NEMO Working Paper #18

Knowledge Transmission Processes with Local Thresholds and Coupling to Mean Field Processes

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Supported by the EU FP6-NEST-Adventure Programme

Contract n° 028875 (NEST)

(Milestone 2.4a)

This version June 2009

Abstract

We introduce a model for knowledge diffusion on complex networks using generalized epidemic processes. Knowledge is modeled like a disease spreading over a network and infecting vertices following certain local and global rules. We distinguish active and passive knowledge, and the possibility of forgetting knowledge is implemented. Several simulations are done and show the behaviour of our model of knowledge diffusion on complex networks.

Contents

1 Introduction 4

2 Notation 5

3 Generalized Epidemics 5
- 3.1 Learning: From "no knowledge" to "passive knowledge" 6
  - 3.1.1 Infection below the threshold: the ϵ-process 6
  - 3.1.2 Infection above the threshold: the α-process 7
  - 3.1.3 Mean field effects: the β-process 7
- 3.2 Understanding: From "passive knowledge" to "active knowledge" 7
  - 3.2.1 Activation by neighbors 8
  - 3.2.2 Activation by the whole network 8
  - 3.2.3 Spontaneous activation 8
- 3.3 Forgetting knowledge 8
- 3.4 Examples 9

4 Results 10

1 Introduction

To describe and understand the dynamics of knowledge diffusion within a group of individuals or a society is a difficult and challenging task. It is a specific example of a complex and therefore mathematical models are an appropriate tool to uncover some of the hidden dynamical properties. Since knowledge transfer and exchange has so many faces and each of these different aspects would perhaps require an entire own and specific model we want to focus in this paper mainly on the following:

1. local threshold dynamic
2. mean field dynamics and
3. active versus passive knowledge.

Since good data about knowledge transmission are notoriously difficult to obtain and even for simple "fact - knowledge" essentially not available, we will concentrate primarily on questions of qualitative nature. In this spirit one should also interpret the various simulation results discusses in section 3. Although concrete and reasonable parameters are chosen for the single simulation runs their main purpose is the illustration of the possible qualitative scenarios.
One of the most striking phenomena in knowledge spread is the likely presence of phase transitions. For most things we know, either they belong to the class of widespread knowledge or it is knowledge restricted to a rather small group of people. This observation extends as well for subcommunities like scientific fields: most articles are known only to a small group of scientist centered around the author group and just a few articles are known to the majority of researchers in the field. Although one can find specific examples which seem to contradict this zero-one law, like the level of awareness of a new product on the market, a more detailed look often shows that these examples are just on the way to a high prevalence or on the way to become forgotten.
The local transition rules we imposed in the model are of a (local) threshold type. In essence this means that below a critical number of infected neighbors the infection probability is very small and above this threshold it becomes comparable large. Additionally to face to face transmission we include the transmission of knowledge via public sources like mass media, books or journals. Since the likeliness of something to appear in such public sources is often proportional to the prevalence of the active "knowing" individuals in the society we therefore model this type of infection path by a mean field term infection probability depending only on the infection density but not on the network structure. Finally due to the very nature of human beings knowledge may share the fate of all of us namely become forgotten. It turns out that for both types of stochastic processes there are phase transitions of mainly two types. One relates to parameters describing the transmission probabilities and to parameters describing the network structure (primarily the edge density). Below some critical values an initial infection will not be able to spread over the network at all. This phase transition resembles the ones known from percolation theory. The second type of phase transition is more of a dynamical nature and unknown from classical epidemic processes. Namely even in the overcritical parameter domain where epidemic growth is in principle possible, one still needs to start above of a critical initial

density of infected individuals (depending of course on the chosen parameters) to reach high prevalences. A more detailed analyses of this phenomena in case the underlying network is a classical Erdös&Renyi random graph, is given in the last section.
The paper is organized as follows. After a description of the different infection paths in separate subsections we discuss possible applications and finally turn to the presentation of some simulation results for real networks and a mathematical analysis for the case of classical Erdös&Renyi type graphs.

2 Notation

Let $G = (V, E)$ be a network with $\#V = n$ vertices and assign to each vertex $v \in V$ a number $s_t(v) \in \{0, 1, 2\}$, which is called *the knowledge state* of v at time t, or simply state of v. We say that v has *no knowledge* if $s_t(v) = 0$, v has *passive knowledge* if $s_t(v) = 1$, v has *active knowledge* if $s_t(v) = 2$.
There are local observables for the neighbours of the vertices in the network, for each $v \in V$ we define $N_{i,t}(v) = \#\{w \in V : w \sim v, s_t(w) = i\}$, which is the number of neighbors of v in the state i at time t.
Define the *effective number* $\Omega_t(v)$ *of knowing neighbors of* v *at time* t to be $\Omega_t(v) = N_{1,t}(v) + cN_{2,t}(v)$, with some $c > 1$, which should take into account that neighbors with active knowledge have a greater influence on v than the others. Now denote by d_v the degree of a vertex v. The normalized quantity

$$\omega_t(v) = \frac{N_{1,t}(v) + cN_{2,t}(v)}{cd_v} \tag{1}$$

is *the effective frequency of knowing neighbors of* v *at time* t.

3 Generalized Epidemics

To understand knowledge diffusion in networks we need to discuss the probabilities $P_{i\to j}(v)$ of a vertex v to switch from state i to j. Since there are three distinct states, there are six transitions possible, but not all of them must be considered to be physically meaningful. The transition "$0 \to 2$" for instance corresponds to the change of "not knowing" into "active knowledge", which makes no sense as one needs to acquire passive knowledge first. Thus we set $P_{0\to 2} = 0$. Since we want to model knowledge diffusion over a relatively short period of time only, we assume that knowledge once activated can not be forgotten any more, i.e. transitions from active knowledge to passive knowledge or to not knowing are forbidden. Thus, $P_{2\to 1} = P_{2\to 0} = 0$.
This leaves us with three transitions to be modeled and discussed. The corresponding probabilities $P_{0\to 1}(v), P_{1\to 2}(v), P_{1\to 0}(v)$ of a vertex to switch its state each are determined by different independent processes, arising from local effects, which are related to the neighborhood of a vertex, global effects related to the whole network and spontaneous effects related to the vertex itself.
First of all we discuss the transition $0 \to 1$, i.e. achieving passive knowledge or simply learning. We introduce three parameters, two of them belonging to a common epidemic process with a threshold, the third one is associated with a mean field effect on the network. Then we turn our view to the transition

$1 \to 2$, the evolution of passive knowledge to active knowledge, where we establish another three parameters, one belonging to a classical epidemic process, one belonging to a mean field process and one to include the possibility of a spontaneous transition. Finally we introduce two more parameters to describe loss of passive knowledge, the transition $1 \to 0$. Table 1 summarizes all parameters and their meanings.

3.1 Learning: From "no knowledge" to "passive knowledge"

The probabilities $P_{0\to1}(v)$ consist of three components, each component belonging to a different type of process. To each vertex v we assign a threshold $\Delta(v)$, which describes the resilience of v against learning something new. Thus, there a two cases, which we need to discuss, infection below the threshold and infection above it. One reasonable choice for the distribution of $\Delta(v)$ is a Poisson distribution with mean λ, which will differ from network to network. Of course, in some cases it may be useful to choose a completely different distribution for $\Delta(v)$, but we will not consider such distributions here. Besides these local effects, there is a global effect influencing the transition from "no knowledge" to "passive knowledge".
The strength of the influence of the neighborhood of some vertex is determined by the number of infected neighbors. We use the effective number of knowing neighbors $\Omega_t(v) = N_{1,t}(v) + cN_{2,t}(v)$ to take into account, that neighbors of v with active knowledge have a greater influence than neighbors having passive knowledge only.

3.1.1 Infection below the threshold: the ϵ-process

If the effective number of knowing neighbours is below a threshold, i.e. $\Omega_t(v) < \Delta(v)$, then every knowing neighbor contributes a small probability proportional to some $\epsilon > 0$ to the probability of switching he state from 0 to 1. Usually one chooses the probability of infection to be $P = \epsilon K$, where K is the number of infected neighbors, but here we make a slight modification. We take into account that the vertices use each time step to spend on communication with their neighbours to learn and acquire knowledge. We assume that each vertex v distributes its communication time equally on each neighbour, ending up communicating a time of $1/d_v$ with each neighbour. Thus, the probability of a vertex v of switching from no knowledge to passive knowledge below the threshold is proportional to the ratio of knowing neighbours and its degree d_v,

$$P^{\epsilon}_{0\to1}(v) = \epsilon\omega_t(v) = \epsilon\frac{N_{1,t}(v) + cN_{2,t}(v)}{cd_v}.$$

The factor c in the denominator is a normalization constant for the effective frequency (1), which we choose rather than the unweighted sum of knowing neighbors because neighbors with active knowledge have a greater influence on the vertex, due to a natural authority they achieved by having active knowledge and using it.

3.1.2 Infection above the threshold: the α-process

When the number knowing neighbors exceeds the threshold, $\Omega_t(v) \geq \Delta(v)$, the contribution of single probabilities gets replaced by a probability $\alpha >> \epsilon$ of getting infected, which we choose to be a constant here,

$$P^{\alpha}_{0\to 1}(v) = \alpha.$$

The idea is, that a vertex above the threshold is willing to switch ist state, but still has some issues, for instance switching costs too much money, too much time, something depending on the specific knowledge diffusion under investigation, and any more contact with knowing vertices will not change its probability to switch anymore. The value of α is then just a measure for these issues.

3.1.3 Mean field effects: the β-process

The influence of mass media and other global effects is modeled by a mean-field process with parameter β and a function $f : [0, 1] \to [0, 1]$, which takes the total prevalence density b_t/n of vertices with any active or passive knowledge at time t as arguments. Which form f takes depends on the model under consideration, the simplest choice is the identity function, which is in many cases a good approximation to reality. We choose $f(b_t) = b_t^2/n^2$, allowing higher values of the prevalence to have a higher weight. So,

$$P^{\beta}_{0\to 1}(v) = \beta f(b_{t-\tau}/n),$$

where τ is some time lag describing the incapacity or disability of the network to influence vertices instantaneously. For instance, let the network be the network of scientific publications. Scientists with active knowledge produce paper, but the publication process needs some time to get recognised by other scientists and even more time to get scientists to produce similar, i.e. to achieve active knowledge.
From the considerations above it follows that the total probability $P_{0\to 1}(v)$ of a vertex achieving passive knowledge is

$$\begin{aligned} P_{0\to 1}(v) &= 1 - (1 - P^{\epsilon}_{0\to 1}(v)\Theta(\Delta - \Omega))(1 - P^{\alpha}_{0\to 1}(v)\Theta(\Omega - \Delta))(1 - P^{\beta}_{0\to 1}(v)) \\ &\approx P^{\Delta}_{0\to 1}(v) + P^{\beta}_{0\to 1}(v), \end{aligned}$$

where we set $P^{\Delta}_{0\to 1}(v) = P^{\epsilon}_{0\to 1}(v)\Theta(\Delta - \Omega) + P^{\alpha}_{0\to 1}(v)\Theta(\Omega - \Delta)$ and neglected all products of probabilities.

3.2 Understanding: From "passive knowledge" to "active knowledge"

Similar to the transition "not knowing" to "passive knowledge", there are three main effects, that compose the probability of switching from "passive knowledge" to "active knowledge". The local effects express the infection by neighbors with "active knowledge", describing the influence of active neighbors on a vertex. Global effects arise from the total prevalence of the network, i.e. the total number of vertices with active knowledge. Also we admit spontaneous infection with active knowledge, which describes the possibility of a vertex discovering knowledge (and therefore activating it) on his own.

3.2.1 Activation by neighbors

Steady communication of neighbors with active knowledge may lead to the activation of passive knowledge, which one already has achieved. We suppose, that this probability is proportional to the density $N_{2,t}/d_v$ of actively knowing neighbors, with proportionality factor ζ^{loc},

$$P_{1\to 2}^{\text{loc}}(v) = \zeta^{\text{loc}} \frac{N_{2,t}(v)}{d_v}.$$

3.2.2 Activation by the whole network

Similar to the mean field effect in gaining passive knowledge, the total prevalence density of vertices in the network with knowledge has an influence on every vertex v, with the difference that only vertices with active knowledge can contribute to the probability of switching from passive to active. The probability is given by

$$P_{1\to 2}^{\text{glo}}(v) = \zeta^{\text{glo}} f(A_{t-\tau}/n),$$

where $A_{t-\tau}$ is the total prevalence of vertices with active knowledge in the network, at a time lag τ.

3.2.3 Spontaneous activation

It may happen that one activates its passive knowledge on his own:

$$P_{1\to 2}^{\text{spo}}(v) = \zeta^{\text{spo}}.$$

For instance, a vertex discovers something new accidently and lifts himself from passive to active or it just decides to switch from passive to active on its own by learning and working all alone. See the examples below for an illustration of this effect.
The total probability $P_{1\to 2}$ of a vertex of changing its state from passively knowing to actively knowing is therefore given by

$$\begin{aligned} P_{1\to 2}(v) &= 1 - (1 - P_{1\to 2}^{\text{loc}}(v))(1 - P_{1\to 2}^{\text{glo}}(v))(1 - P_{1\to 2}^{\text{spo}}(v)) \\ &\approx P_{1\to 2}^{\text{loc}}(v) + P_{1\to 2}^{\text{glo}}(v) + P_{1\to 2}^{\text{spo}}(v) \\ &= \zeta^{\text{loc}} N_{2,t}(v)/d_v + \zeta^{\text{glo}} A_{t-\tau}/n + \zeta^{\text{spo}}, \end{aligned}$$

where we neglected all products of probabilities.

3.3 Forgetting knowledge

Since the times under consideration are not too long, we propose that it is impossible to forget active knowledge, i.e. once a vertex has activated knowledge it stays active. Also there is no global effect triggering forgetting of knowledge, we only take spontaneous effects and local effects into account. We claim that the probability of forgetting knowledge by a local effect is proportional to the density of not knowing neighbors, here without any special weighing of neighbours with active knowledge. Of course, any vertex may forget knowledge spontaneously, just by not remembering anymore. This effect is characterized by a global constant γ^{spo}, which ensures additionally, that if all neighbours are knowing, one may forget knowledge anyway. Thus

$$P_{1\to 0}(v) = \gamma^{\text{loc}}(1 - \frac{N_{1,t}(v) + N_{2,t}(v)}{d_v}) + \gamma^{\text{spo}}.$$

Table 1 summarizes all parameters and their meanings.

parameter	transition	affiliation
ϵ	$0 \to 1$	infection below the threshold
α	$0 \to 1$	infection above the threshold
λ	$0 \to 1$	mean of the distribution of the threshold
β	$0 \to 1$	mean field
ζ^{loc}	$1 \to 2$	local effects
ζ^{glo}	$1 \to 2$	global effects
ζ^{spo}	$1 \to 2$	spontaneous effects
γ^{loc}	$1 \to 0$	local effects
γ^{spo}	$1 \to 0$	spontaneous effects

Table 1: Summary of all parameters

3.4 Examples

To illustrate the various parameters of the model of knowledge diffusion in networks we give some examples. To do so, we need to specify for each example, what is meant by passive knowledge and by active knowledge.
Table 2 summarizes all examples and the feasible values for the parameters.

- **The spread of popular media titles**
  An example of knowledge diffusion on complex networks is the spread of popular media titles, like books, movies or music. The underlying network is the social network of people using the media. A person has passive knowledge, if it is aware of the existence of the media, i.e. it knows that a certain book or movie exists (e.g. Harry Potter) and is curious about it, but did not read or watch it for several reasons, yet. Active knowledge then is the consumption of that media and talking about it in public.

- **Trends: Nordic walking**
  Nordic walking is a popular example for a trend on a social network, what can be described by the knowledge diffusion model as well. Having active knowledge means performing nordic walking in public, having passive knowledge means that one has recognized nordic walking and has a positive attitude about it. The positive attitude is important, since this is a necessary condition to switch to active knowledge. So, a person with passive knowledge is willing to perform nordic walking but has some issues, like a lack of time or equipment.

- **The usage of certain hardware**
  The knowledge diffusion model can describe the spread of certain hardware

like MacBooks or iPhones on a network. Active knowledge means using that hardware and showing and telling it to friends. Passive knowledge is, similar to the nordic walking trend, the recognition and positive attitude about the hardware.

- **The usage of certain software**
  As in the case of hardware, one can describe the spreading of certain software products, like internet browsers or different media players, in a network. Having passive knowledge means having the software installed and used at least once and keeping it on the computer, having active knowledge means using that certain software as standard (browser or player), using other competing software only occasionally.

- **The usage of scientific products**
  With scientific products we mean special science related products like a new alternative model for an observed effect or scientific software such as SKIN, which is an agent based simulation software. Having passive knowledge means knowing the product and unsing it occasionally, active knowledge means using the product and perhaps developing it further.

- **Citing the NEMO project**
  Citation can also be described as a knowledge diffusion process on a network. The vertices have passive knowledge if they know about the NEMO project and what is developed there, and active knowledge if they are writing papers and citing NEMO work.

	ϵ	α	λ	β	ζ^{loc}	ζ^{glo}	ζ^{spo}	γ^{loc}	γ^{spo}	τ
popular media	lo	hi	3	hi	lo	lo	lo	lo	lo	lo
nordic walking	lo	hi	5	lo	hi	lo	lo	hi	hi	hi
computers	lo	hi	4	hi	hi	hi	lo	lo	lo	lo
software	lo	hi	4	lo	hi	hi	lo	lo	lo	lo
scientific products	lo	hi	4	lo	lo	hi	lo	hi	lo	hi
citing NEMO	lo	hi	4	lo	hi	lo	lo	lo	lo	hi

Table 2: List of examples for types of knowledge. "hi" means a value close to 1, "lo" means a value close to zero. In the column for the time lag τ "hi" means the time lag is huge and it takes a long time until time lag related effects take place, "lo" means they influence the network almost instantaneously.

4 Results

Our simulations use the framework program database from the NEMO project, strictly speaking the data from the framework programs FP1, FP2 and FP3. In order to test the main features of the model, our simulations did not use the full set of parameters. This is still work in progress and will be done later, with an even more sophisticated version of the model. The simulations were done using a 5 parameter model, where the parameters of the transition $0 \to 1$ are included, but the threshold Δ is always chosen to be $\Delta = 4$ for each vertex in the network. The activation of knowledge in the simulation is simplified to one

single parameter ζ, which is taken to be constant, as well as the paramter γ describing loss of knowledge via $P_{1\to 0}(v) = \gamma^{\mathrm{loc}}(1 - \frac{N_{1,t}(v)+N_{2,t}(v)}{d_v})$. Furtheron we did not make use of the weighing parameter c in the effective frequency (1), setting $c = 1$ in all simulations.
The simulations were done using two different setups, both concerning the initial conditions of the distribution of vertices with passive knowledge. One the one hand, a cluster consisting of 200 vertices was set to "passive knowledge" in each of the three FPs. On the other hand a fraction of 5 percent of the vertices of the network was chosen to haven passive knowledge at random. This enables us not only to compare the knowledge diffusion on the framework projects, but also a possible dependence on the initial conditions. Notice, that since the model forbids forgetting knowledge once activated, each network ends up in a state, where all vertices have active knowledge. Each run was therefore stopped, when the relative prevalence ω_t reached its maximum value $\omega_t^{\max} = 1$.
Figure 1 shows the behavior of the relative prevalence curve depending on time with fixed total initial infection of 200 vertices. It can be seen that the FP3 reaches saturation of the relative prevalence first, FP2 and FP3 getting saturated with the same slope about 3 times later. The reasons for that are discussed later in this section.

Figure 1: Comparison of the relative prevalence of vertices having active or passive knowledege for the three Framework programmes FP1, FP2, FP3. The initial infection is a cluster of 200 vertices having passive knowledge.

Figure 2 shows the behaviour of the relative prevalence curve depending on time with fixed relative initial infection of 5 percent of all vertices. The most striking difference is the behaviour of the prevalence of the FP1. While FP2 and FP3 are saturated much faster, what seems clear since the initial total prevalence is higher, FP1 needs even more time to get saturated.

Figure 2: Comparison of the relative prevalence of vertices having active or passive knowledege for the three Framework programmes FP1, FP2, FP3. In each of these there are 5% of all vertices initially infected and randomly distributed over the network.

Each of the figures below displays the curve for relative prevalence (black) and is resolved into 4 curves, which contribute to it. The cyan curve belongs to the ϵ-process, which belings to the local infection below the threshold. On each of the figures its influence is weak, due to the specific choice of the parameters. The dark blue curve belongs to the α-process, the process of local infection above the threshold. The green curve belongs to the β-process, which is the mean field influence, the red one belongs to the γ-process, which counts the ratio of vertices, that forget knowledge. Notice that in each figure below the black curve for relative prevalence is always the sum of all other curves, but the red curve of the γ-process has a minus sign, since this is the ratio of vertices, which switches back from being infected with knowledge to being not infected.
There are two more curves, displaying the behaviour of the ratio of vertices with active knowledge. The orange curve belongs to the ζ-process, which counts the

ratio of vertices having active knowledge. The pink curve "ratioActive" is the ratio of the total number A_t of vertices having active knowledge at time t and the toal number Ω_t of infected vertices at t, i.e. "ratioActive"$= A_t/\Omega_t$. Both curves always coincide when the relative prevalence reaches its maximum.

Figure 3: Prevalences of each process contributing to the total prevalence for a single run on FP1 in figure 2. The system needs a long time to be saturated due to the weakness of the α-process and the high rate of forgetting knowledge.

The main reason for FP1 getting saturated that late is the lower averagre degree of the network, declining the vertices to climb above the necessary threshold of 4 infected neighbours to get infected by the α-process. Since the infectivity of the ϵ-process is always small, local effect do not almost not contribute to the total prevalence at all. Figure 3 shows, that then time exceeds $t = 600$, the main contributor to prevalence is the mean field process, that gets fired by its square dependence of the total prevalence, and grows faster than the γ-process, which lets vertices forget (passive) knowledge. The most remarkable effect is that the ratio of vertices in the network infected by the mean field rises to a total of 1.1, indicating that some vertices get infected twice per step, they get infected with passive knowledge, forget it, and get infected again.
Since new vertices get infected very slowly the "activeRatio" increases strongly, because the number of vertices with active knowledge increases faster than the number of vertices with passive knowledge. Not until the mean field effect gts dominant the number of vertices with passive knowledge increases more the the number of vertices with active knowledge.

Figure 4: Prevalences of each process contributing to the total prevalence for a single run on FP2 in figure 2. The system gets saturated very fast since the local and global infection processes compensate the effect of forgetting knowledge.

The situation is altered if the simulation is run on FP2. Figure 4 shows, that the α-process is contributing from the start and therefore compensating the γ-process of forgetting. Thus, there is no counterforce weakening the mean field process and the network is saturated very fast. The "activeRatio" curve has a similar behaviour but is damped in contrast to FP1. As the number of vertices with passive knowledge increases faster than the number of vertices with active knowledge, the maximum is reached earlier and its total value is lower than for FP1.
All this is true for the FP3 as well, seen in figure 5. The α-process dominates all other ways of infection, increases the prevalence and prepares the way for the mean field process with its square dependence of the relative prevalence. Already at time $t = 100$ the repelling effect of forgetting knowledge plays no significant role any more. Since the increase of the number vertices with passive knowledge is enormous, the "activeRatio" stays below values of 0.1, but shows a similar behaviour to FP1 and FP2. When the simulation starts, the number of vertices with active knowledge increases always faster than the number of vertices with passive knowledge. This effect is reversed not until the infection

processes dominate the γ-process of forgetting and cancel out the loss of passive vertices.

Figure 5: Prevalences of each process contributing to the total prevalence for a single run on FP3 in figure 2. Forgetting knowledge does almost not take place, the system runs into saturation rapidly.

We now turn our view to the other setup, with a fixed initial total infection of a cluster of 200 vertices with passive knowledge. The most striking difference can be observed in the behaviour of the prevalence FP2. It needs even longer to get saturated than the FP1. While the infection proecesses almost look the same for the FP1 and the setting with 5% initial total infection, see figure 6, for FP2 things have changed. With an initial infection of 200 vertices instead of 476, most of the vertices can not climb from the start above the threshold to trigger the α-process. Thus, the mean field process must take over. Since vertices with active knowledge can not forget, their number increases, what is illustrated by the "activeRatio" curve. Vertices with active knowledge contribute at any single time step to the mean field process, with each time step a little more. Therefore, dominance of the mean field process is unavoidable, but it takes longer the less the α-process contributes.

Figure 6: Prevalences of each process contributing to the total prevalence for a single run on FP1 in figure 1. The graph hardly differs from that one seen in figure 3 ,since there is only a difference of 40 vertices being infected initially. Remarkably, it does make no difference if the initially infected vertices are placed in a cluster or are chosen at random over the whole network.

This can be seen in figure 8, in that setting, FP3 is the network with the loweset reltive initial infection, but gets neverthelessw saturated very fast, since the α-process is from the start the dominating infection process, until at time $t \approx 270$ the mean field process takes over and brings the network into saturation rapidly.

Figure 7: Prevalences of each process contributing to the total prevalence for a single run on FP2 in figure 1. The γ-process dominates all other infection processes and is hindering the network to get saturated. Hence, the "activeRatio" curve grows a long time and has a remarkable maximum.

Figure 8: Prevalences of each process contributing to the total prevalence for a single run on FP3 in figure 1. The α-process is the dominating process and fires the relative prevalence, until the mean field process (at about $t = 270$) is strong enough to saturate the network.