AGENT-BASED AND POPULATION-BASED SIMULATION: A COMPARATIVE CASE STUDY FOR EPIDEMICS

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ABSTRACT

This paper reports a comparative evaluation of population-based simulation in comparison to agent-based simulation for different numbers of agents. Population-based simulation, such as for example in the classical approaches to predator-prey modelling and modelling of epidemics, has computational advantages over agent-based modelling with larger numbers of agents. Therefore the latter approaches can be considered useful only when the results are expected to deviate from the results of population-based simulation, and are considered more realistic. However, there is sometimes also a silent assumption that for larger numbers of agents, agent-based simulations approximate population-based simulations, which would indicate that agent-based simulation just can be replaced by population-based simulation. The paper evaluates this assumption by a detailed comparative case study in epidemics.

1. INTRODUCTION

The classical approaches to simulation of processes in which groups of larger numbers of agents are involved are population-based: a number of groups are distinguished (populations) and each of these populations is represented by a numerical variable indicating their number or density (within a given area) at a certain time point. The simulation model takes the form of a system of difference or differential equations expressing temporal relationships for the dynamics of these variables. Wellknown classical examples of such population-based models are systems of difference or differential equations for predator-prey dynamics (e.g., Lotka, 1924; Volterra, 1926, 1931; Maynard Smith, 1974; Burghes and Borrie, 1981) and the dynamics of epidemics (e.g., Ross, 1916; Kermack and McKendrick, 1927; Burghes and Borrie, 1981; Anderson and May, 1992; Ellner and Guckenheimer, 2006). Such models can be studied by simulation and by using analysis techniques from mathematics and dynamical systems theory.

From the more recently developed agent system area it is often taken as a presupposition that simulations based on individual agents are a more natural or faithful way of modellling, and thus will provide better results (e.g., Davidsson, Gasser, Logan, and Takadama, 2005; Sichman and Antunes, 2006; Antunes and Takadama, 2007). Although for larger numbers of agents such agent-based modelling approaches are more expensive computationally than population-based modelling approaches, such a presupposition may provide a justification of preferring their use over population-based modelling approaches, in spite of the computational disadvantages. In other words agent-based approaches with larger numbers of agents are justified because the results are expected to deviate from the results of population-based simulation, and are considered more realistic.

However, in contrast there is another silent assumption sometimes made, namely that for larger numbers of agents (in the limit), agent-based simulations approximate population-based simulations. This would indicate that agent-based simulation just can be replaced by population-based simulation, which would weaken the justification for agent-based simulation discussed above. In this paper, by a case study in epidemics, these considerations are explored in more detail. Comparative simulation experiments have been conducted based on different simulation models, both agent-based (for different numbers of agents), and population-based. The results are analysed and related to the presupposition and assumption discussed above.

2. THE DOMAIN OF EPIDEMICS

Microbes such as viruses, bacteria, fungi and parasites, may have disturbing effects when they enter the human body. Not seldom humans suffer from such infections and in the mean time propagate them to each other. Examples of types of infectious diseases are influenza, chlamydia, HIV, hepatitis, tuberculosis, and many others. The battle against such infections takes place both at the biological level in the body, and at the behavioural and social level. At the behavioural and social level, humans sometimes try to adapt their interaction behaviour to prevent propagation of infections from one human to another one. This paper focuses on the propagation of infections in populations, in relation to the interaction behaviour, in particular the frequency and intensity of contacts that individuals have in the population.

Agents within a population can be in different states: susceptible (not infected yet), infective, or recovered (immune and not infectious). When an agent who is infective, has contact with another, susceptible agent then there is a chance that the other agent will also be infected due to this contact. This chance depends on the intensity of the contact. The overall chance that a susceptible agent is infected, also depends on the number of contacts with

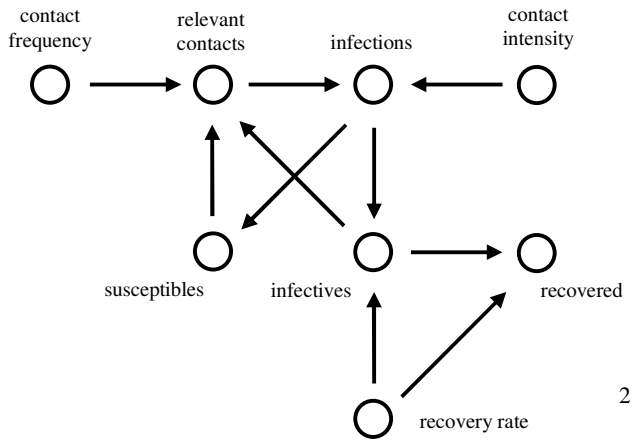
infective agents. An example of a possible pattern, for example, for an easy transmittable infection such as influenza, is that the propagation goes so fast that only in a few weeks time almost the whole population is infected. In such a case the term epidemic is used to indicate the spreading of the infection over the population. For other types of infections, for example HIV or chlamydia, more intensive contacts (which usually occur less frequently) are needed for transmission, and therefore propagation may proceed slower.

An important question, especially for the more harmful infections, in a society is whether by measures at the behavioural and social level, it is possible to keep the number of infected persons in a population limited. And if so, how far should such measures go? It is clear that by avoiding any contact between agents, propagation can be stopped, but that is often not a realistic option. On the other hand, if there are still some contacts between agents, will at the end the infection not be spread (perhaps by very slow propagation) over the whole population? Such questions are addressed in this paper by two types of models: population-based models (with populations of susceptible, infectives and recovered agents, respectively) and agent-based models.

3. A POPULATION-BASED MODEL

This section describes the population-based model. The analysis of epidemics has a long history, going back, for example, to (Ross, 1916; Kermack and McKendrick, 1927). More recent presentations can be found in (Anderson and May, 1992) and (Ellner and Guckenheimer, 2006, Ch. 6, pp. 183-215). First of all, a distinction is made between the population of *susceptibles* vs. the population of *infectives*, the latter of which are infectious for the former. A third population consists of those that already were infected, but have *recovered* and therefore are immune and not infectious anymore, based on a *recovery rate* indicating the fraction of infectives that recovers per day. Furthermore the *frequency* of *contacts* (per day, the time unit chosen) plays a main role; the chance that in a contact *infection transmission* occurs depends on the *contact intensity*.

Figure 1 Dynamic relationships in the model



The populations can be described by their sizes, but often they are characterised by their densities: size divided by area. As the area is considered fixed, the sizes (numbers) will be used to characterize the populations. The dynamic relationships between these concepts are depicted in Figure 1. For a mathematical formalisation usually the contact frequency times the contact intensity divided by the overall size of the populations together is combined in one parameter, called the *contact rate*. Thus the following variables and parameters are used.

size of the population of susceptible individuals	S
size of the population of infective individuals	I
size of the population of recovered individuals	R
size of all populations together	N
contact rate	β
recovery rate	γ
threshold	ρ

Here $\beta = ContactFrequency*ContactIntensity/N$. Note that for given values of contact frequency and contact intensity, this parameter β depends on the overall population size. The dynamics of these concepts involve temporal relationships, which are analysed in more detail below. Each susceptible person has (per day) a number of contacts indicated by *ContactFrequency*. From these contacts a fraction $I(t)/N$ is with infective individuals, where N is the size of the three populations together (assumed fixed). Therefore the number of relevant contacts per day is: $RelevantContacts(t) = ContactFrequency*S(t)*I(t)/N$. Moreover, in a fraction of the contacts the infection is transmitted. This fraction is indicated by *ContactIntensity*; therefore the number of new infections per day is: $Infections(t) = ContactIntensity*RelevantContacts(t) = ContactIntensity* ContactFrequency*S(t)*I(t)/N = \beta*S(t)*I(t)$. Given the number of infections $Infections(t)$ per time unit, in a time interval between t and $t+\Delta t$ the number of (new) infections is $Infections(t)*\Delta t$. This is subtracted from the susceptible population, and added to the infective population. Furthermore, γ indicates the fraction of the infective population per day that becomes recovered (and not infective anymore): over the interval between t and $t+\Delta t$ a number of $\gamma*I(t)*\Delta t$ is taken from the infective population and added to the recovered population. Therefore the following temporal relationships are used.

$$S(t+\Delta t) = S(t) - Infections(t)*\Delta t$$
$$I(t+\Delta t) = I(t) + (Infections(t) - \gamma* I(t)) *\Delta t$$
$$R(t+\Delta t) = R(t) + \gamma* I(t) *\Delta t$$

Note that by these relationships the sum of the three populations always remains the same: what adds to the recovered population subtracts from the infective population, and what subtracts from the susceptible population adds to the infective population. In the more usual notation, by replacing $Infections(t)$ the equations can be written as:

$S(t+\Delta t) = S(t) - \beta^*S(t)^*I(t)^*\Delta t$
$I(t+\Delta t) = I(t) + (\beta^*S(t)^*I(t) - \gamma^* I(t))^*\Delta t$
$R(t+\Delta t) = R(t) + \gamma^* I(t)^*\Delta t$

In differential equation form they are represented in the following manner; for example, see also in (Kermack and McKendrick, 1927; Burghes and Borrie, 1981; Anderson and May, 1992; Ellner and Guckenheimer, 2006):

$\frac{dS(t)}{dt} = - \beta^*S(t)^*I(t)$ $\frac{dI(t)}{dt} = \beta^*S(t)^*I(t) - \gamma^* I(t)$ $\frac{dR(t)}{dt} = \gamma^* I(t)$

Note again, that the parameter β in principle depends on the overall population size. This means that to do experiments with different overall population sizes, different values for β may have to be used. Based on these equations the following analysis has been made:

(a) Threshold for increase/decrease of infective population

Increase and decrease of the size of the population of infectives are characterised by

$\frac{dI(t)}{dt} \geq 0 \Leftrightarrow \beta^*S(t)^*I(t) - \gamma^* I(t) \geq 0$ $\frac{dI(t)}{dt} \leq 0 \Leftrightarrow \beta^*S(t)^*I(t) - \gamma^* I(t) \leq 0$

This can be characterised by the size of the population of susceptibles as follows with $\rho = \gamma/\beta$:

$I(t)$ increasing $\Leftrightarrow S(t) \geq \rho$ $I(t)$ decreasing $\Leftrightarrow S(t) \leq \rho$

This shows that the usual pattern is that the size of the population of infectives will increase until the size of the population of susceptibles has become lower than the threshold ρ, after which it will decrease. In particular, when the initial size $S(0)$ is already less than this threshold ρ, then the number of infectives will decrease right from the start. This is called the *epidemic threshold law* with *threshold* ρ.

(b) Equilibria

An equilibrium occurs if and only if $\frac{dS(t)}{dt} = \frac{dI(t)}{dt} = \frac{dR(t)}{dt} = 0$, which is characterised by $\beta^*S(t)^*I(t) = \beta^*S(t)^*I(t) - \gamma^* I(t) = \gamma^* I(t) = 0$. This is equivalent to $I(t) = 0$. Notice that by itself this does not put any constraint on $S(t)$ or $R(t)$. Equilibria may depend on initial values as well. However, taken together with a) in the usual pattern in an equilibrium state $S(t)$ will have become below ρ. So, when ρ is rather small (e.g., individuals remain infective for a long time, or contact intensity is high), the number of individuals that never become infected will also be small, or even zero. These observations are illustrated by simulations in the next section.

(c) Relation between equilibria and initial values

From the set of differential equations, in particular the first and third one, it can be derived that

$\frac{dS(t)}{dt} = - \beta^*S(t)^*I(t) = -\frac{1}{\rho} S(t) \frac{dR(t)}{dt}$ or $\frac{dR(t)}{dt} = -\frac{\rho}{S(t)} \frac{dS(t)}{dt}$. By integration, using the natural logarithm, it follows for all t it holds $R(t) = C - \rho \log(S(t))$ with C a constant. Assuming $R(0) = 0$, it holds $C = \rho \log(S(0))$. Therefore

$R(t) = \rho \log(S(0)) - \rho \log(S(t)) = \rho \log(S(0)/S(t))$.

Equivalent forms are: $e^{R(t)/\rho} = S(0)/S(t)$ $S(t) = S(0) e^{-R(t)/\rho}$ $S(0) = S(t) e^{R(t)/\rho}$. Now, according to (a) for an equilibrium occurs if and only if $I(t) = 0$, which is equivalent to $S(t) + R(t) = N$. Filled in the above formula this obtains: $S(0) = (N - R(t)) e^{R(t)/\rho}$ or $S(0) = S(t) e^{(N-S(t))/\rho}$. This shows a relation between the population sizes in an equilibrium state and the initial values; note that $S(0) = N - I(0)$. Note that conversely, each of these relations also implies that $S(t) + R(t) = N$, hence $I(0) = 0$ and an equilibrium state occurs. So, these relations provide if and only if criteria for an equilibrium to occur.

4. Population-Based Simulations

A number of population-based simulation experiments have been performed using standrad simulation software. In Figures 2 and 3 results are shown of one of them, with time scale in days. In the first simulation shown in Figure 2 the whole population gets infected; it used the following parameter settings:

N	100
ContactFrequency	0.8
ContactIntensity	0.5
β	0.004
γ	0.05
ρ	12.5

Initially the size of the infective population is 1. Given the analysis above, in this case it may be expected that the size of the population of susceptibl-es will become below *12.5*. Note that in this and next figures the scales on the vertical axis differ.

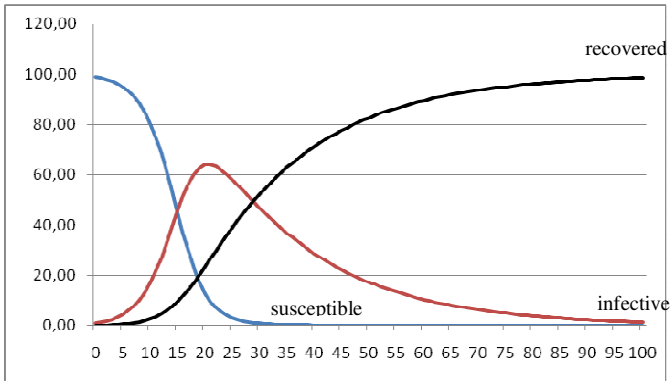


Figure 2 Pattern in which the whole population gets infected

The size of the susceptibles decreases to zero, while the size of the infective population increases until day 20 and decreases after this day. The size of the recovered population shows a logistic growth pattern with the whole

population of *100* as limit. Notice that the maximal size of the population of infectives is taken at the time point that the size of the susceptibles population is around *12.5*, which is the value of the threshold ρ.

In the second simulation, shown in Figure 4, only part of the population gets infected; parameter settings were:

N	*100*
ContactFrequency	*0.6*
ContactIntensity	*0.2*
β	*0.0012*
γ	*0.1*
ρ	*83.3*

Here initially the size of the infective population is 10. Apparently here the contact frequency and intensity were low enough to let the infection die out: around 50% of the population is never infected. The logistic growth pattern of the (infected and) recovered population has its limit around 50. Nevertheless, the individuals still did not bring their contacts down to zero, or even close to zero.

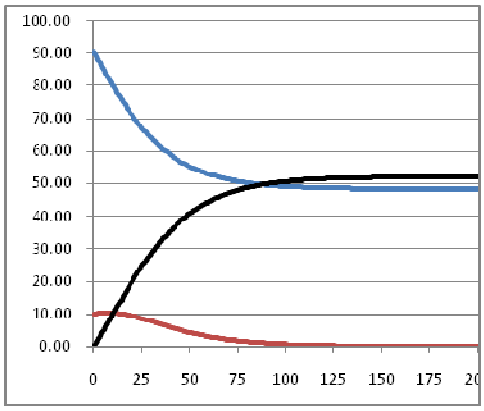


Figure 3 Only part gets infected, starting with 10

This shows that by relatively small differences in behaviour at the individual level, relatively big differences at the collective level can be realised. Notice that for this case the maximal size of the population of infectives is at the time point that the size of the susceptibles population is around *83*, which is the value of the thershold ρ.

5. AN AGENT-BASED MODEL

To obtain a model at the level of individual agents, *N* distinct agents and *L* distinct locations are introduced. At every time point each agent is at some location, at random. Contacts between agents are modelled as being at the same location. By taking the number *L* of locations (numbered by *1, 2, …, L*) lower or higher, a specific contact frequency is modelled. Each agent is in precisely one of three infection states (susceptible, infective, recovered).

The number of locations has a relationship with the contact frequency in the following manner. If *L* is the number of locations, and *N* the number of agents, then the average number of agents at one location is *N/L*, so the average number of contacts of one agent at such a location is *N/L – 1*. This is equal to the contact frequency. Therefore $ContactFrequency = N/L - 1$ gives the relation between number of locations and contact frequency. To be able to compare this model at the agent level to the model at the population level, it is convenient to have contact frequency as a basic parameter. To this end, the relation between the number of locations and contact frequency shown above is used in an inverse manner to determine the number of locations for a given value of the contact frequency: $L = N/(ContactFrequency + 1)$.

For a given contact frequency, this *L* is taken as a bound for the number of locations: the locations are indexed by the natural numbers *k* with $1 \leq k \leq L$. At each time point locations of the agents are determined at random, using this bound *L* by taking at random one of the natural numbers between *1* and *L*.

When a suceptible agent *A* is at a certain location, the probability that infection takes place depends on the contact intensity, but also on the number *k* of infective agents present at that location. Although in an agent-based model, contact intensity may be taken as depending on the agent or even on the pair of agents involved in a contact, for reasons of comparability with the population-based model the contact intensities are taken uniform: in any contact between any susceptible agent *A* and any infective agent *B*, the probability that *A* will be infected is *ContactIntensity*. Given this assumption, the probability that agent *A* will not be infected by a specific infective agent at the same location is $1 - ContactIntensity$. Assuming independence of these probabilities, the probability that a will not be infected by any of the infective agents present at that location is $(1 - ContactIntensity)^k$. Therefore the probability that *A* will be infected at that location (at that time point) is $1 - (1 - ContactIntensity)^k$.

The following relationships describe the changes of the infection state of an agent *A*. Here *r1* and *r2* are two independent random numbers between *0* and *1:* fixed per time point, but refreshed at new time points. When a susceptible agent is at a location where one or more infective agents are present, the transmission of the infection at that time point has a probability given by the contact intensity. Moreover, for someone who is infective there is a probability of recovery given by the recovery rate. This is modelled by: $InfectionState(A, t+1) = infective$ if $InfectionState(A, t) = susceptible$ and there are *k* infective agents at the same location as *A* and $r1 < 1 - (1 - ContactIntensity)^k$ or $InfectionState(A, t) = infective$ and $r2 \geq RecoveryRate$. Moreover, $InfectionState(A, t+1) = recovered$ if $InfectionState(A, t) = infective$ and $r2 < RecoveryRate$ or $InfectionState(A, t) = recovered$. In all other cases $InfectionState(A, t+1) = susceptible$.

6. AGENT-BASED SIMULATIONS

Similar simulation experiments as the ones described above have been performed using the model at the level of the individuals. As this model is based on random choices,

the patterns can vary. In Figure 4 two example traces based on the following parameter settings are shown.

N	*10*
ContactFrequency	*0.8*
ContactIntensity	*0.5*
RecoveryRate	*0.05*
$\beta = 0.04$ $\gamma = 0.05$ $\rho = 1.25$	

The infection states are indicated by numbers *0* (susceptible), *1* (infective) and *2* (recovered).

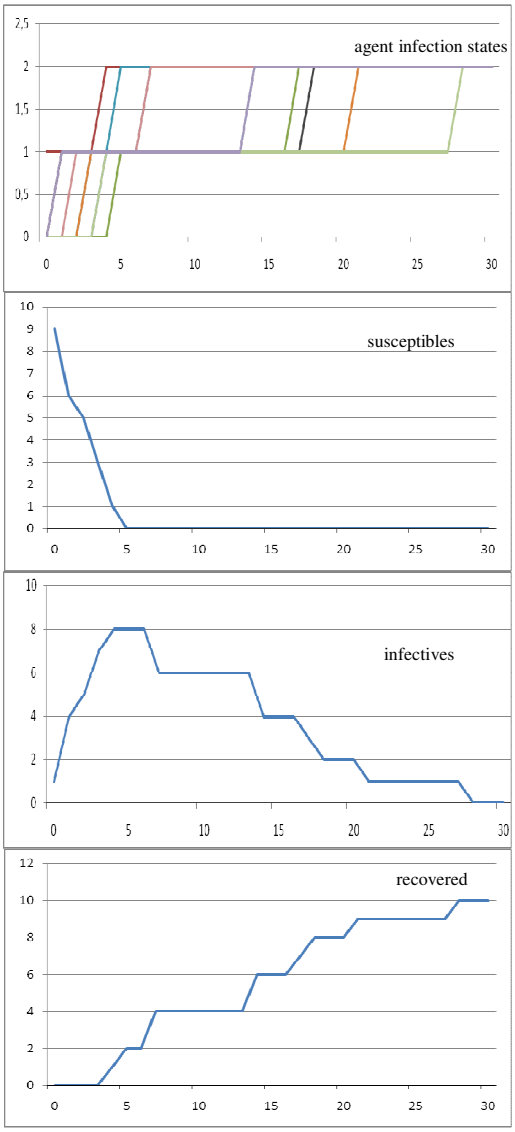


Figure 4 Pattern in which the whole population gets infected

These settings correspond to the ones for the trace shown in Figure 2. Initially one agent is infective. In the upper graph it is shown how individuals get infected one after the other. At the start A2 is infective. Already from the second day on, A4, A7 and A10 get infected. After that A8, A5 and A6 follow, and finally A1, A9 and A3 get infected. In the second graph in Figure 4 the aggregated number of susceptibles is shown, in the third graph the

N	*10*
ContactFrequency	*0.6*
ContactIntensity	*0.2*
RecoveryRate	*0.1*
$\beta = 0.012$ $\gamma = 0.1$ $\rho = 8.3$	

number of infectives, and in the lower graph the number of recovered individuals.

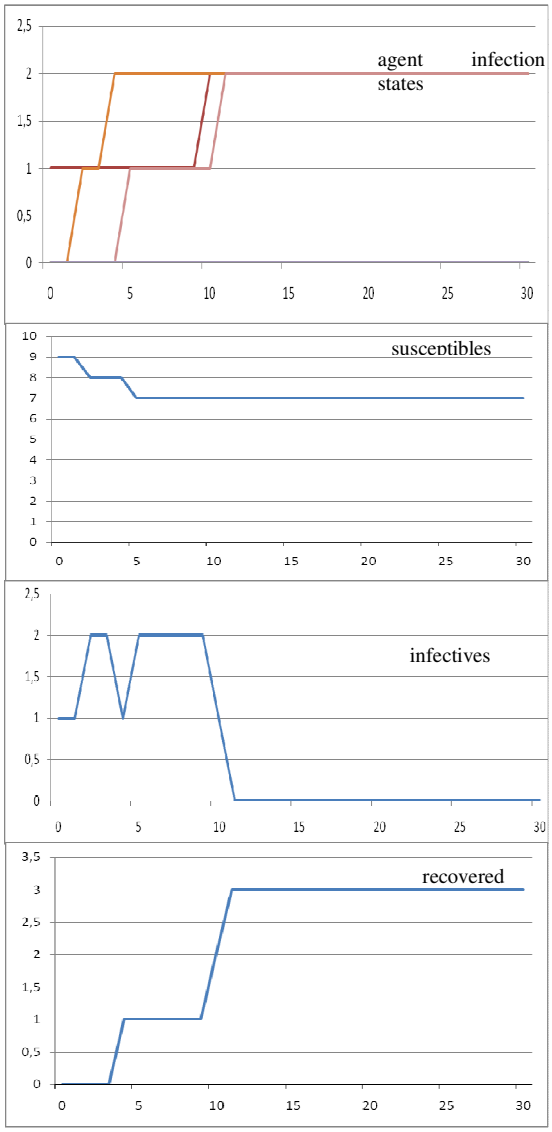


Figure 5 Pattern in which part of the population gets infected

The pattern is similar to the pattern shown in Figure 2. Note that also here the maximal number of infectives is reached at the time point that the number of susceptibles drops under ρ. Initially 1 agent is infective. In the upper graph it is shown how three individuals get infected. At the start A2 is infective. Soon A6 gets infected but recovers already in two days. Since A2 takes longer to recover, A8 is infected on day 5. After 6 days A8 recovers, and in the meantime also A2 recovered. No further

infections took place. The pattern is similar to the pattern shown in Figure 4. Note that also here the maximal number of infectives coincides with the number of susceptibles dropping below the threshold ρ.

From other simulations it was found out that this example trace is a bit exceptional for this setting. Most traces of the individual model show either only one or two infectives, after which the epidemic dies out, or (almost) all individuals become infected. See Figure 6 for an overview of 100, resp. 1000 experiments with the model for 10 agents. The average number of recovered agents for this sample is 5.71. Note that this means that the model at the collective level shows a kind of average pattern that for the model at the individual level for 10 agents almost never occurs.

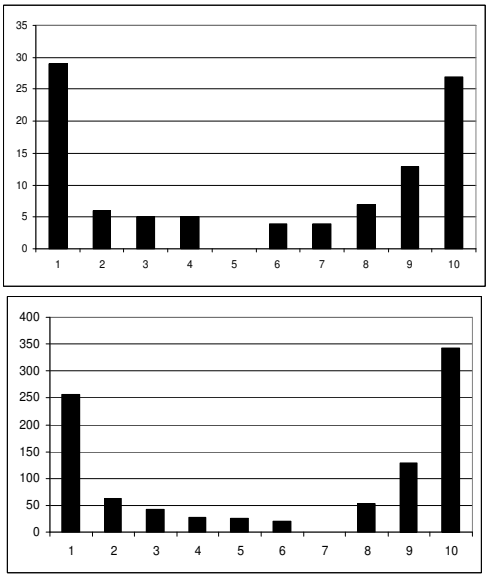


Figure 6 Numbers recovered for 100, resp. 1000 runs for 10 agents with 10% initially infected

Under similar experimental configurations simulations for larger population has been conducted through simulation software developed in the C++ language. Figure 7 shows the results of 1000 simulations conducted for both populations of 100 and 1000 agents carrying 10 percent initially infected. In these simulations the agent-based model shows a different pattern. Rather than an average pattern as for the case of 10 agents; see Figure 6, it shows single peak towards the higher number of recovered agents with an average of (approximately) 93 percent recovered agents in both cases. Variation in number of recovered agents for 1000 samples in case of population count 100 and 1000 was 31 and 9 percent respectively, which is much lower then 90 percent variation in all samples observed in population count 10. Moreover, the average on all simulations were also close to the peak that differs a lot from both the outcome of agent based model at low population as 10 agents; See Table 1, and the population-based model.

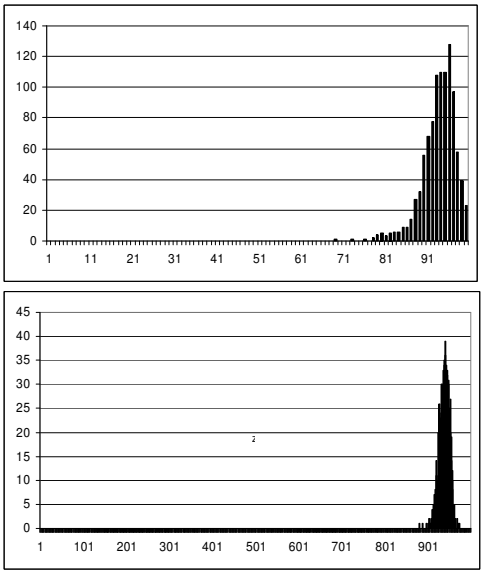


Figure 7 Numbers recovered for 1000 runs for 100, resp. 1000 agents with 10% initially infected.

Total Population	10	100	1000
Initial Susceptible	9	90	900
Initial Infected	1	10	100
Average Recovered	61.62	93.50	93.83
Min Recovered	10.00	69.00	88.20
Max Recovered	100.00	100.00	97.30
Variation in Samples	90.00	31.00	9.10

Table 1 Average Percentage of min, max and average recovered in the sample of 1000 simulations for 10, 100 and 1000 agents respectively with 10% initially infected.

In Fig. 8 the results for similar populations have been shown with 1% initially infected. In these simulations for a population of overall size 100 an average of 60.5% recovered and 99% variation in all samples was observed which is somewhat similar in behaviour as of population count 10; see Fig. 6, that also shows two peaks with nearly average pattern. But the population of overall size 1000 has shown a graph almost similar to an average of 92.98% and 8.40% variation in all samples as that has been seen for 10% initially infected for the same population count; see Fig. 7. To further investigate this behaviour change as seen in a population of overall size 100 with a change of percentage of initially infected, simulations were performed for 0.1% initially infected for a population count of 1000. These simulations have confirmed the graph change pattern observed in case of population count 100; see Fig. 9.

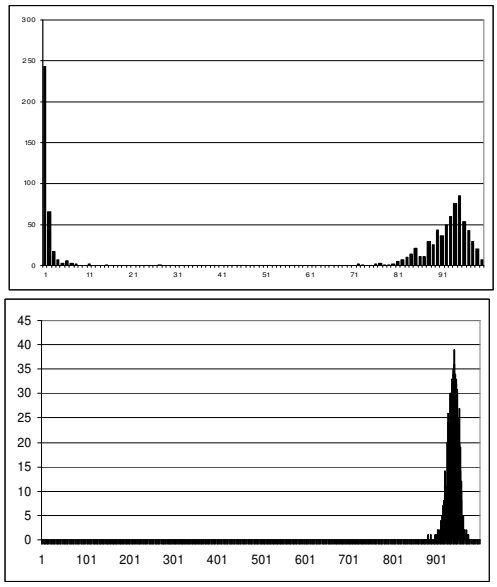


Figure 8 Numbers of recovered agents for a sample of 1000 simulations for 100 and 1000 agents respectively with 1 percent [1 and 10 agents respectively] initially infected.

From the above simulations it is evident that in agent-based simulations for epidemics the percentage of the initially infected population is not the factor to be taken same for similar experimental configuration for different population sizes but it is the number of initially infected agents that should be taken same; see also Table 2.

Table 2 Average Percentage of min, max and average recovered in the sample of 1000 simulations for 10, 100 and 1000 agents respectively with 1 agent initially infected.

Total Population	10	100	1000
Initial Susceptible	9	99	999
Initial Infected	1	1	1
Average Recovered	61.62	60.4	58.5
Min Recovered	10.00	1.00	0.10
Max Recovered	100.00	100.00	96.40
Variation in Samples	90.00	99.00	96.30

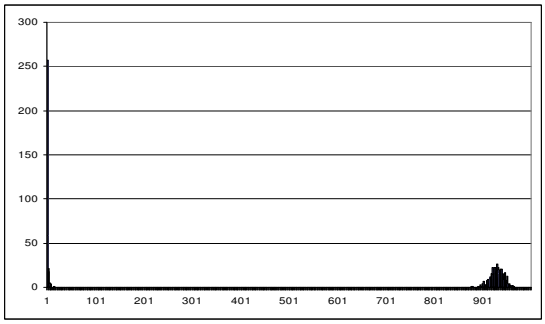


Figure 9 Numbers of recovered agents for a sample of 1000 simulations for 1000 agents respectively with 0.1 percent [1 agent] initially infected

From above it is found that in population-based modelling of epidemics a similar percentage of initially infected population yields in a similar percentage of recovered population for all total population sizes; see Table 3. But in case of agent-based modelling the initial count of the infected population the (approximately) yields a similar percentage of recovered population for all total population sizes; see Table 4.

N	**10**	**100**	**1000**
I (0)			
10%	55.620	55.620	55.620
1%	34.910	34.910	34.910
0.10%	27.070	27.070	27.070

Table 3 Percentage of recovered population in total population count [N] 10, 100 and 1000 agents respectively with 10, 1 and 0.1 percent initially infected [I(0)] in population based simulation.

N	**10**	**100**	**1000**
I (0)			
1	61.620	60.485	58.502
10	100.00	93.506	92.982
100	-----	100.00	93.830

Table 4 Average Percentage of recovered in the sample of 1000 simulations for total population count [N] 10, 100 and 1000 agents respectively with 1, 10 and 100 initially infected agents [I(0)] in agent based simulation.

Taking this number of initially infected agents rather then percentage of initially infected population as a parameter for similar experimental configuration is yet another subtle difference between population-based and agent-based simulation results of epidemics; see Table 3 and Table 4.

7. DISCUSSION

Papers addressing agent-based simulation of epidemics usually do not make a comparison between population-based models and agent-based models; see for example (Emrich, Suslov, Judex, 2007). Although in (Bagni, Berchi, and Cariello, 2002), a number of different types of models are briefly discussed, these models have not been compared by applying them to certain scenarios.

The comparative exploration of population-based simulation and agent-based simulation reported in this paper shows different phenomena that were not directly easy to predict. For the settings that were used as an illustration, a large number of agent-based simulations based on only 10 agents provided an average of infected and recovered persons around 5 that is not far from the results of the population-based model with the same settings. However, the variation was very wide. It was very rare that the simulation came up with a result that is close to the average. On the contrary, almost half of them ended up in 1, and almost the other half in the maximal number of 10 recovered persons. For higher numbers of agents (100 and 1000) the outcome is completely different.

For these cases the outcomes concentrate on the maximal number of infected and recovered persons; the variation is very low for these cases. Furthermore, the averages are also close to the maximal number of persons and therefore deviate a lot (around 100%) from the outcome of the population-based model with the same settings with average around 5.

Based on the results of this comparative case study the following can be noted:

- *Average outcomes do not match well*

The assumption that a population-based model shows the same results as the average of agent-based models only holds for the smaller number of agents, not for the larger numbers; this is the opposite as is sometimes assumed: that for larger numbers of agents the averages will approximate the outcome of a population-based simulation. This assumption is refuted by the simulation experiments.

- *Variation low for large numbers of agents*

However, for small numbers of agents the variation is so high that the average number gets less meaning.

- *Agent-based simulations more faithful?*

The answer on this question can be yes or don't know. Yes because a real difference is shown, so probably the agent-based model will be closer to reality. The answer can also be 'don't know' because it is not clear at forehand which of the two different outcomes is closer to reality. Possibly reality is even in between the two outcomes. To verify this, detailed empirical data have to be analysed, which was not (yet) performed for this first explorative study.

- *The threshold law*

The threshold law shows in both the agent-based and population-based simulations.

Further work would be to take empirical data and compare the two types of models with these data. Moreover, the relationship between equilibria and the initial values for susceptibles and infectives can be explored further in the context of empirical data.

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