Simulation of a Horizontal and Vertical Disease Spread in Population

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Abstract. The vertical disease spreading from parent to offspring and/or horizontal transmission through infection is discussed, using cellular automata approach implemented on a $N \times N$ lattice. We concentrate on age distribution of the population, resulting from different scenario, such as whether newborns are placed in close vicinity of parents or separated from them. We also include migration aspect in context of disease spreading. Main conclusions drawn are that the vertical version is resistant to manipulations of parameters which control migration. Horizontal version represents self-recovering population unless migration of grown-ups is introduced for the case of offsprings located in vicinity of parents. Then the migration seems to be beneficial for highly infectious and lethally diseases, while it brings more deaths for milder infections.

1 Introduction

Most population evolution models are based on differential equations which describes a statistically significant and representative member of the population and applies deterministic rules to its time evolution. Time is then a continuous variable. However it is not so often that then we may solve the set of differential equations and then we apply discrete time and set rules to predict the $t \to t+1$ transition of the system. Obviously, it may work for the time step sufficiently small, when changes in parameters characterizing the system are small, too. In simulation iterations, time is discrete. The the system often shows elementary interactions between its components on microscopic scale, which is not well described in terms of spatially continuous distribution. In fact, we often get quite different result [1]. Cellular automata [2] is a proper tool for that case, either in the standard deterministic version or for the probabilistic rules. If the system is also vulnerable to some non-deterministic component, it is easy to implement erratic behaviour as a noise or more correlated deviations from the deterministic picture.

The cellular automata technique is often used to describe dynamics of the infection by some viruses [3]. In basic epidemiological models, a disease may be transmitted horizontally through infection (say, due to a direct contact) and/or

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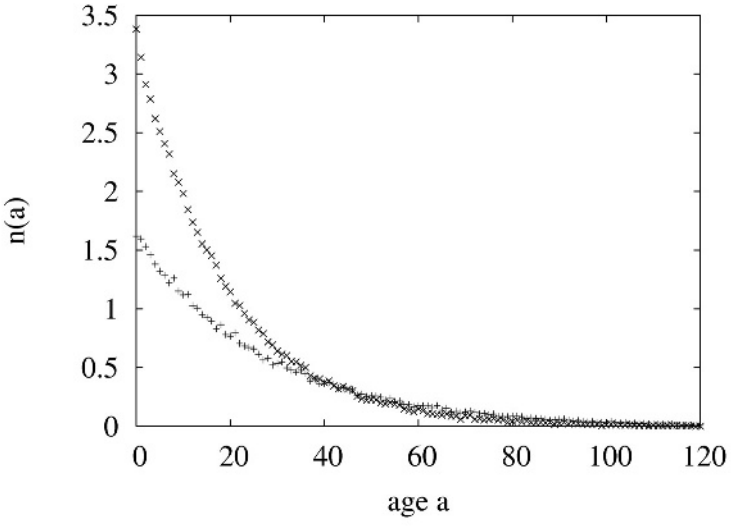

Fig. 1. Simulated age a distribution of population $n(a)$, in arbitrary units, of infection-free population. Upper case corresponds to newborns randomly placed at any lattice cell. Lower case is obtained if babies may be placed only in the nearest neighbours cells of parent's site

vertically, i.e. from parent to offspring. More mathematical approach of parasites as carriers in can be found in [4,5]. However, we do not intend to interpret the typical task of immune system simulation which involves the many different specialized biological cells (B-cells, macrophages, helpers and others), apart from the virus itself, and all related interactions and relations between them. Here we confine ourselves to very simple description of a two dimensional $N \times N$ lattice with cells free or occupied by one item, either infected or free of the virus. The dynamics is controlled by a proposed set of parameters in each evolution step $t \to t+1$ evolution. After the many iterations we mostly concentrate of space or/and age distribution of items, infected or not. Typical age distribution of the non-infected population is shown in figure 1.

We intend to simulate both vertical and horizontal version of the infection pass, and see how it may influence the $n(a)$ distribution.

2 Model

The basic algorithm assumes given number of iteration cycles, for every cycle we scan the $N \times N$ lattice and apply evolution rules to non-empty i-cells, $i = 1..N^2$. Each item is characterized with its age $a(i)$, parameters $c(i)$ responsible for an overall health condition and $v(i)$ indicating the virus infection. At each time step, the i-th individual is verified:

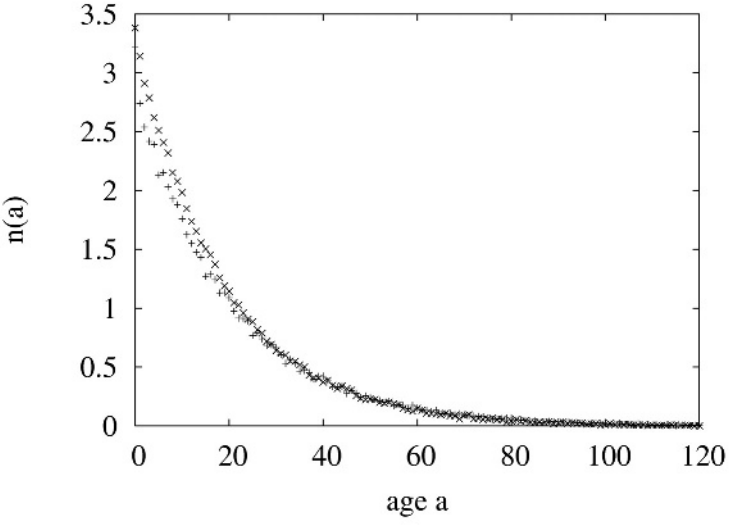

Fig. 2. Age distribution $n(a)$ for vertical transmission of infection. Babies are freely scattered over the lattice. Upper case is for no infection passed, lower case is for 80% offsprings affected and 80% of them later killed by the developed disease

- if its age a is above a biological maximum age $maxA = 120$, the item dies,
- then it also may die with probability $p \propto n/N^2$, the Verhulst factor [6], where n is the current population size,
- if the individual survived, and its age is above minimum reproduction age (here 16) yet still below maximum reproduction age (here 48), it gives birth to $B = 0.2$ babies (It means a baby with a copy of parent's c, is born with conditional probability 0.2, if the proposed destination is an empty cell. It can be chosen either on the whole lattice or limited to the nearest neighbourhood of parents),
- infection may take place according to specification below,
- item's c is corrected according to the nearest neighbours c-values, so that better neighbours pushes its c up, (and *vice versa*)
- also the intensity v of the already infected is up as the disease develops,
- further elimination process continuous - the item is out if its c is below a threshold value $minC$, here $minC = 0$,
- if v is more that a maximum value $maxV$, the item' future is decided: it dies with probability pV, else it is cured and $v = 0$,
- at this stage the individual has survived and enters the next time $t + 1$, perhaps after some movements due to migration process which takes place with probability $pMov$, then getting one year older, $a \to a + 1$ and a little less fit, $c \to c - \Delta c$.

In the vertical transmission version, the baby catches disease $v = 0 \to v = 1$ with probability pI if parent's v is from $v1$ to $v2$. Horizontal transmission is

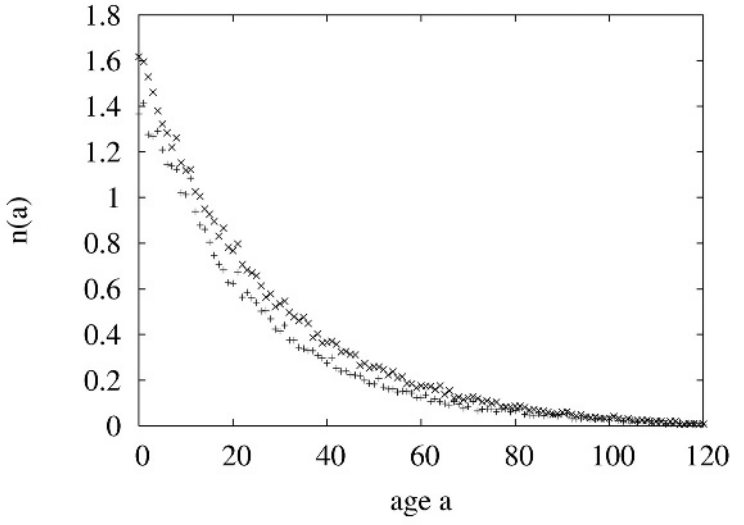

Fig. 3. Age distribution $n(a)$ for horizontal transmission of infection through neighbours. Babies are fixed in vicinity of parents. Upper case is the infection-free population, lower case is for 20% offsprings affected, and 80% of them are later killed by the disease

similar, yet the virus is passed from the nearest neighbour. When the scan all over the lattice is finished, the iteration cycle is completed with standard cyclic boundary conditions.

3 Results and Discussion

The age distribution of the population for vertical transmission of infection to babies is shown on Fig. 2. The two branches of points correspond to (a) no disease passed (upper set), and (b) $pI = 0.8$ fraction of offsprings of ill parents picking up the disease (lower set). When the disease develops and the critical v is reached, the model assumes only 20% of the individuals do recover, $pV = 0.8$. It is seen that a reduction of 10-20% of population is the net result of the vertical disease transmissions, yet the overall characteristics of age distribution is nearly the same. It reflects the long time scale of this type of infection, since the harmless period of the illness development must last long enough for the parent to reach the minimum reproduction age, and more to give chance to produce new items. The discussed case of babies free to sit at any empty lattice site may be verified against the version when babies are kept near the parents. As expected, apart from the general trend of smaller population as result of less room for new members, recall Fig. 1, we observe similar effect of 10-20% further reduction of population and no change in age distribution after it is normalized to cancel out the population size effect.

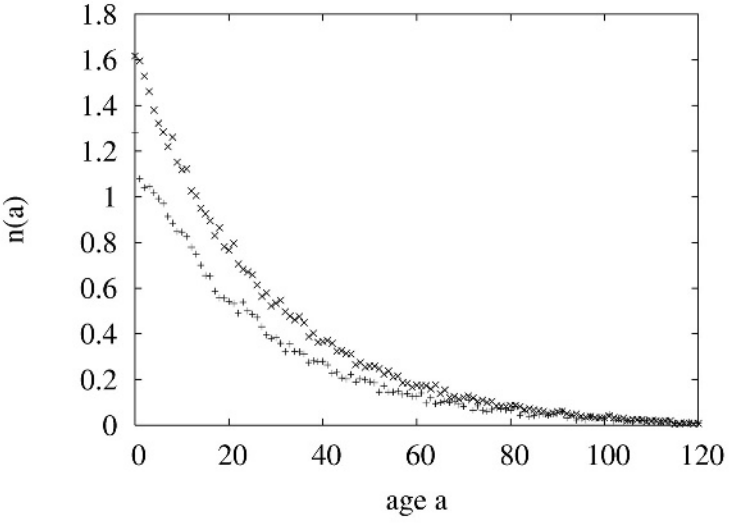

Fig. 4. Age distribution $n(a)$ for simultaneous vertical and horizontal transmission of infection, with babies bound to stay with parents. Upper case is the reference of the infection-free population

Contrary to the vertical case, the horizontal transmission may yield different effects for bounded and unbounded location for children. One can anticipate more significant differences for the bounded case since the nearest neighgours are responsible for the disease spread. Indeed, Fig. 3 for the bounded case shows a decrease in $n(a)$ distribution, in comparison with infection-free population, especially in the middle age fraction of population. The free choice of location for children gives only a tiny smaller $n(a)$ with respects to the disease free reference case. For calculations we used $pI = 0.2$ and $pV = 0.8$. The number 0.2, which replaces 0.8 for vertical case, was chosen as there are 4 nearest neighbours and each of them may infect. Also the time scale is generally much shorter - this time it is not essential that the disease carrier must live long enough to pass the virus. In fact, we applied half of the whole life span as the time for which the vertical disease develops, while only 3% of maximum age limit for the horizontal diseases. If both horizontal and vertical (h&v) mechanisms are present, see Fig. 4, the deviation from the disease-free population is bigger than if only one of the two named mechanisms are active. This is obvious, yet significant difference is the interference which make the net result is not the simple sum of contributions coming from the two contributions. (This may be seen for more detailed analysis when we compare (h&v) data against the independent contributions (h)+(v).) Such interplay is the consequence of the elimination mechanism implemented in the model. We treat the two cases as corresponding to different units, yet weakening by one of the disease makes the item is less resistant to the other sickness and so the death is then more likely. The $n(a)$ distribution may also

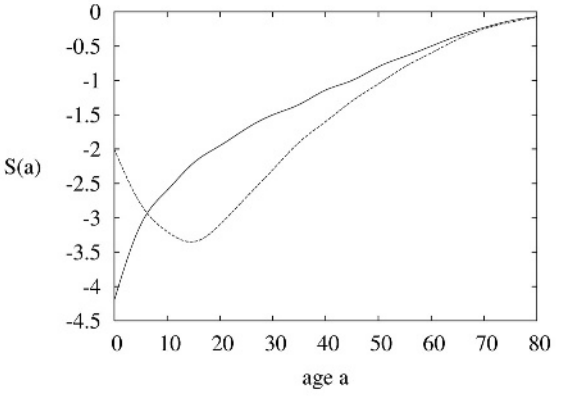

Fig. 5. Results of simulation of age dependence of $S(a) = \log q$, the exponential Gompertz law of mortality $q(a)$ predicts a straight line. The line showing minimum is for the case of simultaneous vertical and horizontal transmission of infection, the other line corresponds to the infection-free population

be seen in terms of the usually discussed mortality $q(a) = 1 - n(a+1)/n(a)$, which is a fraction of the population eliminated at age a. The Gompertz law predicts exponential dependence of $q(a)$, $q(a) \sim e^{b \cdot a}$, which yields a straight line on logarithmic plot of $S(a) = \log q$ against age a. The results of simulations presented on Fig. 5 is the translation of Fig. 4. It shows the general tendency that the disease presence in population changes the mortality distribution. This change leads to a minimum in the $S(a)$ dependence, a feature observed in human population. Deviations from the Gompertz law is of a main interest in many publications, see for example [8] for review.

It is interesting to include migration processes for the horizontal version of disease spread, in the case of babies staying with the parents. The alternative case brings in no effect of migration which is already present in the form of offfsprings dispersed all over. For the new members kept close by, the migration may play dual role. Firstly, for highly infected clusters of local communities, it is a chance to escape from the doom of unavoidably getting infected. Then migration lessen the infection pressure and so the whole picture is shifted towards less infectious environment. This effect is illustrated in Fig. 6. The reference case (upper points) with 30% risk of the disease leading to the death, $pV = 0.3$, is recalculated with higher risk $pV = 0.72$. Not only the population drastically drops (bottom points), but also the distorted age distribution $n(a)$ due to a jump at age $a = 12 \to 13$, is well pronounced. This is so as we applied a sharp disease development limit, $maxV = 12$. If intense migration is allowed, the middle points, some recovery is then observed and the population size is higher.

However, if only a tiny fraction of population is infected, migration helps the infection to spread all over, especially if the pI parameter, indicating of how likely is the virus transmittable, is high.

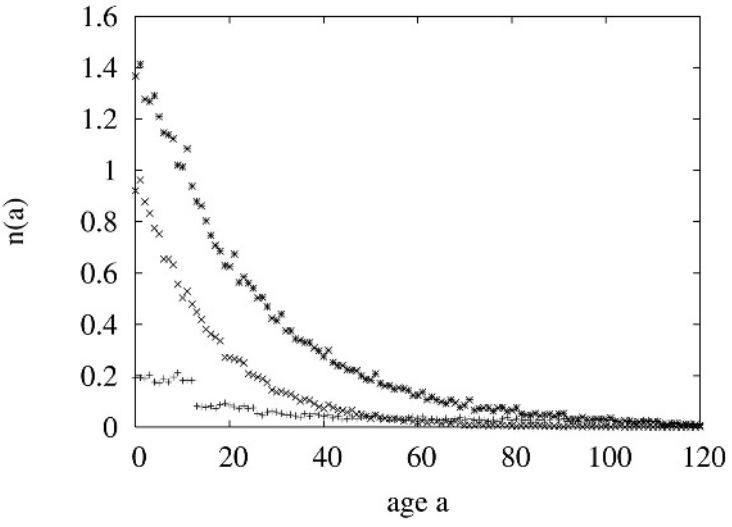

Fig. 6. Babies by parents case. The reference (upper points) set of death risk $pV = 0.3$, is confronted against much higher risk $pV = 0.72$, the bottom points. When high rate of migration $pMov = 0.9$ is switched on, the situation is improved, the set of middle points

4 Conclusions

One can notice some resemblance of the cellular automata approach and the Penna model [7,8,9]. In a way, the parameter c coding the item's health condition, plays similar role as the genome in the Penna model. Activated mutations in Penna model and the threshold maximum value of bad mutations correspond roughly to the decrease in c as time flows. Limited environmental capacity is equivalent to the lattice size $N \times N$, or more precisely $(N \times N - n)$ sites left free as n sites are already occupied. Therefore to some extend the proposed approach may be considered as an alternative.

Main conclusions of the proposed approach were already discussed in the main text. In short, the vertical transmission of infection makes population a little smaller by 10-20%, yet with nearly no effect on age distribution, apart from arbitrary normalization factor. In this case neither migration nor possible fixing of the offsprings make difference in output.

The horizontally passed infections are not important if babies are isolated from parents. It is only during the initial transient period that the population in small isolated clusters suffer and die, or get cured, while babies being free from infection do not carry the disease to their far destination. If babies are in vicinity of parents, they are also vulnerable to the disease. Then the age distribution $n(a)$ differs from the reference sickness-free case, and also possible migration influences the results. The migration itself is helpful for rapidly spreading and

deadly infections. For milder infections, migration is responsible for spreading the disease and also increases the death toll.

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