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A simple qualitative multi-scale corona model (a report)

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Abstract In this document I propose an over-simplified, qualitative model for the evolution of infections in a random world. I do not claim this to be a valid quantitative predictive model – in particular, neglecting geographical, societal and financial facts that better models will have to consider – rather the model is quite minimalistic. Nevertheless, in this simple world one can test the effect of some parameters like (i) infection probabilities, (ii) interaction/travel probabilites, (iii) community/family sizes, as well as the effect of serious measures – and their duration – taken agains the pandemic, such as (1) cutting down on long distance travels (flights), (2) immobilizing the population, (3) reducing the infection probability, or combinations thereof. Note that the results are qualitative and from an over-simplified model and thus should not be over-interpreted, but might be taken a source of inspiration for more quantitative models.

Keywords COVID-19 · corona · Monte-Carlo · random walk

1 Introduction

The unprecedented situation in 2020, when the corona (COVID-19) pandemic started, made me remember (after a while) what I learned during my master/diploma thesis about modeling of populations and reaction-diffusion systems.

While I was still traveling in early 2020, from early March on I stopped, I reduced my mobility, and joined everyone in the shock we went. At first, I though that the best I could do was following the guidelines to stay home and reduce contacts and continue online doing my teaching and research job. Unlike the many people who do the more serious services to the community, like nurses, doctors, as well as super-market personnel, to name only a few, I thought I cannot do more. However, then I remembered that I can model, and maybe can help on that end, as this document maybe inspires others, or helps further, better research, or even helps us to understand how to better fight the present problem.

This video [1] I saw just after I had written most of this document and the model-script. It explains very nicely what such a model does and how to set it up – much better than I do in the following. But it also addresses where the limits of simple models are. The same caution as described in [1] has to be also considered here, and in many other articles [2].

I do *not* claim that the model proposed here is better than others, but it is maybe simpler than many (more realistic, more detailed) approaches, out of which I only want to refer to one paper [3], and references therein. *Remark by the author: I did not have the time (yet) for a more thorough literature study but rather wanted to make public the ideas I spent the last days with. Those ideas were based on my older work during my MSc/Diploma thesis – and I just was using those methods – and did not see the point to cite my older papers here either.*

In the following, I first describe the model before I provide a series of simulation results, varying some

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[1] [1] `https://youtu.be/gxAaO2rsdIs`

[2] [2] https://medium.com/data-for-science/epidemic-modeling-101-or-why-your-covid19-exponential-fits-are-wrong-97aa50c55f8

[3] [3] N. Harding, R. Nigmatullin, M. Prokopenko (2018) Thermodynamic efficiency of contagions: a statistical mechanical analysis of the SIS epidemic model. Interface Focus 8: 20180036. http://dx.doi.org/10.1098/rsfs.2018.0036

of the model parameters, with the goal to understand their effect on the spreading of an infection. The second set of simulations simulates the effect of the duration of special measures taken on how the disease spreads thereafter.

2 Stochastic Model

The model described in the following is very similar to traditional Monte-Carlo simulations of population dynamics or reaction diffusion models. It concerns a population that randomly take actions like moving, interacting, or getting infected.

2.1 Model description

The population model is realized with a population of N random walkers in a square model world of area $A = L \times L$, and periodic boundaries to avoid wall effects. The population is initially placed completely randomly throughout the model world, all people are healthy; then, very few are infected with probability $p_0 \ll 1$ and the infections start to transfer to others (as described below). A few of the infected population will die, with probability, p_m, but the remainder will heal, and become immune, i.e., not to be infected anymore. Unless specified otherwise, the reference case parameters in table 1 are used, for a simulation of $t = 100$ days, with time-step $\Delta t = 1/5$ days.

no.	parameter	symbol	unit	value
0.1	population	N	10k	500000
0.2	interaction cell size	c	km	20
1.1	system size	L	km	2000
1.2	av. step size	$x_s/4$	km	5
1.3	av. flight-distance	$x_f = \frac{L}{8}$	-	250
2.1	time-step	Δt	days	1/5
2.2	probability for flight	p_f	1/day	0.002
3.1	initial infections prob.	p_0	-	2×10^{-5}
3.2	infection rate	r_i	-	0.004
3.3	days of sickness	t_s	days	20
3.4	infectious period	η_i	-	0.6
3.5	reduced mobility	x_q/x_s	–	0.2
3.6	mortality (during t_s)	p_m	$1/t_s$	0.1

Table 1 Summary of the parameters and their values as used for the refernce numerical solutions, sorted in groups of (0.) population, (1.) geometrical, motion, (2.) probabilities, and (3.) infection-related.

2.1.1 Motion model – diffusion and flights

Normal motion of the population is realized in both directions by a random walk with steps $x \in [-1/2 : 1/2]x_s$, with $x_s = 20$, and thus average step-size $\Delta x = \langle|x|\rangle = x_s/4 = 5$, every time-step Δt, ignoring social, geographical and topological details of travel in the real world. *Theory:* This leads to short distance diffusive behavior, $\langle r^2(t)\rangle = 2Dt$, with coefficient of diffusion, $D = \frac{\Delta x^2}{2\Delta t}$, so that the typical diffusive spread distance is, after 100 days, $r_d(100) = \sqrt{\langle r^2(100)\rangle} = \sqrt{125 \times 100} \approx 110$. [4]

On top of the normal motion, long-distance travel is implemented with a probability $p_f = 0.002$, assuming that 0.2% of the population are traveling farther than others, per day, with typical travel distance, $\Delta x_f = x_f/4 = (L/5)/4 = 100$, which allows the infection to create new infection centra beyond the diffusive spreading.

In addition, the infected population, after an incubation period (see next subsection), is assumed to move five times slower, with step size, x_q, whereas dead are not moving anyway.

2.1.2 Infection model

Given a person is infected, each infection is assumed to last for a time t_s (think of 20 days), with mortality probability, p_m, during the full period, $1/t_s$, for which fatalities are tested every time-step with a random number $r < p_m \Delta t/t_s$. Next, we split the sick time, $t_s = t_0 + t_i$, into an infectious/dangerous period of duration $t_i = \eta_i t_f$, after the incubation time $t_0 = (1-\eta_i)t_f$, with the infectuous period, η_i. Here, the fact that not every infection is taking the same course, or is equally heavy, is only indirectly taken into account by the stochastic nature of the Monte-Carlo model used here, in particular, some people will infect others, some will not, as described next.

During the infectious period, t_i, every healthy person that gets into contact with an infected one can also become infected with probability p_i, per day, tested every time step as random infection, $r < p_i \Delta t$.

But how to define a contact? In this multi-scale model for infection spreading, I propose to distribute all the population in areas with a given cell-size c/L, and area $a = (c/L)^2$, populated by $N_c = aN$, in average. The rate of infection is now increased due to multiple infected, N_i, within one cell, so that $r < N_i p_i \Delta t$ is tested every time-step, for every healthy in the cell.

In this simple version of the model, there is only one cell-size representing communities (like families or companies), or social events or centres; in future there should be a hierarchy of cell-sizes, as well as overlapping cells. For the sake of simplicity, only one cell level,

[4] [TODO: Not verified, but looking at the plots it seems plausible.]

without any overlaps is considered – but note that the population can easily move from one cell to another, diffusively, as described above.

2.2 Simulation results

The infection model is realized with a population of $N = 500,000$ random walkers in a square world of area $A = L \times L$, with $L = 2000$, and periodic boundaries to avoid wall effects. Unless specified otherwise, the reference case parameters are used, for a simulation of $t = 100$ days, with time-step $\Delta t = 1/5$ days.

The initial infection probability is $p_0 = 2 \times 10^{-5}$, which results in $N_i = p_0 N \approx 10$ centres, in the cases shown, from which the infection spreads.

2.2.1 Parameter studies

In Fig. 1, different infection rates, r_i, are compared. In the top panels, the red curves display the fraction of infected, while in the bottom panels, the numbers are shown in two different zoom-in versions. The magenta curve represents the infectious (infected after the incubation period of $\eta t_s = 8$ days, with $(1 - \eta) = 0.4$ and $t_s = 20$ days), just delayed relative to the total infections. The green lines represent the healed (and immunized) population, while the black lines represent the fatalities, with assumed mortality probability, $p_m = 0.1$.

For the smallest $r_i = 0.001$, the initial infections reduce and vanish after about 60 days, while twice this rate, $r_i = 0.002$, results in a steady, small fraction of infections. For the $r_i = 0.004$ (reference) case, the infections grow exponentially until the end of the observation period, day 100, while for $r_i = 0.008$, the double exponential growth rate starts to flatten, since the local population was already completely infected.

In Fig. 2, different flight probabilities, p_f, are compared, showing the wider and faster spreading, and the creation of new infection centres due to long distance travel.

In Fig. 3, different step sizes, x_s, are compared, showing the increased spreading with increasing step-sizes, i.e., with increased mobility. In different words, the infection can be kept local quite well by seriously reducing the mobility.

In Fig. 4, different cell sizes, c, are compared, showing the effect of the size of the local communities, or compartments, on the spreading of infections. Smaller cells clearly reduce the spreading of the infections seriously, due to the lower probability to encounter an infected person, i.e., compartmentalizing and avoiding bigger crowds surely helps.

2.2.2 Taking actions – different duration of action

In this subsection we compare the effect of different actions: (1) reducing the long-distance flights, (2) reducing short distance mobility, (3) reducing the infection probability, or applying combinations of those measures.

In Fig. 5, the consequences of a reduction of probability for flights, p_f, by a factor 1/100, are shown, or starting at day 30, and ending 15, 30, or 45 days later, together with the reference case (run1), where no action is taken. Reducing the flight-distance has rather little effect on the overall infections, only a few new centres are avoided after day 30, while the existing ones are not much affected by reduction of travel distance, neither has the duration a big effect.

In Fig. 6, the consequences of a reduction of the mobility, step-size, x_s, by a factor 1/10, are shown, starting at day 30, and ending 15, 30, or 45 days later, together with the reference case, where no action is taken. Also here there is rather little effect on the overall population, but the centres of infection are a bit more localized.

In Fig. 7, the consequences of a reduction of the infection propbability, p_i, by a factor 1/2, are shown, starting at day 30, and ending 15, 30, or 45 days later, together with the reference case, where no action is taken. This has a strong effect on the overall infections, but not much on the growth after the action is stopped and the probabilities go back to the original value at the end of action. The action just delays the trend, but has no effect after giving up the action.

Actions 2 and 3 combined, i.e., reducing travel-distances and infection probability, see Fig. 9, shows a visible reduction in infections only for the longest application period of 45 days, even leading to a dip (which action 3 alone did hardly achieve) and thus also a considerable further delay of future growth.

Combining all actions 1, 2, and 3, see Fig. 9, almost shows the same effect as actions 2 and 3, but again only for the duration of the action, with the major influence of action 3, the reduction of infection probability, while the others have a comparatively lesser effect, when taken as late as day 30.

The effect of taking actions earlier is dicussed next.

2.2.3 Effect of actions – time of action

The effect of taking actions 1,2,3 not only at day 30, but earlier (or later), for the same duration of 30 days, is displayed in Fig. 10. Clearly, the earlier an action is taken, the better – on the short term, but it can be

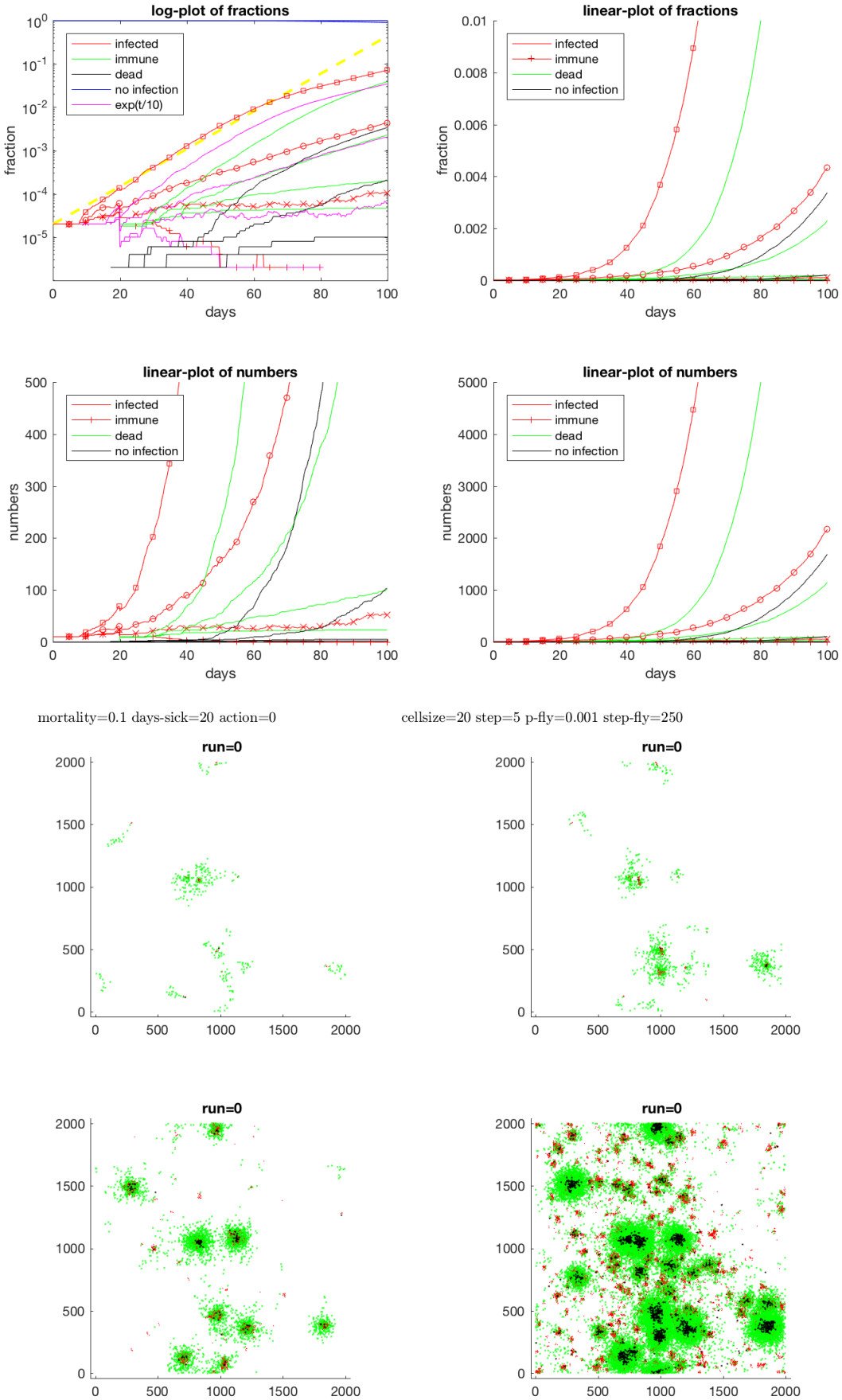

Fig. 1 Effect of different infection probabilities, $p_i = 0.001$ (+), 0.002 (x), 0.004 (o), 0.008 (□). (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

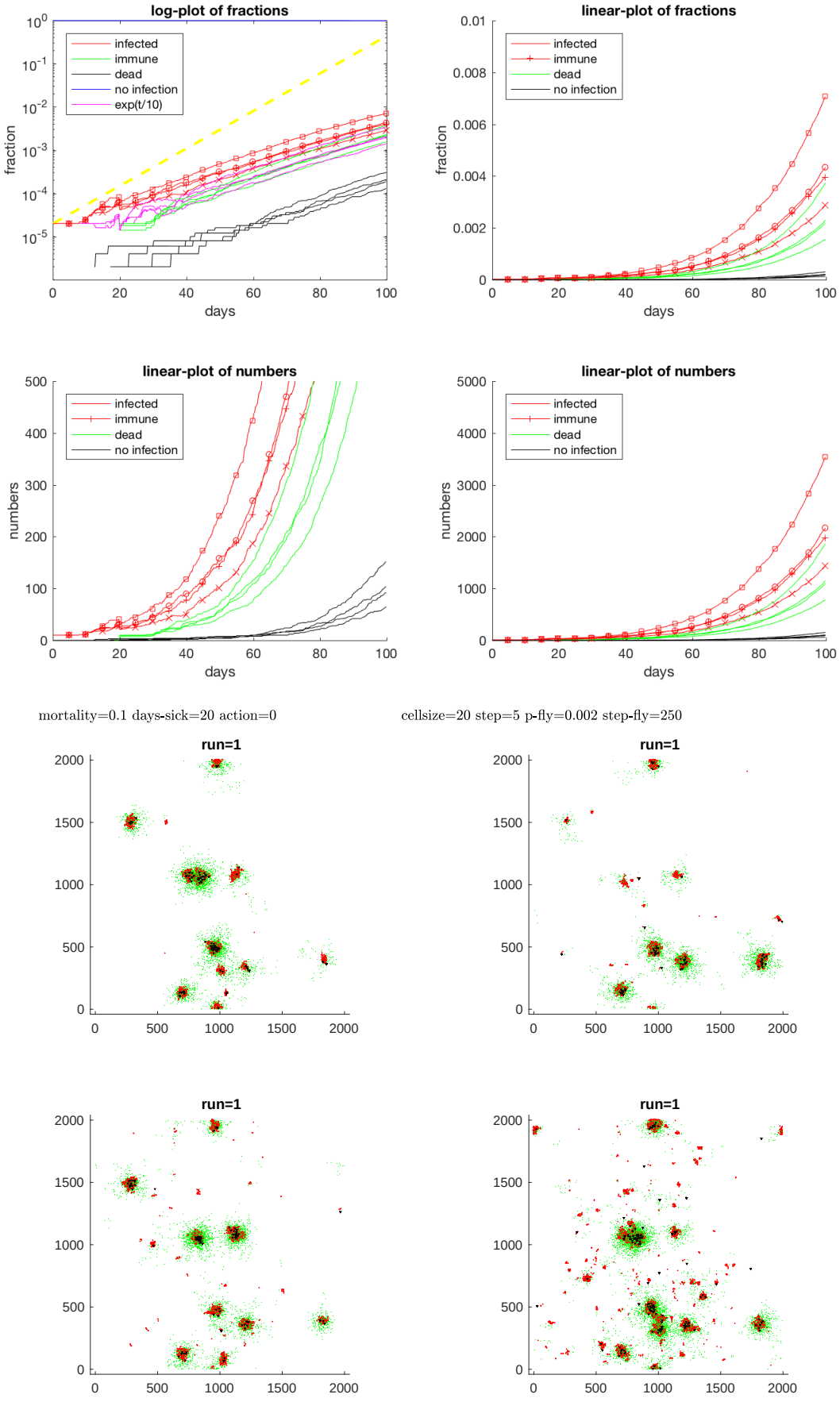

Fig. 2 Effect of different flight probabilities, $p_f = 0.00025$ (+), 0.0005 (x), 0.001 (o), 0.002 (□). (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

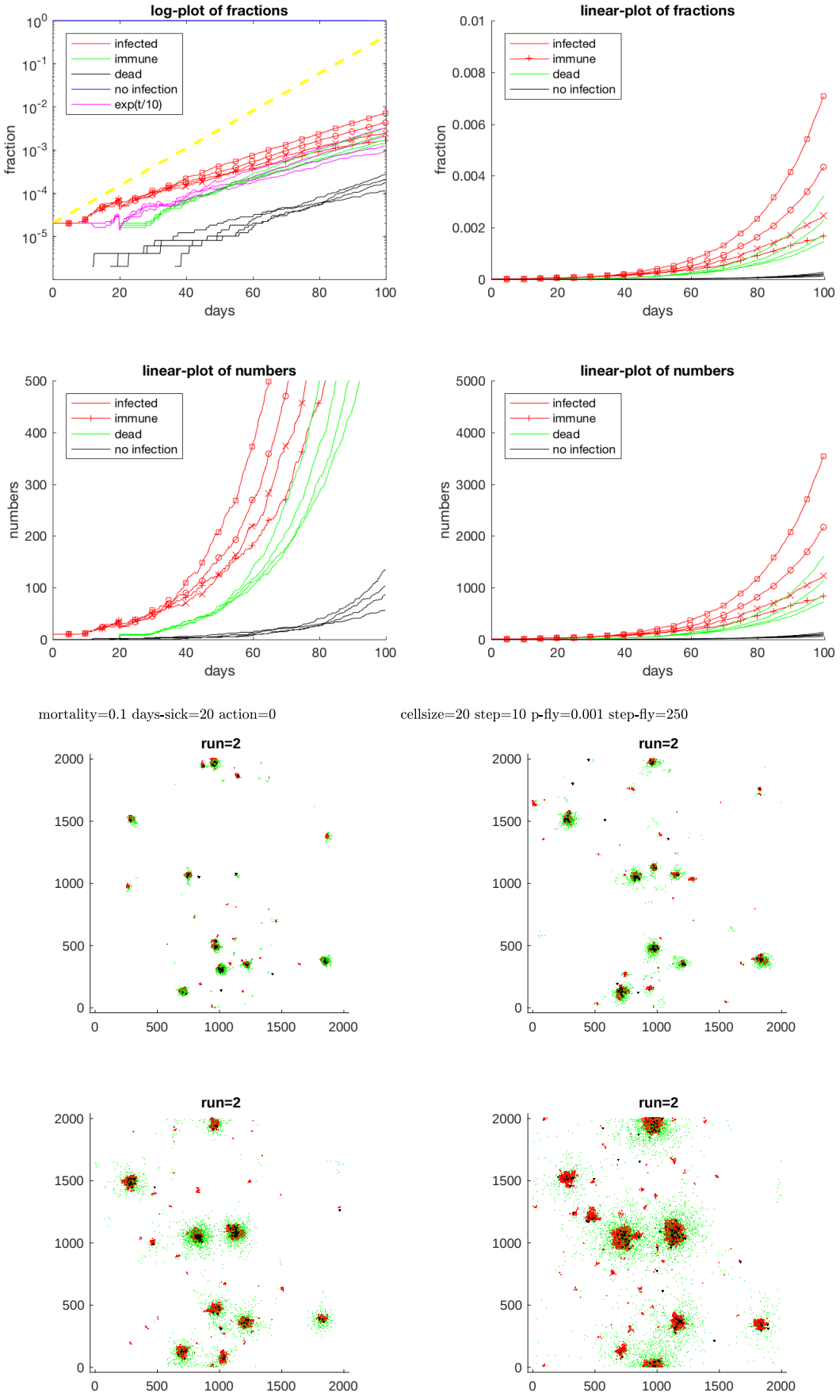

Fig. 3 Effect of different step sizes, $x_s = 5$ (+), 10 (x), 20 (o), 40 (□). (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

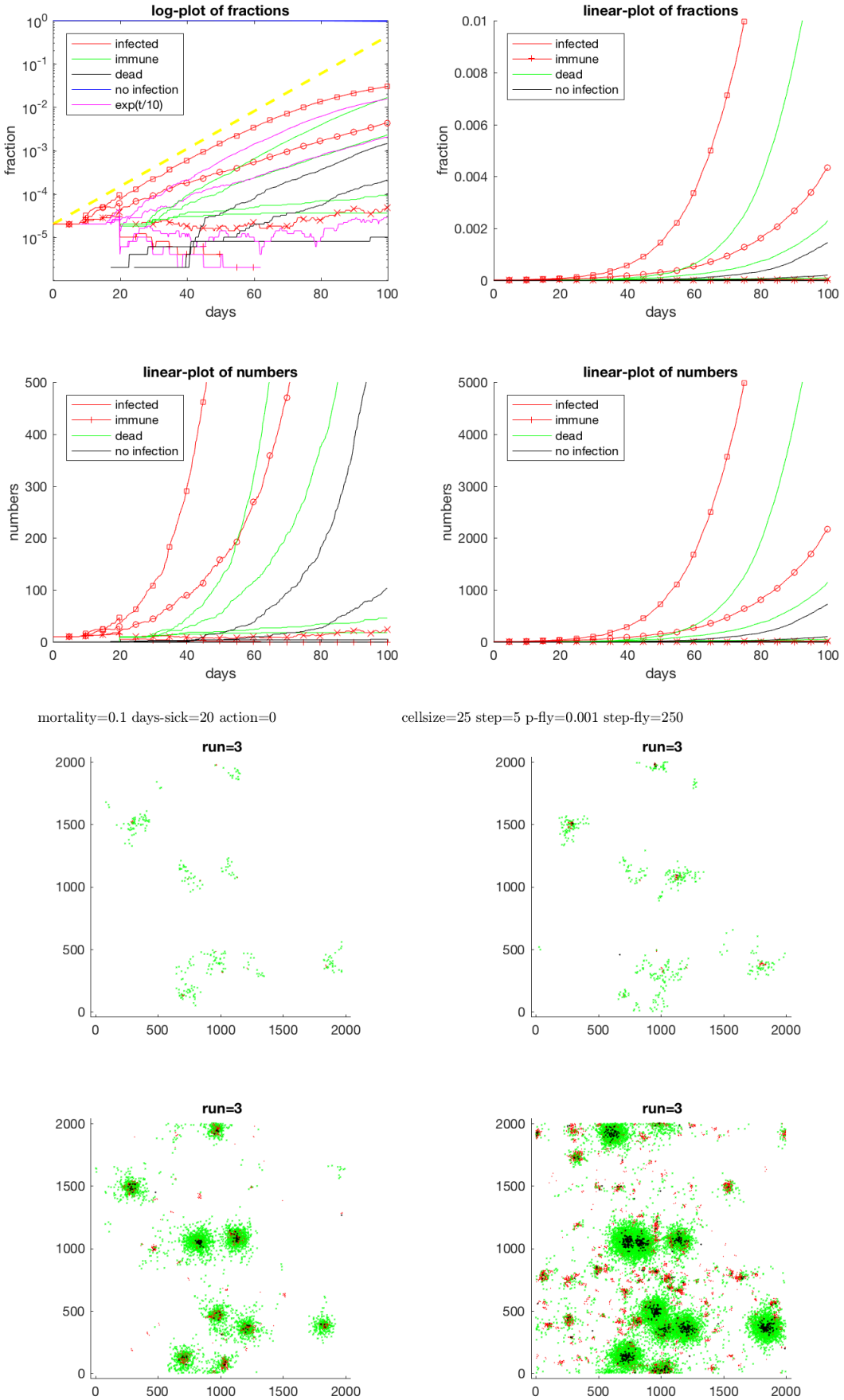

Fig. 4 Effect of different cell sizes, $c = 10$ (+), 15 (x), 20 (o), 25 (□). (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

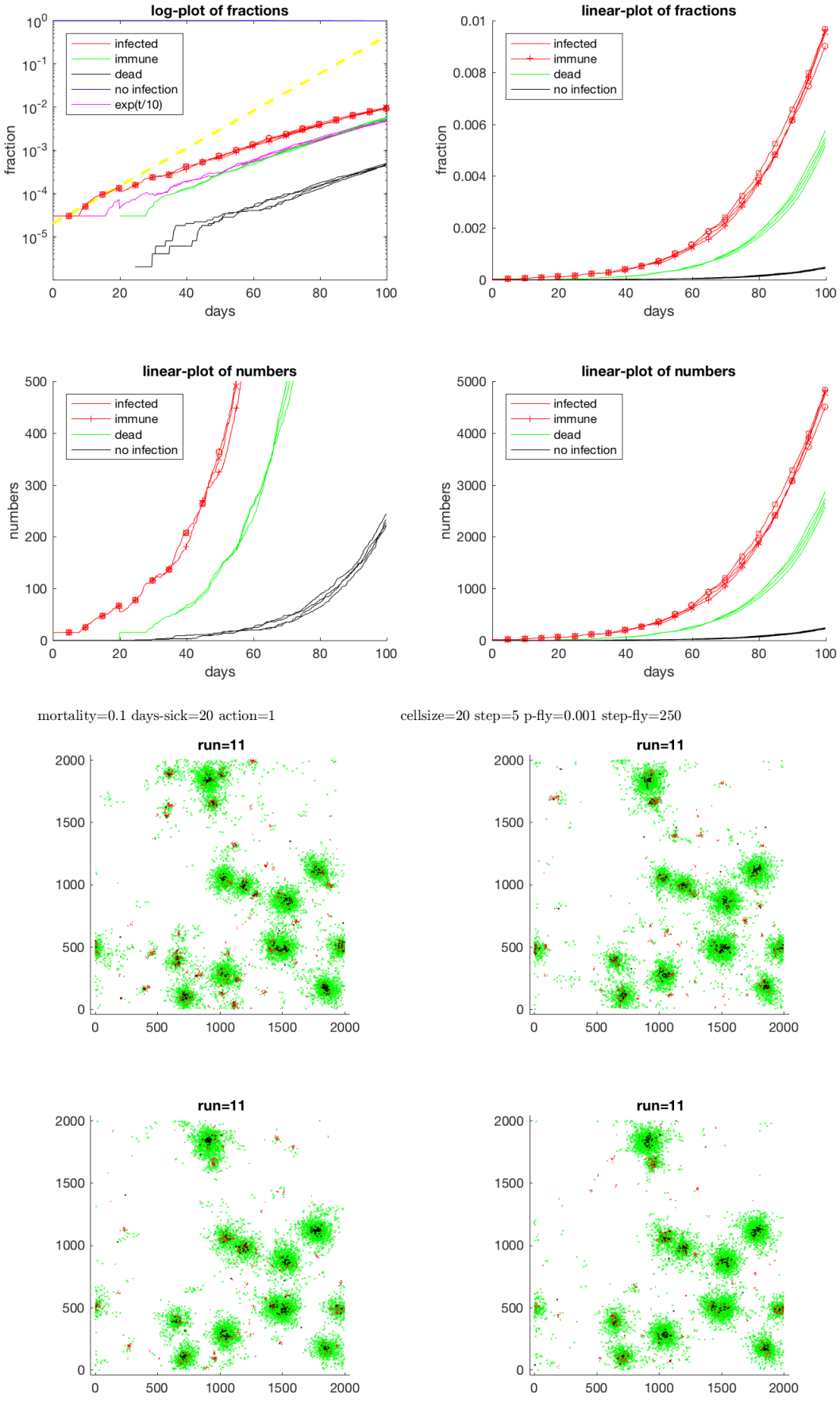

Fig. 5 Effect of action 1: reducing the frequency of flights to 1/100, for zero (+), 15 (x), 30 (o), 45 (□) days, starting from day 30. (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

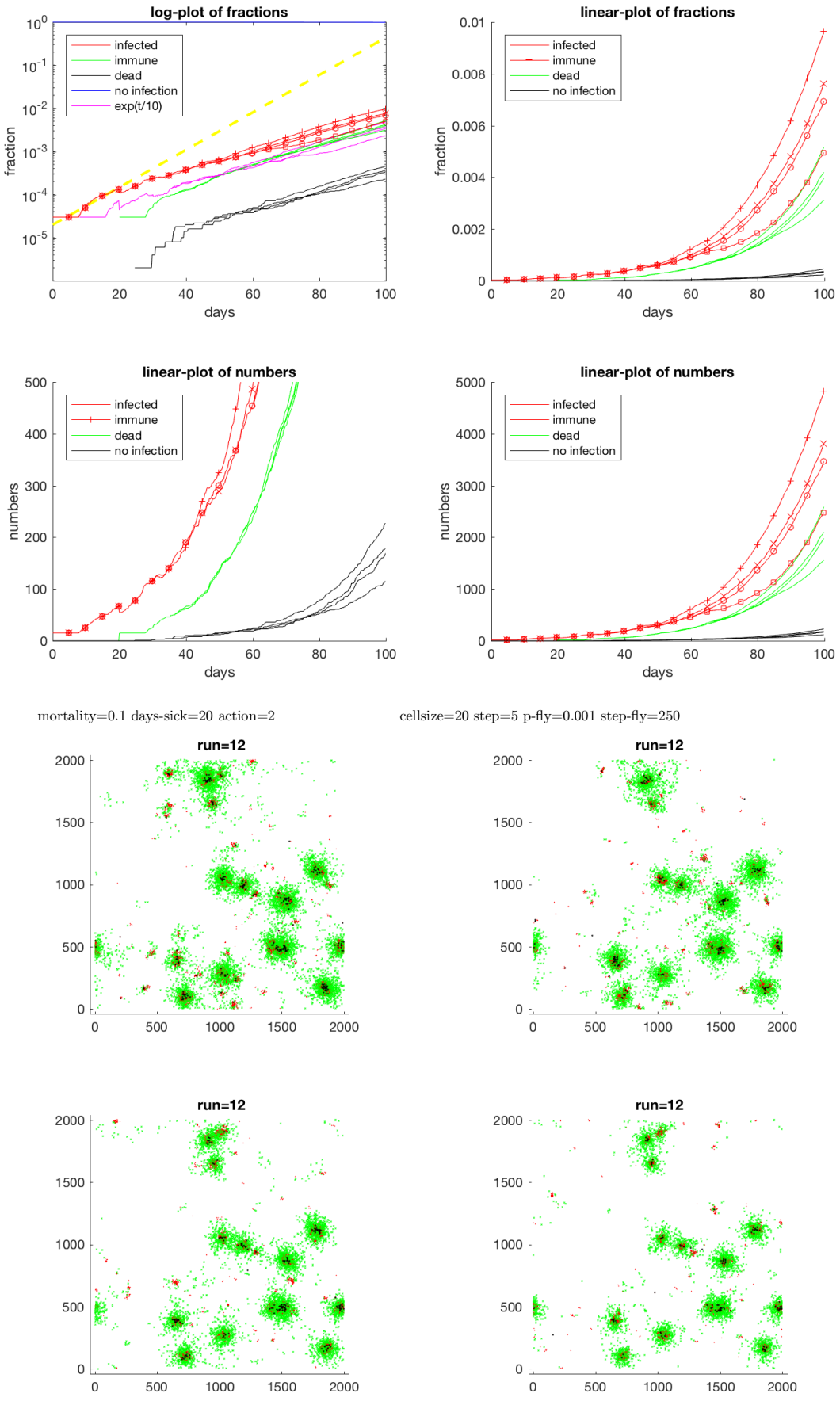

Fig. 6 Effect of action 2: reducing the mobility to 1/10, for zero (+), 15 (x), 30 (o), 45 (□) days, starting from day 30. (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

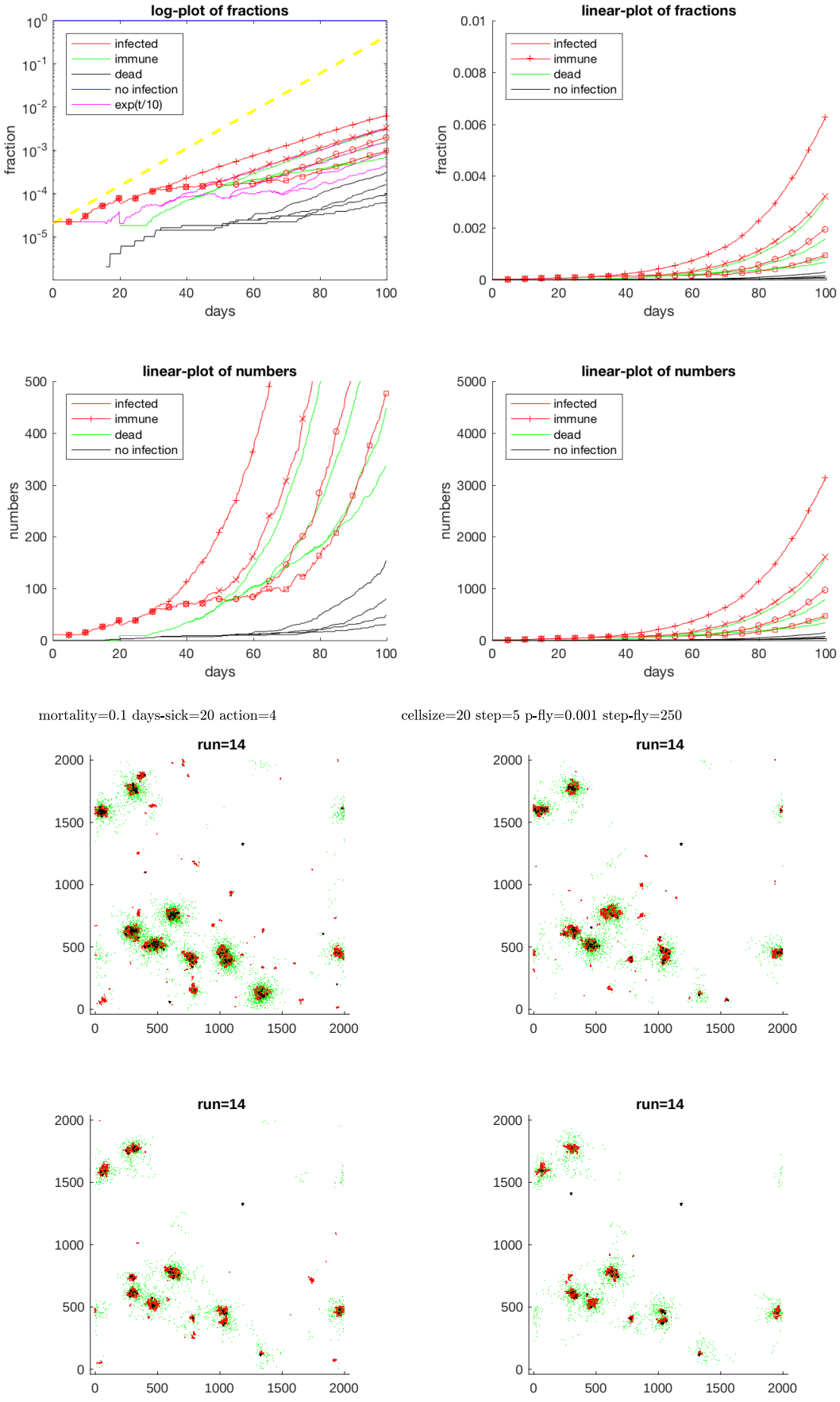

Fig. 7 Effect of action 3: reducing the infection rate to 1/2, for zero (+), 15 (x), 30 (o), 45 (□) days, starting from day 30. (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

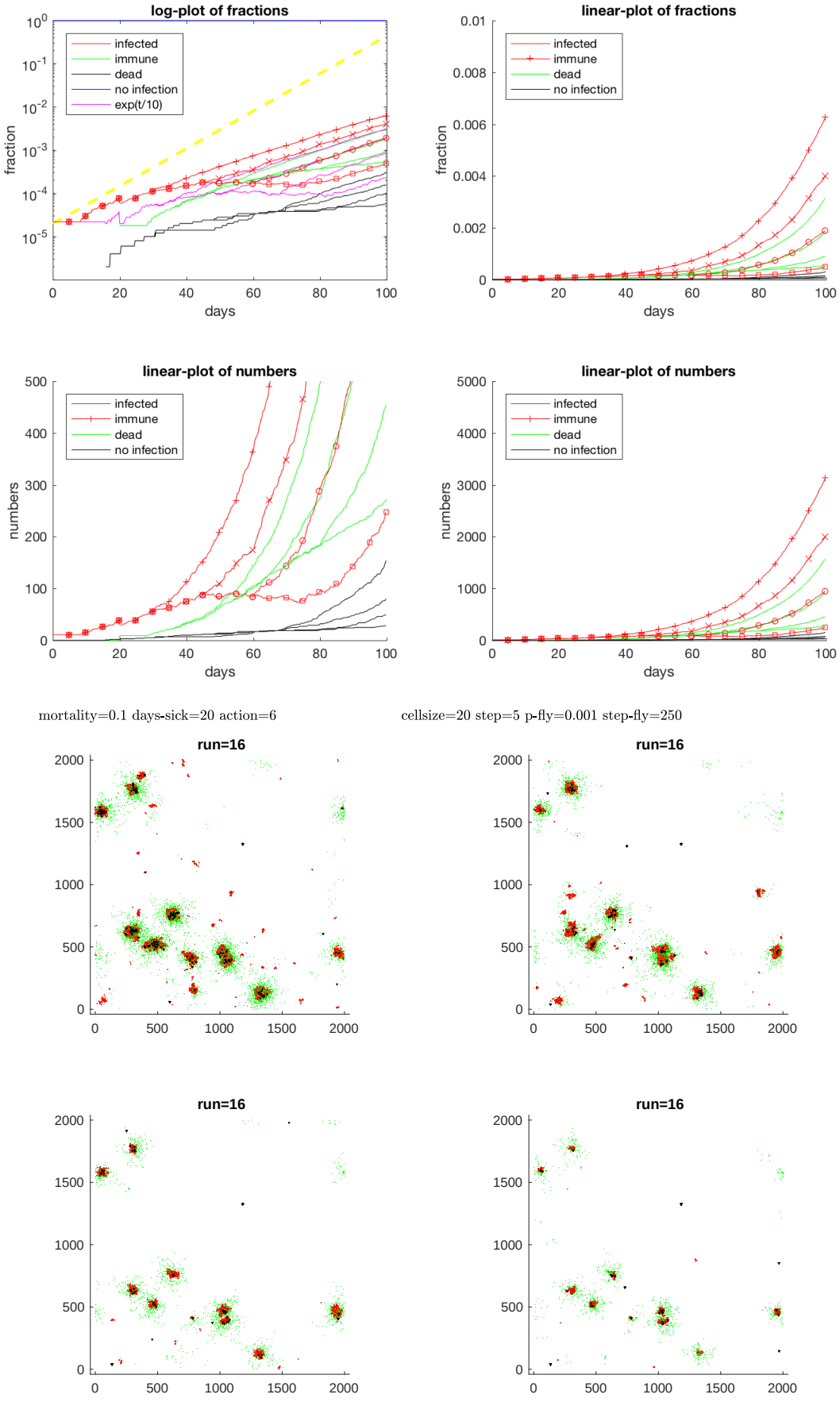

Fig. 8 (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

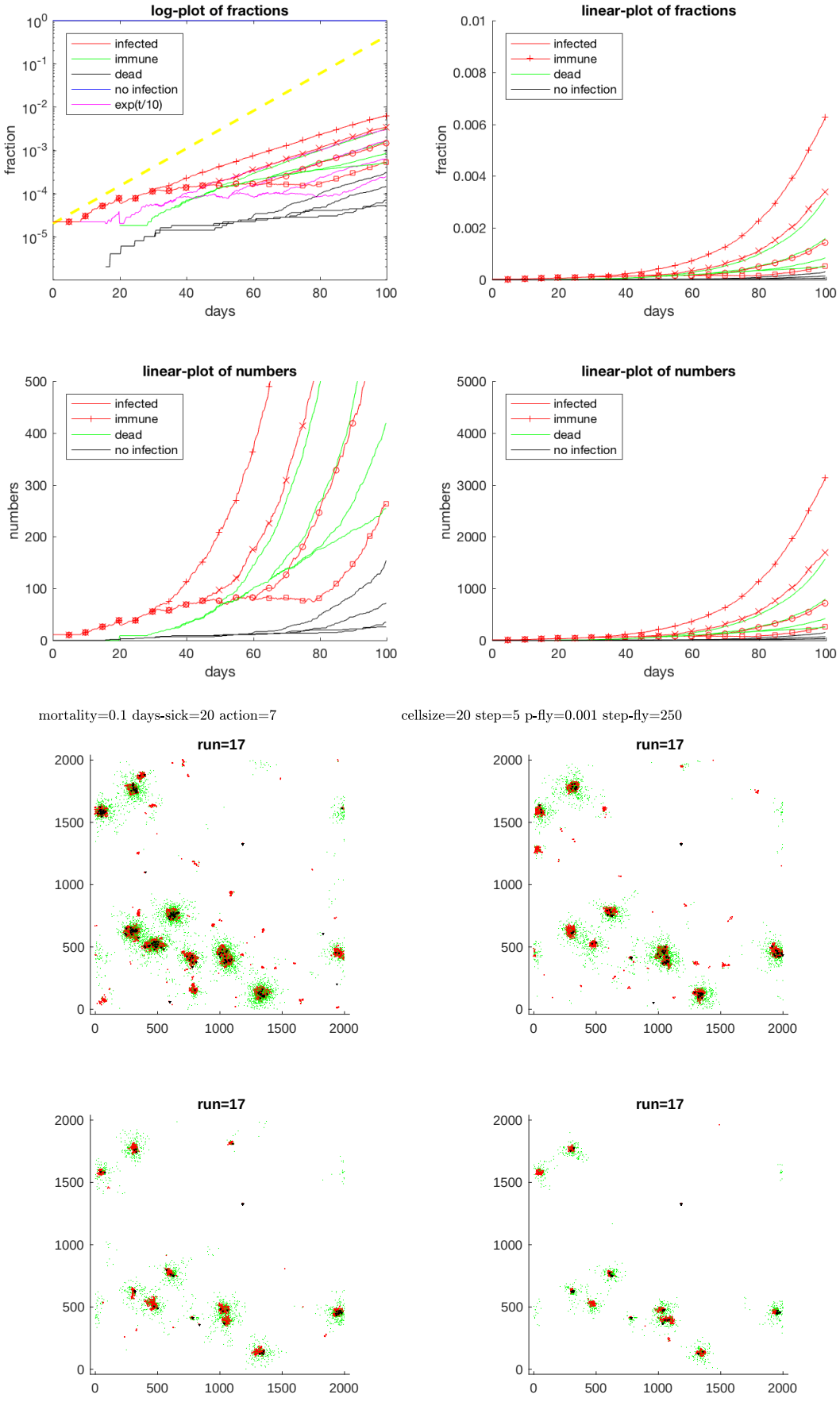

Fig. 9 (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).

worse on the long-term, if the action is stopped too early, since not sustainable for longer.

3 Summary and Conclusion

An over-simplified, qualitative model for the evolution of infections in a random world was proposed, neglecting geographical, societal and financial facts that better models will have to consider. Nevertheless, in this simple world one can study and understand the effect of some of the parameters like: (i) infection probabilities, (ii) interaction/travel probabilites, (iii) community/family sizes, from which one learns that the infection probability has a most direct effect on the growth rate.

The model is multi-scale in several aspects, one of which being short- or long-distance mobility. Reducing either of those mobilities (by reducing the random step-sizes) has the expected influence on the spreading, keeping the centers of infection smaller or avoiding too many far distant new centers from which infections in so far unaffected areas spread. Reducing long-distance mobility, like flights, however, has to be done as early as possible, since the effect is weaker if the action is applied too late.

The second multi-scale aspect is the interaction range. In contrast to more detailed models, there is no interaction between single people, i.e., nothing like a repulsive or attractive force in the present model. However, some type of social interaction is taken into account in form of compartments or cells, whithin which people do interact directly, while they interact with other cells only interact if they move there. A single of this cell-sizes was used, but one might think of multiple levels like, families, neighborhoods, social/cultural events, sport-events, or even cities, or countries, with different scales and/or patterns, on which regular, or isolated high-frequency interaction could be implemented in the model.

The present model has no quarantine mechanism, however, the infected are assumed to be infectious and less mobile after a certain incubation time.

Short distance mo as well as the effect of serious measures – and their duration – taken agains the pandemic, such as (1) cutting down on long distance travels (flights), (2) immobilizing the population, (3) reducing the infection probability, or combinations thereof. Note that the results are qualitative and from an over-simplified model and thus should not be over-interpreted, but might be taken a source of inspiration for more quantitative models.

A more careful size- and ensemble-analysis of the model has not been performed by the author to date, due to lack of time, but this is work in progress. Also more theoretical treatment, going beyond the trivial diffusion equation shown above, with the goal to provide better continuum/deterministic models for infection evolutions might be a goal of future research, besides adding more realistic features, or dding complexities of the real world like topology and/or network-connectivities, however this goes beyond the scope of the present simple study.

Even though endless improvements of the model can be thought of – only few are mentioned – I think there is some value in simplicity also. Getting faster predictions of future trends, increasing the size of populations and samples, as well as using a simple model for machine learning approaches could all help to better understand and fight infections. The present model is so simple and fast that everyone can run a decent population on a decent computer, and play with parameters and improvements themselves. I have just started using the ideas that led to this model also for education of students and hope that many of them will profit from such an example. The dangers of simplicity must not be ignored, however; therefore, the results of a simple, minimalist model also should not be over-interpreted.

Acknowledgements With this I want to thank the people I worked with, and learned from, about stochastic modeling of reaction-diffusion systems, back in 1989-1994, in particular Alexander Blumen, Horst Schnörer, Vladimir Kuzovkov and Igor Sokolov. Also thanks to those colleagues who sent me the links and references I give here, while I did not have the time for a more ccareful literature study.

Appendix

This pdf is available at:
www2.msm.ctw.utwente.nl/sluding/PAPERS/cor1_010420.pdf
as well as the matlab script that creates the figures:
www2.msm.ctw.utwente.nl/sluding/PAPERS/cor1_010420.m
both in the version of 01.04.2020.
My apologies that I did this in matlab without thinking it through beforehand, an open script language would have been a better choice.
Technical remark: using the cell-interaction model speeds up the code a lot as compared to more advanced molecular dynamics like, or even social/traffic inspired interaction models, so that quite big numbers N can be done easily.
Statistics remark: In the figures plotted below, for every run, the random number generator was using always the same random seed $s = 101$, as an arbitrary choice, to keep the simulations comparable against each other in cases where one parameter is changed, or an action is taken. A more detailed analysis with many different ensembles, different seeds, different system sizes, etc., has to be added.

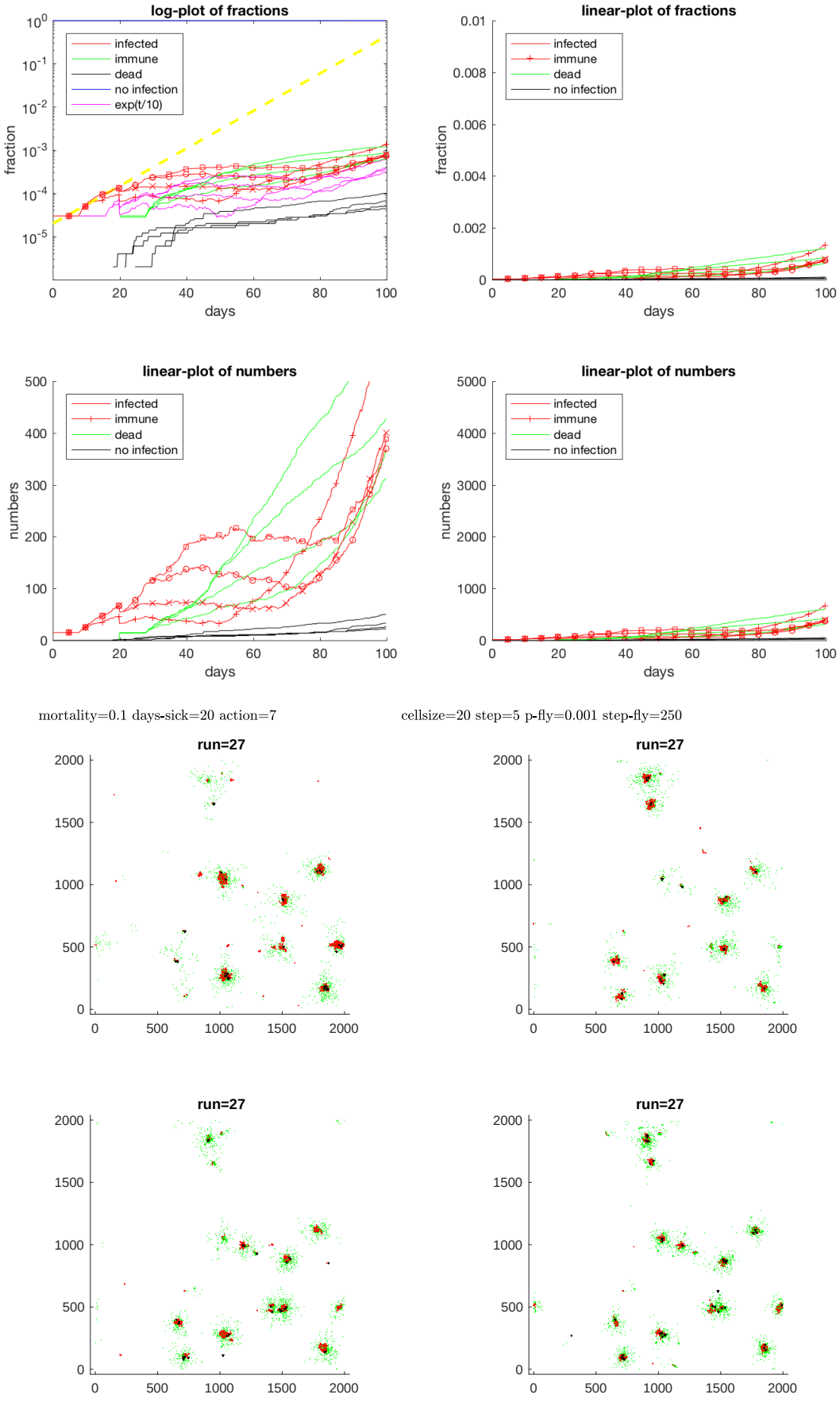

Fig. 10 (Top) Logarithmic and linear plots of infected (red), healed/immunized (green) and dead (black) fractions of the population, as well as numbers (to be read as k=1000). (Bottom) Day 100 population plots of infected (red), healed/immunized (green) and dead (black).