tice it would have, for the time being, no effect, since the characteristic periods of the illness have not been measured in these terms.

Epidemics transmitted by vectors come to an end either when the susceptible population has been sufficiently exposed so that the replication of the virus is slowed down (the classical consideration in SIR models) or when the vector's population is decimated by other (for example, climatic) reasons. The model shows that both situations can be distinguished in terms of the mortality statistics.

We have also shown that the total mortality of the epidemic is not difficult to adjust by changing the death probability of the toxic phase, and as such, it is not a demanding test for a model. The daily mortality, when normalized, shows sensitivity to the mosquito abundance, specially in the evolution times involved, since the general qualitative shape appears to be fixed. In particular, the date in which the epidemic reaches half the total mortality is advanced by larger mosquito populations. However, only comparison of the simulated and historical daily mortality put enough constraints to the free data in the model (date of arrival of infected people and mosquito population) to allow for a selection of possible combinations of their values.

As successful as the model appears to be, it is completely unable to produce the total mortality in the city, or the spatial extension of the full epidemic. The simulations produce with BSx4 less than 4500 deaths, while in the historic record, the total mortality in the city is above 13000 cases. The historical account, and the recorded data, show that after the initial San Telmo focus has developed, a second focus in the police district 13 (see Fig. 2) developed, shortly several other foci developed that could not be tracked [4]. Unless the spreading of the illness by infected humans is introduced (or some other method to make long jumps by the illness), such events cannot be described. It is worth noticing that the mobility patterns in 1871 are expected to be drastically different from present patterns, and as such, the application of models with human mobility [43] is not straightforward and requires a historical study.

One of the most important conclusions of this work is that the logical consistency of mathematical modeling puts a limit to ad-hoc hypotheses, so often used in a-posteriori explanations, as it forces to accept not just the desired consequence of the hypotheses, but all other consequences as well.

Last, eco-epidemiological models are adjusted to vector populations pre-existing the actual epidemics and can therefore be used in prevention to determine epidemic risk and monitor eradication campaigns. In the present work, the tuning was performed in epidemic data only because it is actually impossible to know the environmental conditions more than one hundred years ago. Yet, our wild initial guess for the density of breeding sites resulted sufficiently close to allow further tuning.

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A Appendix

i. Populations and events of the stochastic transmission model

We consider a two dimensional space as a mesh of squared patches where the dynamics of vectors, hosts and the disease take place. Only adult mosquitoes, Flyers, can fly from one patch to a next one according to a diffusion-like process. The coordinates of a patch are given by two indices, i and j, corresponding to the row and column in the mesh. If X_k is a subpopulation in the stage k, then $X_k(i,j)$ is the X_k subpopulation in the patch of coordinates (i,j).

Population of both hosts (Humans) and vectors (*Aedes aegypti*) were divided into subpopulations representing disease status: SEI for the vectors and SEIrRTD for the human population.

Ten different subpopulations for the mosquito were taken into account, three immature subpopulations: eggs $E_{(i,j)}$, larvae $L_{(i,j)}$ and pupae $P_{(i,j)}$, and seven adult subpopulations: non parous adults $A1_{(i,j)}$, susceptible flyers $Fs_{(i,j)}$, exposed flyers

$Fe_{(i,j)}$, infectious flyers $Fi_{(i,j)}$ and parous adults in the three disease status: susceptible $A2s_{(i,j)}$, exposed $A2e_{(i,j)}$ and infectious $A2i_{(i,j)}$.

The $A1_{(i,j)}$ is always susceptible, after a blood meal it becomes a flyer, susceptible $Fs_{(i,j)}$ or exposed $Fe_{(i,j)}$, depending on the disease status of the host. If the host is infectious, $A1_{(i,j)}$ becomes an exposed flyer $Fe_{(i,j)}$ but if the host is not infectious, then the $A1_{(i,j)}$ becomes a susceptible flyer $Fs_{(i,j)}$. The transmission of the virus depends not only on the contact between vector and host but also on the transmission probability of the virus. In this case, we have two transmission probabilities: the transmission probability from host to vector ahv and the transmission probability from vector to host avh.

Human population $Nh_{(i,j)}$ was split into seven different subpopulations according to the disease status: susceptible humans $Hs_{(i,j)}$, exposed humans $He_{(i,j)}$, infectious humans $Hi_{(i,j)}$, humans in remission state $Hr_{(i,j)}$, toxic humans $Ht_{(i,j)}$, removed humans $HR_{(i,j)}$ and dead humans because of the disease $Hd_{(i,j)}$.

The evolution of the seventeen subpopulations is affected by events that occur at rates that depend on subpopulation values and some of them also on temperature, which is a function of time since it changes over the course of the year seasonally [8,9].

ii. Events related to immature stages

Table 4 summarizes the events and rates related to immature stages of the mosquito during their first gonotrophic cycle. The construction of the transition rates and the election of model parameters related to the mosquito biology such as: me mortality of eggs, elr hatching rate, ml mortality of larvae, α density-dependent mortality of larvae, lpr pupation rate, mp: mortality of pupae, par pupae into adults development coefficient and the ef emergence factor were described in detail previously [8, 9].

The natural regulation of *Aedes aegypti* populations is due to intra-specific competition for food and other resources in the larval stage. This regulation was incorporated into the model as a density-dependent transition probability which introduces the necessary nonlinearities that prevent a Malthusian growth of the population. This effect was incorporated as a nonlinear correction to the temperature dependent larval mortality.

Then, larval mortality can be written as: $mlL_{(i,j)} + \alpha L_{(i,j)} \times (L_{(i,j)} - 1)$ where the value of α can be further decomposed as $\alpha = \alpha_0/\mathrm{BS}_{(i,j)}$ with α_0 being associated with the carrying capacity of one (standardised) breeding site and $\mathrm{BS}_{(i,j)}$ being the density of breeding sites in the (i, j) patch [8,9].

iii. Events related to the adult stage

Aedes aegypti females ($A1$ and $A2$) require blood to complete their gonotrophic cycles. In this process, the female may ingest viruses with the blood meal from an infectious human during the human Viremic Period VP. The viruses develop within the mosquito during the Extrinsic Incubation Period EIP and then are reinjected into the blood stream of a new susceptible human with the saliva of the mosquito in later blood meals. The virus in the exposed human develops during the Intrinsic incubation Period IIP and then begin to circulate in the blood stream (Viremic Period), the human becoming infectious. The flow from susceptible to exposed subpopulations (in the vector and the host) depends not only on the contact between vector and host but also on the transmission probability of the virus. In our case, there are two transmission probabilities: the transmission probability from host to vector ahv and the transmission probability from vector to host avh.

The events related to the adult stage are shown in Table 5 to 8. Table 5 summarizes the events and rates related to adults during their first gonotrophic cycle and related to oviposition by flyers according to their disease status.

Table 6 and Table 7 summarize the events and rates related to adult 2 gonotrophic cycles, exposed Adults 2 and exposed flyers becoming infectious and human contagion.

Table 8 summarizes the events and rates related to non parous adult (Adult 2) and Flyer death.

iv. Events related to flyer dispersal

Some experimental results and observational studies show that the *Aedes aegypti* dispersal is driven by the availability of oviposition sites [44–46]. According to these observations, we considered that only the Flyers $F_{(i,j)}$ can fly from patch to patch in search of oviposition sites. The implementation of flyer dispersal has been described elsewhere [9].

Event	Effect	Transition rate
Egg death	$E_{(i,j)} \to E_{(i,j)} - 1$	$me \times E_{(i,j)}$
Egg hatching	$E_{(i,j)} \to E_{(i,j)} - 1\ L_{(i,j)} \to L_{(i,j)} + 1$	$elr \times E_{(i,j)}$
Larval death	$L_{(i,j)} \to L_{(i,j)} - 1$	$ml \times L_{(i,j)} + \alpha \times L_{(i,j)} \times (L_{(i,j)} - 1)$
Pupation	$L_{(i,j)} \to L_{(i,j)} - 1\ P_{(i,j)} \to P_{(i,j)} + 1$	$lpr \times L_{(i,j)}$
Pupal death	$P_{(i,j)} \to P_{(i,j)} - 1$	$(mp + par \times (1 - (ef/2))) \times P_{(i,j)}$
Adult emergence	$P_{(i,j)} \to P_{(i,j)} - 1$ $A1_{(i,j)} \to A1_{(i,j)} + 1$	$par \times (ef/2) \times P_{(i,j)}$

Table 4: Event type, effects on the populations and transition rates for the developmental model. The coefficients are me: mortality of eggs; elr: hatching rate; ml: mortality of larvae; α: density-dependent mortality of larvae; lpr: pupation rate; mp: mortality of pupae; par: pupae into adults development coefficient; ef: emergence factor. The values of the coefficients are available in subsections vi. and vii..

Event	Effect	Transition rate
Adults 1 Death	$A1_{(i,j)} \to A1_{(i,j)} - 1$	$ma \times A1_{(i,j)}$
I Gonotrophic cycle with virus exposure	$A1_{(i,j)} \to A1_{(i,j)} - 1$ $Fe_{(i,j)} \to Fe_{(i,j)} + 1$	$cycle1 \times A1_{(i,j)} \times (Hi_{(i,j)}/Nh_{(i,j)}) \times ahv$
I Gonotrophic cycle without virus exposure	$A1_{(i,j)} \to A1_{(i,j)} - 1$ $Fs_{(i,j)} \to Fs_{(i,j)} + 1$	$cycle1 \times A1_{(i,j)} \times ((((Nh_{(i,j)} - Hi_{(i,j)})/Nh_{(i,j)}) + (1 - ahv) \times (Hi_{(i,j)}/Nh_{(i,j)}))$
Oviposition of susceptible flyers	$E_{(i,j)} \to E_{(i,j)} + egn$ $Fs_{(i,j)} \to Fs_{(i,j)} - 1$ $A2s_{(i,j)} \to A2s_{(i,j)} + 1$	$ovr_{(i,j)} \times Fs_{(i,j)}$
Oviposition of exposed flyers	$E_{(i,j)} \to E_{(i,j)} + egn$ $Fe_{(i,j)} \to Fe_{(i,j)} - 1$ $A2e_{(i,j)} \to A2e_{(i,j)} + 1$	$ovr_{(i,j)} \times Fe_{(i,j)}$
Oviposition of infected flyers	$E_{(i,j)} \to E_{(i,j)} + egn$ $Fi_{(i,j)} \to Fi_{(i,j)} - 1$ $A2i_{(i,j)} \to A2i_{(i,j)} + 1$	$ovr_{(i,j)} \times Fi_{(i,j)}$

Table 5: Event type, effects on the subpopulations and transition rates for the developmental model. The coefficients are ma: mortality of adults; $cycle1$: gonotrophic cycle coefficient (number of daily cycles) for adult females in stages $A1$.; ahv: transmission probability from host to vector; $ovr_{(i,j)}$: oviposition rate by flyers in the (i,j) patch; egn: average number of eggs laid in an oviposition. The values of the coefficients are available in Table 1, subsections vi., vii., viii. and ix..

The general rate of the dispersal event is given by: $\beta \times F_{(i,j)}$, where β is the dispersal coefficient and $F_{(i,j)}$ is the Flyer population which can be susceptible $Fs_{(i,j)}$, exposed $Fe_{(i,j)}$ or infectious $Fi_{(i,j)}$ depending on the disease status.

The dispersal coefficient β can be written as

$$\beta = \begin{cases} 0 & \text{if the patches are disjoint} \\ diff/d_{ij}^2 & \text{if the patches have at least a common point} \end{cases} \quad (3)$$

where d_{ij} is the distance between the centres of the patches and $diff$ is a diffusion-like coefficient so that dispersal is compatible with a diffusion-like process [9].

v. Events related to human population

Human contagion has been already described in Table 7. Table 9 summarizes the events and rates in which humans are involved. The human popula-

Event	Effect	Transition rate
II Gonotrophic cycle of susceptible Adults 2 with virus exposure	$A2s_{(i,j)} \rightarrow A2s_{(i,j)} - 1$ $Fe_{(i,j)} \rightarrow Fe_{(i,j)} + 1$	$cycle2 \times A2s_{(i,j)} \times (Hi_{(i,j)}/Nh_{(i,j)}) \times ahv$
II Gonotrophic cycle of susceptible Adults 2 without virus exposure	$A2s_{(i,j)} \rightarrow A1_{(i,j)} - 1$ $Fs_{(i,j)} \rightarrow Fs_{(i,j)} + 1$	$cycle2 \times A2s_{(i,j)} \times ((((Nh_{(i,j)} - Hi_{(i,j)})/Nh_{(i,j)}) + (1 - ahv) \times (Hi_{(i,j)}/Nh_{(i,j)}))$
II Gonotrophic cycle of exposed Adults 2	$A2e_{(i,j)} \rightarrow A2e_{(i,j)} - 1$ $Fe_{(i,j)} \rightarrow Fe_{(i,j)} + 1$	$cycle2 \times A2e_{(i,j)}$

Table 6: Event type, effects on the subpopulations and transition rates for the developmental model. The coefficients are $cycle2$: gonotrophic cycle coefficient (number of daily cycles) for adult females in stages $A2.$; ahv: transmission probability from host to vector. The values of the coefficients are available in Table 1, subsections vi., vii., viii. and ix..

Event	Effect	Transition rate
Exposed Adults 2 becoming infectious	$A2e_{(i,j)} \rightarrow A2e_{(i,j)} - 1$ $A2i_{(i,j)} \rightarrow A2i_{(i,j)} + 1$	$(1/(EIP - (1/ovr_{(i,j)})))A2e_{(i,j)}$
Exposed flyers becoming infectious	$Fe_{(i,j)} \rightarrow Fe_{(i,j)} - 1$ $Fi_{(i,j)} \rightarrow Fi_{(i,j)} + 1$	$(1/(EIP - (1/ovr_{(i,j)})))Fe_{(i,j)}$
II Gonotrophic cycle of infectious Adults 2 without human contagion	$A2i_{(i,j)} \rightarrow A2i_{(i,j)} - 1$ $Fi_{(i,j)} \rightarrow Fi_{(i,j)} + 1$ $Hs_{(i,j)} \rightarrow Hs_{(i,j)} - 1$ $He_{(i,j)} \rightarrow He_{(i,j)} + 1$	$cycle2 \times A2i_{(i,j)} \times (Hs_{(i,j)}/Nh_{(i,j)}) \times avh$
II Gonotrophic cycle of infectious Adults 2 without human contagion	$A2i_{(i,j)} \rightarrow A2i_{(i,j)} - 1$ $Fi_{(i,j)} \rightarrow Fi_{(i,j)} + 1$	$cycle2 \times A2i_{(i,j)} \times ((((Nh_{(i,j)} - Hs_{(i,j)})/Nh_{(i,j)}) + (1 - avh) \times (Hs_{(i,j)}/Nh_{(i,j)}))$

Table 7: Event type, effects on the subpopulations and transition rates for the developmental model. The coefficients are $cycle2$: gonotrophic cycle coefficient (number of daily cycles) for adult females in stages $A2$; $ovr_{(i,j)}$: oviposition rate by flyers in the (i,j) patch; avh: transmission probability from vector to host; EIP: extrinsic incubation period. The values of the coefficients are available in Table 1, subsections vi., vii., viii. and ix.

tion was fluctuating but balanced, meaning that the birth coefficient was considered equal to the mortality coefficient mh.

vi. Developmental Rate coefficients

The developmental rates that correspond to egg hatching, pupation, adult emergence and the gonotrophic cycles were evaluated using the results of the thermodynamic model developed by Sharp and DeMichele [47] and simplified by Schoofield *et al.* [48]. According to this model, the maturation process is controlled by one enzyme which is active in a given temperature range and is deactivated only at high temperatures. The development is stochastic in nature and is controlled by a Poisson process with rate $R_D(T)$. In general terms, $R_D(T)$ takes the form

$$R_D(T) = R_D(298\text{ K}) \times \frac{(T/298\text{ K})\exp((\Delta H_A/R)(1/298\text{ K} - 1/T))}{1 + \exp(\Delta H_H/R)(1/T_{1/2} - 1/T))} \quad (4)$$

where T is the absolute temperature, ΔH_A and ΔH_H are thermodynamics enthalpies characteristic of the organism, R is the universal gas constant, and $T_{1/2}$ is the temperature when half of the enzyme is deactivated because of high temperature.

Event	Effect	Transition rate
Susceptible flyer Death	$Fs_{(i,j)} \to Fs_{(i,j)} - 1$	$ma \times Fs_{(i,j)}$
Exposed flyer Death	$Fe_{(i,j)} \to Fe_{(i,j)} - 1$	$ma \times Fe_{(i,j)}$
Infectious flyer Death	$Fi_{(i,j)} \to Fi_{(i,j)} - 1$	$ma \times Fi_{(i,j)}$
Susceptible Adult 2 Death	$A2s_{(i,j)} \to A2s_{(i,j)} - 1$	$ma \times A2s_{(i,j)}$
Exposed Adult 2 Death	$A2e_{(i,j)} \to A2e_{(i,j)} - 1$	$ma \times A2e_{(i,j)}$
Infectious Adult 2 Death	$A2i_{(i,j)} \to A2i_{(i,j)} - 1$	$ma \times A2i_{(i,j)}$

Table 8: Event type, effects on the subpopulations and transition rates for the developmental model. The coefficients are ma: adult mortality. The values of the coefficients are available in subsection vii.

Event	Effect	Transition rate
Born of susceptible humans	$Hs_{(i,j)} \to Hs_{(i,j)} + 1$	$mh \times Nh_{(i,j)}$
Death of susceptible humans	$Hs_{(i,j)} \to Hs_{(i,j)} - 1$	$mh \times Hs_{(i,j)}$
Death of exposed humans	$He_{(i,j)} \to He_{(i,j)} - 1$	$mh \times He_{(i,j)}$
Transition from exposed to viraemic	$He_{(i,j)} \to He_{(i,j)} - 1$ $Hi_{(i,j)} \to Hi_{(i,j)} + 1$	$(1/IIP) \times He_{(i,j)}$
Death of Infectious humans	$Hi_{(i,j)} \to Hi_{(i,j)} - 1$	$mh \times Hi_{(i,j)}$
Transition from infectious humans to humans in remission state	$Hi_{(i,j)} \to Hi_{(i,j)} - 1$ $Hr_{(i,j)} \to Hr_{(i,j)} + 1$	$(1/VP) \times Hi_{(i,j)}$
Death of humans in remission state	$Hr_{(i,j)} \to Hr_{(i,j)} - 1$	$mh \times Hr_{(i,j)}$
Transition from humans in remission to toxic humans	$Hr_{(i,j)} \to Hr_{(i,j)} - 1$ $Ht_{(i,j)} \to Ht_{(i,j)} + 1$	$((1 - rar)/rP) \times Hr_{(i,j)}$
Recovery of humans in remission	$Hr_{(i,j)} \to Hr_{(i,j)} - 1$ $HR_{(i,j)} \to HR_{(i,j)} + 1$	$(rar/rP) \times Hr_{(i,j)}$
Death of removed humans	$HR_{(i,j)} \to HR_{(i,j)} - 1$	$mh \times HR_{(i,j)}$
Death of toxic humans	$Ht_{(i,j)} \to Ht_{(i,j)} - 1$ $Hd_{(i,j)} \to Hd_{(i,j)} + 1$	$(mt/tP) \times Ht_{(i,j)}$
Recovery of toxic humans	$Ht_{(i,j)} \to Ht_{(i,j)} - 1$ $HR_{(i,j)} \to HR_{(i,j)} + 1$	$((1 - mt)/tP) \times Ht_{(i,j)}$

Table 9: Event type, effects on the subpopulations and transition rates for the developmental model. The coefficients are mh: human mortality coefficient; VP: human viremic period; mh: human mortality coefficient; IIP: intrinsic incubation period; rP: remission period; tP: toxic period; rar: recovery after remission probability; mt: mortality probability for toxic patients. The values of the coefficients are available in Table 1.

Table 10 presents the values of the different coefficients involved in the events: egg hatching, pupation, adult emergence and gonotrophic cycles. The values are taken from Ref. [30] and are discussed in Ref. [8].

vii. Mortality coefficients

Egg mortality. The mortality coefficient of eggs is $me = 0.01$ 1/day, independent of temperature in the range 278 K $\leq T \leq$ 303 K [49].

Larval mortality. The value of α_0 (associated to the carrying capacity of a single breeding site) is $\alpha_0 = 1.5$, and was assigned by fitting the model to observed values of immatures in the cemeteries of Buenos Aires [8]. The temperature dependent larval death coefficient is approximated by $ml = 0.01 + 0.9725 \exp(-(T-278)/2.7035)$ and it is valid in the range 278 K $\leq T \leq$ 303 K [50–52].

Pupal mortality. The intrinsic mortality of a pupa has been considered as $mp = 0.01 +$

Develop. Cycle (4)	$R_D(T)$	R_D(298 K)	ΔH_A	ΔH_H	$T_{1/2}$
Egg hatching	elr	0.24	10798	100000	14184
Larval develop.	lpr	0.2088	26018	55990	304.6
Pupal Develop.	par	0.384	14931	-472379	148
Gonotrophic c. ($A1$)	cycle1	0.216	15725	1756481	447.2
Gonotrophic c. ($A2$)	cycle2	0.372	15725	1756481	447.2

Table 10: Coefficients for the enzymatic model of maturation [Eq. (4)]. R_D is measured in day^{-1}, enthalpies are measured in (cal / mol) and the temperature T is measured in absolute (Kelvin) degrees.

$0.9725exp(-(T-278)/2.7035)$ [50–52]. Besides the daily mortality in the pupal stage, there is an additional mortality contribution associated to the emergence of the adults. We considered a mortality of 17% of the pupae at this event, which is added to the mortality rate of pupae. Hence, the emergence factor is $ef = 0.83$ [53].

Adult mortality. Adult mortality coefficient is $ma = 0.091$/day and it is considered independent of temperature in the range 278 K $\leq T \leq$ 303 K [2, 50, 54].

viii. Fecundity and oviposition coefficient

Females lay a number of eggs that is roughly proportional to their body weight (46.5 eggs/mg) [55, 56]. Considering that the mean weight of a three-day-old female is 1.35 mg [2], we estimated the average number of eggs laid in one oviposition as $egn = 63$.

The oviposition coefficient $ovr_{(i,j)}$ depends on breeding site density $\mathrm{BS}_{(i,j)}$ and it is defined as:

$$ovr_{(i,j)} = \begin{cases} \theta/tdep & \text{if} \quad \mathrm{BS}_{(i,j)} \leq 150 \\ 1/tdep & \text{if} \quad \mathrm{BS}_{(i,j)} > 150 \end{cases} \qquad (5)$$

where θ was chosen as $\theta = \mathrm{BS}_{(i,j)}/150$, a linear function of the density of breeding sites [9].

ix. Dispersal coefficient

We chose a diffusion-like coefficient of $diff = 830$ m^2/day which corresponds to a short dispersal, approximately a mean dispersal of 30 m in one day, in agreement with short dispersal experiments and field studies analyzed in detail in our previous article [9].

x. Mathematical description of the stochastic model

The evolution of the subpopulations is modeled by a state dependent Poisson process [41,57] where the probability of the state:

$$(E, L, P, A1, A2s, A2e, A2i, Fs, Fe, Fi, Hs, He, Hi, Hr, Ht, HR, Hd)_{(i,j)}$$

evolves in time following a Kolmogorov forward equation that can be constructed directly from the information collected in Tables 4 to 9 and in Eq. 3.

xi. Deterministic rates approximation for the density-dependent Markov process

Let X be an integer vector having as entries the populations under consideration, and $e_\alpha, \alpha = 1 \ldots \kappa$ the events at which the populations change by a fixed amount Δ_α in a Poisson process with density-dependent rates. Then, a theorem by Kurtz [57] allows us to rewrite the stochastic process as:

$$X(t) = X(0) + \sum_{\alpha=1}^{\kappa} \Delta_\alpha Y\left(\int_0^t \omega_\alpha(X(s))ds\right) \qquad (6)$$

where $\omega_\alpha(X(s)$ is the transition rate associated with the event α and $Y(x)$ is a random Poisson process of rate x.

The deterministic rates approximation to the stochastic process represented by Eq. (6) consists of the introduction of a deterministic approximation for the arguments of the Poisson variables $Y(x)$ in Eq. (6) [34,58]. The reasons for such a proposal is that the transition rates change at a slower rate than the populations. The number of each kind of

event is then approximated as independent Poisson processes with deterministic arguments satisfying a differential equation.

The probability of n_α events of type α having occurred after a time dt is approximated by a Poisson distribution with parameter λ_α. Hence, the probability of the population taking the value

$$X = X_0 + \sum_{\alpha=1}^{\kappa} \Delta_\alpha n_\alpha \tag{7}$$

at a time interval dt after being in the state X_0 is approximated by a product of independent Poisson distributions of the form

$$\text{Probability}(n_1 \dots n_\kappa, dt/X_0) = \prod_{\alpha=1}^{\kappa} P_\alpha(\lambda_\alpha) \tag{8}$$

and

$$P^\alpha_{n_1 \dots n_E}(\lambda_\alpha) = \exp(-\lambda_\alpha)\frac{\lambda_\alpha^{n_\alpha}}{n_\alpha!} \tag{9}$$

whenever $X = X_0 + \sum_{\alpha=1}^{\kappa} \Delta_\alpha n_\alpha$ has no negative entries and

$$\begin{aligned} P^\alpha_{n_1 \dots n_E}(\lambda_\alpha) &= \exp(-\lambda_\alpha) \sum_{i=n_\alpha}^{\infty} \frac{\lambda_\alpha^i}{i!} \\ &= 1 - \exp(-\lambda_\alpha) \sum_{i=0}^{n_\alpha - 1} \frac{\lambda_\alpha^i}{i!} \end{aligned} \tag{10}$$

if $\{n_i\}$ makes a component in X zero (see Ref. [34])

Finally,

$$d\lambda_\alpha/dt = < \omega_\alpha(X) > \tag{11}$$

where the averages are taken self-consistently with the proposed distribution ($\lambda_\alpha(0) = 0$).

The use of the Poisson approximation represents a substantial saving of computer time compared to direct (Monte Carlo) implementations of the stochastic process.

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