnancy females over time = \[ \frac{dR^2_{p_f}}{dt} \]

\[ \frac{dR^2_{p_f}}{dt} = p_h R^2_f + \gamma I^2_{p_f} - \delta R^2_{p_f} \]

Females of reproductive age in later pregnancy:

Change in susceptible reproductive age in later pregnancy females over time = \[ \frac{dS^3_{p_f}}{dt} \]

\[ \frac{dS^3_{p_f}}{dt} = \delta S^3_{p_f} - \left( c n^3_1 \beta I^3 / N^3_h \right) S^3_{p_f} - b_h S^3_{p_f} \]

Change in exposed reproductive age in later pregnancy females over time = \[ \frac{dE^3_{p_f}}{dt} \]

\[ \frac{dE^3_{p_f}}{dt} = \delta E^3_{p_f} + \left( c n^3_1 \beta I^3 / N^3_h \right) S^3_{p_f} - \alpha E^3_{p_f} - b_h E^3_{p_f} \]
Change in infected reproductive age in later pregnancy females over time = \( \frac{dI_{p_{2}}^2}{dt} \).
\[
\frac{dI_{p_{2}}^2}{dt} = \beta I_{p_{2}}^2 + \alpha E_{p_{2}}^2 - \gamma I_{p_{2}}^2 - b_{p_{2}}I_{p_{2}}^2
\]

Change in recovered reproductive age in later pregnancy females over time = \( \frac{dR_{p_{2}}^2}{dt} \).
\[
\frac{dR_{p_{2}}^2}{dt} = \delta R_{p_{2}}^2 + \gamma I_{p_{2}}^2 - b_{p_{2}}R_{p_{2}}^2
\]

Humans of post-reproductive age:

Change in susceptible post-reproductive age humans over time = \( \frac{dS_{i}^3}{dt} \).
\[
\frac{dS_{i}^3}{dt} = \rho_{i}^3 S_{i}^3 - \rho_{i}^3 S_{i}^3 - (c_{i} \beta_{i} I_{v} / N_{H}) S_{i}^3
\]

Change in exposed post-reproductive age humans over time = \( \frac{dE_{i}^3}{dt} \).
\[
\frac{dE_{i}^3}{dt} = \rho_{i}^3 E_{i}^3 - \rho_{i}^3 E_{i}^3 + (c_{i} \beta_{i} I_{v} / N_{H}) S_{i}^3 - \gamma E_{i}^3
\]

Change in infected post-reproductive age humans over time = \( \frac{dI_{i}^3}{dt} \).
\[
\frac{dI_{i}^3}{dt} = \rho_{i}^3 I_{i}^3 - \rho_{i}^3 I_{i}^3 + \alpha E_{i}^3 - \gamma I_{i}^3
\]

Change in recovered post-reproductive age humans over time = \( \frac{dR_{i}^3}{dt} \).
\[
\frac{dR_{i}^3}{dt} = \rho_{i}^3 R_{i}^3 - \rho_{i}^3 R_{i}^3 + \gamma I_{i}^3,
\]

\[
N_{H} = \sum_{i,b} (S_{i}^a + E_{i}^a + I_{i}^a + R_{i}^a)
\]
where \( N_{H} \) is the total human population size. To capture seasonal variation in

\( Ae. Aegypti \) population dynamics, we defined the \( Ae. Aegypti \) birth rate as follow

\[
b_{o} = d_{o}(1 + 0.25 \sin(\pi t / 182 + 1.0472)) \quad (19)
\]
empirical entomological data on *Ae. Aegypti* in Brazil from previous studies (19). In the mosquito population, $S_v$ denotes the number of susceptible mosquitoes, $E_v$ the number of exposed mosquitoes, and $I_v$ the number of infected mosquitoes. Denoting by $N_{v_{\text{max}}}$ the total mosquito population size at its peak value during a seasonal cycle, we can scale the mosquito population relative to $N_{v_{\text{max}}}$, such that

$$\tilde{S}_v = S_v / N_{v_{\text{max}}}, \tilde{E}_v = E_v / N_{v_{\text{max}}}, \tilde{I}_v = I_v / N_{v_{\text{max}}}.$$  

We then convert $c\beta_v I_v / N_h$ into $\tilde{c}\tilde{\beta}_v \tilde{I}_v$, where

$$\tilde{\beta}_v = \beta_v (N_{v_{\text{max}}} / N_h)$$  

with $N_{v_{\text{max}}} / N_h$ being the *Ae. Aegypti* index (per human). For simplicity, we henceforth denote $\tilde{O}_j$ as $O_j$ for $j = S, E, I$ and $\tilde{\beta}_v$ as $\beta_v$. This formulation allows our model to implicitly account for a wide range of vector-density (vector index) settings, through parameter fitting of $\beta_v$.

The input parameters of our model are described in Appendix Table. Population distribution between gender and age-groups was informed using demographic data from Colombia, where 20% of the population are men of pre-reproductive age, 15% women of pre-reproduction age, 24% men of reproductive age, 23.8% women of reproductive age, 5.2% men of post-reproductive age, and 12% women of post-reproductive age (37). For men, reproductive age was defined as the period between the average age at first child and age at last child.

We assumed that pregnancy is at risk of Zika-related complications when women are infected during the first trimester (57). Therefore, we set the duration of early pregnancy $9^{-1}$ to be 12 weeks. To measure the risk of birth defects from Zika, perinatal infection was computed only for pregnant women who are infected during the first trimester of pregnancy. In an alternative scenario analysis, we also considered that infection at any point can cause microcephaly.

Epidemiological studies on dengue in the Americas have observed that women are more frequently infected than men (59), and Zika cases data from Colombia exhibit a disproportionate number of reported cases among women of reproductive age (24). This high exposure may be explained by the
tendency for *Ae aegypti* to reside primarily in and around human habitation and women of reproductive age are more likely to be spending more time at home than men and children, resulting in higher exposure to *Ae aegypti* bites. In addition, sexual transmission of Zika from men to women of reproductive age would also increase the risk of infection among them. To account for these empirical observations of higher risk of infection for women of reproductive age, we included a factor $\eta_f^2$ within the force of infection for these women. This also allows us to capture the effect of sexual transmission, although sexual transmission is not explicitly modeled due to insufficient epidemiological data. A description of epidemiological parameters is given in Appendix Table.

**Model Fitting**

We calibrated the model to epidemiological weekly data of suspected and confirmed Zika cases in Columbia between October 11th 2015 and March 31st 2016 from the ongoing outbreak (24), to estimate posterior distributions of the following epidemiological parameters: $\beta_c$, mosquito-to-human transmission, $\beta_h$, human-to-mosquito transmission, $c$, biting rate, $\alpha^{-1}$, human incubation period, $\gamma^{-1}$, human infectious period, $r$, proportion of symptomatic Zika cases, $\Lambda$, epidemic attack rate, $\eta_f^2$, elevated risk of Zika infection for women of reproductive age, $\tau_r^{-1}$, mosquito incubation period, governing Zika transmission, $d_o^{-1}$, *Ae. Aegypti* lifespan, $I_{h0}$, number of human Zika cases at epidemic start, $I_{v0}$, proportion of infected mosquitoes at epidemic start.

To compare model predictions with observed data, we applied a Bayesian melding approach (42) (Figure S2), incorporating a likelihood function and using all available prior information regarding model inputs to generate posterior distributions of model parameters. We used prior distributions defined from epidemiological and clinical studies (Appendix Table) for each of the input model parameters. To account for uncertainty regarding Zika epidemiological and entomological parameters, we defined a prior distribution for each unknown model input parameter as a wide range of plausible values applicable both
to dengue and chikungunya. Through our application of a Bayesian framework, we propagated both data and parameters uncertainty into the credible intervals of our model projections.

To account for over-dispersion in reported cases, we applied a negative binomial distribution for Zika weekly incidence data with mean \( \sum_{\text{week}} \alpha E_i^w(t) \) and dispersion parameter \( \Phi \) (60), where \( \alpha E_i^w(t) \) is the weekly incidence of Zika cases and \( r \) is the proportion of symptomatic cases. To account for heterogeneity in infection between sex and age groups, we used beta distributions for both the proportion of cases that are women and the proportion of cases that are individuals of reproductive ages (47). Given that the distribution of \textit{Aedes aegypti} infestation is not homogeneous across Colombia, and that all Colombians are not necessarily at risk of Zika infection, we derived the number of individuals at direct risk for Zika prior to the outbreak as a function of the proportion of symptomatic/reported cases, \( r \), and the expected attack rate of the first wave of the outbreak, \( \Lambda \), which informed was informed by the attack rate of previous Zika outbreaks (4,55,56).

We considered two disease transmission scenarios: in the first scenario all infected individuals equally contribute to transmission; in the second scenario we assumed that only symptomatic individuals are infectious. The first scenario is described by the above system of equations. For the second scenario, we assumed only a proportion \( r \) of infected individuals are infectious to mosquitoes.

To detail the Bayesian melding algorithm, we denote the simulation model by \( M \), the epidemiological parameters (Appendix Table) by \( \Theta \), and the model-predicted output by \( \Gamma = M(\Theta) \). We denote the prior distribution for each model parameter by \( q(\Theta) \). We denote the data by \( W \) and the associated likelihood of the model outputs by \( L(\Gamma) = \Pr(\Gamma/W) \). The posterior distribution of inputs is then proportional to \( q(\Theta)L(\Gamma) \). We implemented a sample-importance-resample algorithm (23) to approximate the posterior distribution. Specifically, we first generated a set of input parameters, \( \Theta(i) \) by randomly sampling from the respective prior distributions \( i \) times. We then evaluated model output using
the sampled set of parameters, $\Gamma(i) = M(\Theta(i))$, for each run $i$. Next, we calculated the corresponding likelihood for the model run. For each sample $\Theta(j)$, with non-zero corresponding likelihood, the sampling weight was

$$\Omega(j) = L(\Gamma(j))/\sum_{k=1}^{nT} L(\Gamma(k))$$

To ensure a sufficient sample from the posterior distributions, we set $i = 200,000$. We denoted the number of non-zero likelihood samples by $nT$. To obtain an approximation of the posterior distribution for the inputs, we repeated this sampling procedure 10,000 times with replacement, with a probability of selection proportional to the sampling weights. Output from the simulation resampled most frequently (i.e., the simulation that most closely fit the empirical prevalence data) represented the estimated mode for the output parameters of interest. The 2.5$^{th}$ and 97.5$^{th}$ percentiles of the inputs (and corresponding model outputs) correspond to 95% credible limits. Once fitted, we validated our model projection of symptomatic cases by comparing it against weekly reported data from April 1$^{st}$ to May 8$^{th}$ 2016 (24).

**Interventions**

We used our calibrated model to evaluate the impact of pregnancy delay on reducing prenatal exposure to Zika, and thus, Zika-induced microcephaly cases from July 2015 to June 2017. We considered two strategies for pregnancy delay: a mass and an individual-based pregnancy delay. In the mass pregnancy delay strategy, we assumed that non-pregnant women of reproductive age collectively abstain from getting pregnant during the same period as the response to the Zika outbreak, following recommendations from public health authorities. This strategy is in line with current recommendations from several health ministries in Zika-affected countries in the Americas (14). However, under this strategy, a surge in pregnancies is likely to occur after the period of abstention. In our model, we kept track of the women who would have become pregnant during the pregnancy delay period if they had not adhered to the recommendation of delaying pregnancy. After the delay period, these women transition to the “Females of reproductive age non-pregnant” classes, stratified by their epidemiological status.
In the individual-based pregnancy delay strategy, we assumed that women of reproductive age independently decide to postpone pregnancy. Here, the decision of delaying pregnancy is made continuously over time rather than over a fixed period of time as with the mass delay strategy. As with mass pregnancy, we tracked a pregnancy delay group. However, contrary to the mass delay strategy, women in the delayed group were continuously transitioning into “Females of reproductive age in early pregnancy” at a rate equal to the inverse of the delay period.
## Appendix Table: Epidemiological parameters and distributions under the assumption that only symptomatic cases are infectious.

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Parameter</th>
<th>Prior distribution (refs)</th>
<th>Posterior distribution median (95% credible interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\theta)</td>
<td>Sex ratio at birth in Colombia</td>
<td>97 males/100 females (37)</td>
<td>NA</td>
</tr>
<tr>
<td>(\rho_1^{-1})</td>
<td>Average age at first child in the Americas (males, (i=m); females, (i=f))</td>
<td>25 years for men (38); 21.7 years for women (39)</td>
<td>NA</td>
</tr>
<tr>
<td>(\rho_2^{-1})</td>
<td>Human reproductive period in the Americas (males, (i=m); females, (i=f))</td>
<td>35 years for men (38,40); 26.8 years for women (41)</td>
<td>NA</td>
</tr>
<tr>
<td>(\rho_3^{-1})</td>
<td>Human lifespan post-reproduction in the Americas (males, (i=m); females, (i=f))</td>
<td>11.8 years for men (40,42); 29.8 years for women (37,39,41)</td>
<td>NA</td>
</tr>
<tr>
<td>(\rho_4^{-1})</td>
<td>Average number of children per woman in Colombia</td>
<td>1.90(37)</td>
<td>NA</td>
</tr>
<tr>
<td>((b_m + \vartheta)^{-1})</td>
<td>Gestation period</td>
<td>40 weeks</td>
<td>NA</td>
</tr>
<tr>
<td>(d_o^{-1})</td>
<td>(Ae.\ aegypti) lifespan</td>
<td>Uniform(8,42) days (43–47)</td>
<td>15.2 (95%CI: 10.6 – 41.1)</td>
</tr>
<tr>
<td>(\tau_v^{-1})</td>
<td>Mosquito incubation period</td>
<td>Gamma(12,1) days (48)</td>
<td>S1: 6.7 (95% CI: 6.2 – 21.6)</td>
</tr>
<tr>
<td>(\tau_v^{-1})</td>
<td>Mosquito incubation period</td>
<td>Gamma(12,1) days (48)</td>
<td>S2: 13.6 (95%CI: 6.3 – 20.0)</td>
</tr>
<tr>
<td>(c)</td>
<td>Biting rate</td>
<td>Uniform(0.3,1) day(^{-1}) (49,50)</td>
<td>0.90 (95%CI: 0.34 – 0.96)</td>
</tr>
<tr>
<td>(\beta_v)</td>
<td>Mosquito-to-human transmission</td>
<td>Uniform(0.001,1) (51)</td>
<td>0.03 (95%CI: 0.02 – 0.66)</td>
</tr>
<tr>
<td>(\beta_h)</td>
<td>Human-to-mosquito transmission</td>
<td>Uniform(0.001,0.75) (49,51)</td>
<td>0.51 (95%CI: 0.04 – 0.73)</td>
</tr>
<tr>
<td>(\alpha^{-1})</td>
<td>Human incubation period</td>
<td>Uniform(6,17) days (52,53)</td>
<td>10.2 (95%CI: 6.4 – 16.8)</td>
</tr>
<tr>
<td>(\gamma^{-1})</td>
<td>Human infectious period</td>
<td>Uniform(3,22) days (53,54)</td>
<td>19.6 (95%CI: 3.5 – 21.5)</td>
</tr>
<tr>
<td>(r)</td>
<td>Proportion of symptomatic Zika infections</td>
<td>Uniform(0.09,0.27) (4)</td>
<td>0.14 (95%CI: 0.093 – 0.26)</td>
</tr>
<tr>
<td>(\Lambda)</td>
<td>Expected attack rate</td>
<td>Uniform (0.5,0.77) (4,55,56)</td>
<td>0.60 (95%CI: 0.51 – 0.76)</td>
</tr>
<tr>
<td>(\varphi)</td>
<td>Reporting dispersion (describes the underlying variation in reported cases count)</td>
<td>Uniform(0,(\infty))</td>
<td>7.2 (95%CI: 0.8 - 84.6)</td>
</tr>
<tr>
<td>(\eta_f^2)</td>
<td>Elevated risk of exposure to Zika infection for women of reproductive age</td>
<td>Uniform(1,10)</td>
<td>4.3 (95%CI: 2.6 – 4.8)</td>
</tr>
<tr>
<td>$\eta_1, \eta_3$</td>
<td>Elevated risk of exposure to Zika infection for women of pre- and post-reproductive age</td>
<td>1</td>
<td>NA</td>
</tr>
<tr>
<td>-----------------</td>
<td>-----------------------------------------------------------------</td>
<td>----</td>
<td>----</td>
</tr>
<tr>
<td>$I_{H0}$</td>
<td>Number of human Zika cases at epidemic start</td>
<td>Uniform(1.20)</td>
<td>32 (95%CI: 2 – 49)</td>
</tr>
<tr>
<td>$I_{F0}$</td>
<td>Proportion of infected mosquitoes at epidemic start</td>
<td>Uniform(0.002)</td>
<td>0.005 (95%CI: 0 – 0.0047)</td>
</tr>
</tbody>
</table>
Appendix Figure 1: Model diagram of epidemiological compartments (circles) with rates of movement between each compartment (arrows). Further details are presented in the text.
**Appendix Figure 2:** Illustration of the Bayesian melding procedure, in which prior knowledge is drawn from to produce an ensemble of model output that can be sampled from by its likelihood to yield an importance weight. The importance weight of each model then informs the degree to which that model estimate should contribute to the outcome estimate.
Appendix Figure 3: One-way sensitivity analysis of the impact of epidemiological parameters to (A) total microcephaly cases and (B) timing to epidemic incidence peak from epidemic onset.
Appendix Figure 4: Trajectories of the model fitted to data from the October 2015 to March 2016 Zika outbreak in Colombia (black asterisks). The solid line represents the mode of the posterior sample, the dashed line represents the mean, and the grey area indicates the 95% credible interval. Model projections were validated against data from April to May 2016 (red asterisks). Here fitting was conducted under the assumption that only symptomatic cases are infectious.
Appendix Figure 5: Partial rank correlation coefficients (PRCCs) of model parameters. A) Only symptomatic are infectious, B) symptomatic and asymptomatic are equally infectious. A parameter was considered to be important if |PRCC| > 0.5. Parameters with PRCC absolute values higher than 0.2 were statistically significant (P-value < 0.05); $\beta_s$ human-to-mosquito transmission, $\beta_v$ mosquito-to-human transmission, $c$ biting rate, $\alpha$ human incubation period, $\gamma$ human infectious period, $d_0^{-1}$ Ae. aegypti lifespan, $r$ proportion of symptomatic Zika infections, $\eta_f^2$ elevated risk of exposure to Zika infection for women of reproductive age.
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