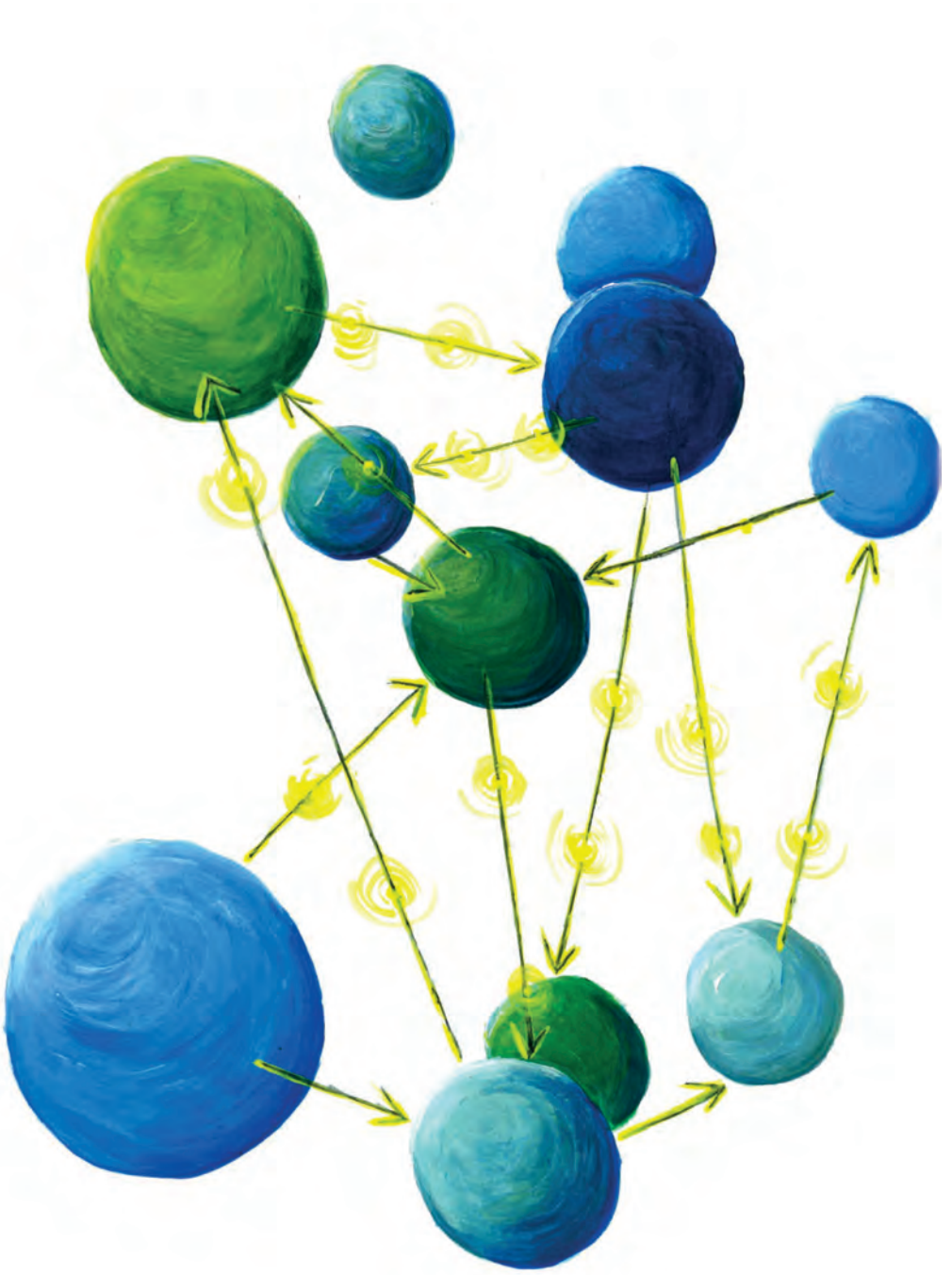
WILHELMUS HUBERTUS (WOUTER) VERMEER

Propagation in Networks

The Impact of Information Processing at the Actor Level on System-wide Propagation Dynamics



Propagation in Networks

The impact of information processing at the actor level on system-wide propagation dynamics

Propagation in Networks:

The impact of information processing at the actor level
on system-wide propagation dynamics

Propagatie in netwerken

De invloed van informatie verwerking op het actor niveau
op systeembrede propagatie dynamiek.

Thesis

to obtain the degree of Doctor from the
Erasmus University Rotterdam
by command of the
rector magnificus

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Wilhelmus Hubertus Vermeer
born in Rotterdam, the Netherlands.

Erasmus University Rotterdam

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social character has put a mark on the department and made it one of the best places to work. Nick; not only your creativity but also the ability to transform ideas into action are truly inspiring. Paul; your ability to put things in perspective is mind boggling, never change! Clint; your ability to make complex things simple (and vice versa) and your positive energy are unparalleled. Martijn, Konstantina, Panos, Xiao, Irina; such amazing times we had, I am proud to call you all my friends. While I believe that everyone in the department contributed to the atmosphere, a special thanks goes out to Cheryl and Ingrid for being the best support staff on campus, and to Joris, Bas, Thomas, Evelien, and Christina just for being amazing colleagues. In a large part the success of the Ph.D. trajectory is conditional on the people you work with; I have enjoyed working with all of you, and I am sure that in the future we will continue to do so.

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Chicago, September 2015
Wouter Vermeer

Executive summary

Often systems can exhibit behavior which is difficult to predict and steer. Interactions on the micro level (between actors within the system) result in propagation of behavior which can cause unforeseen dynamics on the system level. Understanding the effects of propagation, the process by which connected actors influence each other, therefore is crucial in order to understand how the state and behavior of a system will change.

Propagation literature has primary considered the way in which propagation dynamics scale from the local to the system-level, identifying the network structure as prime driver in this process. By focusing on the network structure, the impact of the mechanism by which propagation takes place has however been pushed to the background. In this dissertation it is argued that it is this mechanism which plays a crucial role in determining how propagation dynamics scale from the local to the system-level.

To map the mechanism of propagation, this dissertation puts forward a framework for propagation as an information processing process. It describes the propagation mechanism using the distinct sub-processes; sending out information (Radiation), transferring information (Transmission) and processing information (Reception).

This dissertation shows that using such a framework not only results in a more detailed and methodologically stronger model of propagation, but also that distinguishing these sub-processes is a prerequisite for effective interventions into propagation. It also shows that heterogeneity in different part of the mechanism have radically different effects on the dynamics at the system-level. This implies that specifying the mechanism is critical for understanding the system-level dynamics in cases of heterogeneous actor behavior. finally, it shows that the effects of network structure are highly conditional on the mechanism of propagation. When more complex propagation mechanisms are compared, a single network structure can result in very different dynamics at the system level.

As such, this dissertation identifies the mechanism of propagation as a critical component in understanding how micro-level behavior scales toward the system-level, and hence impacts system-wide dynamics.

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Chapter 1

Introduction

In the last decade a series of phenomena in which the actions of individuals resulted in emergent behavior of large groups of the population can be observed. Take, for example, the Ice-bucket challenge, in which people were challenged to take and ice-bath, has evolved into a global phenomenon. Adding an elements of atonement by donating to ALS foundations and at the same time 'nominate' friends for the same challenge via social media, has resulted in a cascade of ice-bucket videos and donations, raising over $50 million in donations within the time-span of a month for the ALS Association (Time, 2014). Such emergent behavior can have far-reaching consequences, as illustrated by the escalating project X parties in Houston and Haaren, where open party invitations via social media resulted in enormous crowds which ended up reaping havoc and causing substantial damage in the neighborhood surrounding the parties. The 2008 financial crisis, where information about potential risks of institutions drove the financial system to a halt and a near collapse. And the Arab spring in 2010-2012, where social media where used to rally enormous crowds to protest against the rulers, in many cases resulting in their eventual disposition. While the impact of such emergent behavior is apparent, the dynamics by which such behavior occurs remain difficult to identify, predict and manage.

The propagation process —the process by which a change in behavior/state of an actor results in a change of state or behavior of its connected neighbors— is the process that drives the emergent behavior. By means of propagation an individual is able to influence their peers, which can consequently influence their peers, resulting in behaviors that can quickly cascade throughout a population. As propagation describe the influence among peers, the process is suggested to leverage the structure of interaction among actors. Such a structure links actors together into a single interacting system which can be affected by propagation. These systems have become bigger and more integrated as the world has and is continuing to become more connected. This has yielded an increase in observed

phenomena of wide-spread propagation and an increase in the impact of such phenomena on society.

The increasing relevance of propagation has resulted in a wide body of literature on propagation and its dynamics in a vast range of contexts. The most common notions of propagation are those of contagion (of disease) (Dodds and Watts, 2004; Rahmandad and Sterman, 2008), social contagion (Burt, 1987; Galaskiewicz and Burt, 1991; Iyengar et al., 2011; Aral, 2011), social influence (Turner, 1991; Cialdini and Goldstein, 2004; Friedkin, 2006; Ma et al., 2014), cascading processes (of disasters) (Buldyrev et al., 2010; Buzna et al., 2006; Watts and Dodds, 2007), diffusion of innovation (Coleman et al., 1966; Rogers, 1995; Guler et al., 2002; Valente, 1996, 2005) and information diffusion (Aral et al., 2007; Reagans and McEvily, 2003). Each of them in their own right considers a process in which the actors' state or behavior influences that of their peers.
While there is quite some variation in how propagation is studied among these different settings, each of them captures how actors influence one another and focuses on understanding the outcomes of the underlying propagation process: its dynamics. In explaining these propagation dynamics, literature has consistently identified three drivers:

- The structure of interactions in the system, the network structure.
- The location in the system where the propagation process starts, the seed of the infection.
- The process by which the remainder of the system is influenced, the mechanism of propagation.

The network structure

The interactions among actors in a system can be represented in a network or graph. This network captures the infrastructure on which propagation can take place. Much research has been done on how the characteristics of this network, its structure, cause variations in the propagation dynamics. This research has, for example, shown that propagation dynamics vary across network topologies (Newman, 2003); so-called scale-free networks are claimed to facilitate propagation (Barabási et al., 2000) whereas random networks hamper it (Albert et al., 2000). These topologies can be described using a set of structural characteristics such as: path length, clustering coefficient, degree-distribution (both in- and out-degree) which can consequently be linked to the propagation dynamics. These characteristics describe the structure at the system level, and hence consider the structure

of the network as a whole. However, as the structure can vary locally, the local level structure can also affect the propagation outcomes.

The seed of infection

The local variations in the network structure play a crucial role in determining how unusual behavior —an 'infection'— can propagate from a local to a global scale. It is widely accepted that the structure in a network is inherently heterogeneous. This means that, depending on which part of the network is observed, the structural characteristics will differ. As the structure differs locally this would imply that the structure surrounding the seed of infection is crucial for determining the propagation dynamics in the earliest stages of the process. The early stages of propagation determine to a large extent the momentum a propagation process gains, therefore the local network structure can strongly affect the propagation dynamics on a system level. This indicates that the seed of the infection, which in essence determines the local structure available for propagation in the earliest stages of the process, plays a crucial role in determining the local dynamics of the propagation process. These local dynamics in turn can strongly affect the system-level propagation dynamics.

The mechanism of propagation

Not only the (local) structure but also the propagation mechanism influences the dynamics of propagation. Intuitively one would argue that some processes will be quicker and more effective in propagating to a wide population then others, indicating that the characteristics of the mechanism of propagation play some role in determining propagation dynamics. While the importance of these characteristics is generally recognized in literature, there is only limited research (e.g. (Rahmandad and Sterman, 2008; Jackson and Lopez-Pintado, 2013)) that systematically studies the differences in propagation mechanisms and their relationship to variations in propagation dynamics. Variations in the mechanism are commonly captured by implementing a notion of propagation as a stochastic process which occurs with a certain speed or probability, be it the likelihood of transmission, the chance of adoption, the percolation probability or the rate of spreading. Doing so allows for parametrization of the effectiveness of the propagation process by a single parameter on the system level. As this is a stochastic process, it can differ locally and hence allows for local variation in the dynamics. Such approaches, however, treat the propagation process itself as a black box, and provide no insight into the characteristics

of the mechanism which drive the propagation dynamics.

The mechanism of propagation as a key driver

From a management point of view, this lack of insight into the mechanism of propagation is a big shortcoming. In management a wide body of work on (business) process management (e.g. (Becker and Kahn, 2003; van der Aalst et al., 2003)) suggests that knowing and understanding the process will enable one to better steer and manage the outcomes of the process. In this dissertation a similar perspective on propagation is adopted. It is argued that by knowing the propagation process, the mechanism by which it occurs, one will be better able to understand the dynamics of the process, and consequently be better able to manage and steer the process outcomes.

Current literature on propagation has mainly focused on the (local) network structure. By doing so it has often assumed an oversimplified notion of the propagation mechanism, resulting in an incomplete perspective on propagation dynamics. This dissertation aims to close this gap in literature and will focus specifically at the effect of the propagation mechanism on propagation dynamics.

It contributes to the literature on propagation by (partially) untangling the impact of the propagation mechanism on propagation dynamics, and aims to provide evidence that considering the characteristics of the propagation mechanism is crucial in understanding system-wide propagation dynamics.

Literature on propagation has inspired a stream of research focusing on the robustness of network structures (Albert et al., 2000; Dodds et al., 2003; Buldyrev et al., 2010). This body of literature studies the extent to which networks keep functioning, and hence are robust, in the face of (cascading) failure. It links the propagation potential of networks to stability of the network; however while doing so it has assumed an (over)simplified notion of the propagation mechanism. Incorporating the effect of the mechanism of propagation is argued to change our perception of the drivers of propagation dynamics and therefore will improve our understanding of network robustness. This in turn can create insights relevant to designing networks for (in)stability.

A similar argument can be used to provide a new way of looking at interventions in the propagation process. It is argued that interventions can be linked to specific characteristics of the mechanism. Describing the propagation mechanism using multiple characteristics therefore can result in a broader set of parameters that can be tuned to steer the propagation dynamics. This results in a wider set of interventions. Furthermore as such

interventions can be directly linked to what happens during the propagation process this can make interventions more specific and targeted. It will provide decision makers with a richer toolbox for steering propagation.
Consequently, capturing the impact of the mechanism of propagation can facilitate a better understanding of robustness of networks and interventions in the propagation process. Furthermore, doing so puts the impact of the other drivers of propagation —the seed of infection, and the network structure— in a different light, allowing for a more integral perspective on propagation dynamics.

The information processing view of propagation

Much like the logic adopted by Louis Pasteur, this view adopts a micro-level view of propagation. Pasteur, in an attempt to understand how persons could get each other sick, focused on the transfer of germs between them. He considered the disease spreading between actors, in a dyad, and build on the idea that one needs to understand this mechanism at the micro-level before one can understand the impact it will have on the macro- or system-level. Similarly in this dissertation it is argued that only by considering the micro-level mechanism of propagation one can understand the propagation dynamics at the system-level.

Describing the mechanism of propagation requires one to open the black box of propagation, and consider what is actually going on during the process. Therefore, in this dissertation the information processing view of propagation is adopted. This view describes the propagation mechanism on the micro-level as an information processing process. It describes how information regarding behavior or state of one actor is transferred to a connected neighbor and consequently affects the state or behavior of this neighbor. In information processing four elements can be identified; a sender, the information signal, a medium and a receiver (Shannon, 1948). Each of them has a clear role in the processing of the information. The information signal, or shortly signal, is what is being propagated. In order to do so the sender needs to send out the signal, a medium needs to facilitate the transport of this signal, and a receiver needs to process the signal. These three steps are a necessary condition for information about a change in state or behavior to result in a (potential) change of state/behavior of the neighbor. Following this logic, the propagation process can also be decomposed into these three steps.
Hence, building on this notion, the information processing view of propagation argues that the propagation process is composed of three sub-processes:

- Radiation: The focal actor broadcasts a signal to all (or part of) its neighbors
- Transmission: The signal is transported over the tie towards the connected neighbor
- Reception: The incoming signals are processed by the neighbor (potentially) resulting in a change of state/behavior

Characterizing the propagation process in terms of these sub-processes captures what happens during propagation, it describes the mechanism by which the propagation process takes place. Decomposing propagation into three sub-processes not only allows for describing this mechanism, it also provides a more detailed description of the propagation dynamics than traditional models, as they usually only describe the dynamics by a single parameter. Therefore the information processing view of propagation adds an additional level of detail to models of propagation. Additionally, the sub-processes used to describe the propagation mechanism, capture the micro-level behavior during propagation, which directly relates to the actions of actors. Therefore, adopting this view enables linking propagation behavior directly to actor behavior.
The information processing view of propagation, by describing information processing on the actor level, captures the mechanism of propagation. By doing so it can provide a more detailed insights into the propagation dynamics, raising the fundamental question how information processing on the actor level might change the way in which propagation dynamics occurs. In untangling the impact of the mechanism on propagation dynamics the first research question addressed in this dissertation is:

> *(How) do differences in the information processing at the actor level affect system-wide propagation dynamics?*

While the mechanism of propagation can be characterized using the information processing view of propagation, considering the information processing characteristics in isolation is not sufficient. As the network structure and the seed of infection are also drivers of propagation dynamics, all propagation dynamics will be the result of an interaction between these drivers. Consequently a comprehensive understanding of the propagation dynamics requires insight into the interactions between the different drivers.

It is generally accepted that the network structure serves as the infrastructure on which propagation can take place, hence it provides a set of constraints for the propagation dynamics. Extensive knowledge on how the network structure affects these dynamics has been provided in previous studies (e.g. (Watts and Strogatz, 1998; Albert et al., 2000;

Pastor-Satorras and Vespignani, 2001a; Moreno and Vzquez, 2003; Dodds and Watts, 2005; Goldenberg et al., 2009)). However, the knowledge on the impact of network structure is based on a simple representation of the propagation mechanism. As the information processing view of propagation suggests that the mechanism of propagation plays a pivotal role in determining dynamics, this raises a question with regard to the extent to which these findings hold and can be generalized. Therefore the second research question in this dissertation is:

> *How does information processing at the actor level affect the effects of network structure on system-wide propagation dynamics?*

This question explicitly considers the interaction between the network structure and process mechanism as drivers of propagation dynamics. However, also the seed of infection needs to be considered. The seed of infection determines which part of the network structure is available for propagation during the early stages of propagation. The earlier stages of the propagation process are crucial in determining local dynamics and consequently the cascading potential; The local structure surrounding the seed can cause variations in the local dynamics and hence result in different propagation dynamics on the system level. Therefore the seed of infection not only plays a vital role in determining propagation dynamics, but also draws attention to the impact of local variations.

It has been observed that the local network structure (selected due to a varying seed of infection) plays a crucial role in determining the local dynamics, and consequently affects propagation dynamics on the system level (Stonedahl et al., 2010). A similar claim can be made for variations in local dynamics caused by heterogeneity in actor behavior. Actors are inherently different, and hence will behave differently. Therefore, it is plausible to assume that the information processing behavior will differ across actors as well, and that such a heterogeneity will have an effect on system-wide propagation dynamics.
While some research exists addressing actor heterogeneity (Goldenberg et al., 2001; Rahmandad and Sterman, 2008; Young, 2009; Jackson and Lopez-Pintado, 2013), their findings seem to be inconsistent. On the one hand there is work claiming heterogeneity of actors has little effect on propagation dynamics (Rahmandad and Sterman, 2008), while other work claims such an effect does exist (Jackson and Lopez-Pintado, 2013). It should be noted that a simple propagation mechanism underlies these studies, which might explain the inconsistency of the conclusions. As the information processing view describes the information processing on a actor level, it is particularly well suited to assess the

impact of heterogeneity in actor behavior on propagation dynamics. Therefore the third research question in this dissertation will be:

> *What role does heterogeneity of actor information processing behavior play on propagation dynamics?*

1.1 Research methodology

This dissertation adopts a multi-method methodology consisting of:

- A literature study and the development of a conceptual framework of the propagation process
- An empirical validation of this framework using field data
- A multitude of simulation studies focusing on explaining propagation dynamics.

This buildup enables a rich understanding of a wide range of propagation processes, and helps answering the posed research questions in a structured way.

First, building on a multidisciplinary body of literature on propagation, a new framework for propagation is put forward. This framework incorporates the information processing view of propagation and decomposes the propagation mechanism into three sub-processes; Radiation, Transmission and Reception (RTR). This framework is then translated into a new mathematical model of propagation and compared to existing propagation models.

Second, data of Yahoo! Go 2.0 adoptions (Aral et al., 2009) is used to validate the newly proposed model of propagation. This field study is extended by translating the mathematical model of propagation into a Agent-Based simulation model. This Agent-Based Model is then used to study the impact of interventions.

Third, leveraging the Agent-Based Model —which allows for studying propagation in a wide range of settings (in terms of various propagation mechanism and various network structures)— more complex propagation scenarios are studied by introducing network structure and actor heterogeneity in the simulation. In two separate studies the impact of respectively the heterogeneity in actor behavior and heterogeneity in the network structure on the propagation dynamics are considered.

1.2 Contribution of this study

The contribution of this dissertation can be divided into four areas:

- A more detailed conceptual framework and simulation model of propagation using the information processing view
- An increased understanding of intervention effectiveness in propagation
- A nuanced notion of the effect of network structure on propagation dynamics
- An increased insights in the effect of actor heterogeneity on propagation dynamics.

A new framework of propagation is introduced which allows for a more detailed way of considering the mechanism of propagation. Current literature on propagation considers the dynamics mostly at the system level by means of mean-field approaches. This research however puts forward the notion that in order to understand the dynamics on the system-level one first needs to understand the micro-level behavior. The information processing view considers the propagation mechanism at the actor level and by doing so allows for describing the micro-level behavior during the propagation process.

The dissertation increases our understanding of effectiveness of interventions in propagation processes. It shows that the effectiveness of interventions is conditional upon the mechanism of propagation. Therefore effectively intervening in the propagation process requires a detailed view of the propagation mechanism. As the information processing view provides such a view, it links this framework directly to intervention effectiveness and an increased grip on propagation dynamics by managers and policy makers. Consequently, decomposing the propagation process into sub-processes results in a detailed and a more targeted set on interventions, of which the effects can be predicted better.

The findings should stimulate researchers to reevaluate the effects of network structure on propagation dynamics. Considering the information processing at the actor-level shows that the network structure does not have a single consistent effect on propagation dynamics. Instead the effect of network structure is shown to be conditional on the mechanism of propagation. While for one process a structural element might facilitate propagation, the same structural element can have a dampening effect in another. Such observations suggest that the effect of network structure cannot be seen without the context of the propagation mechanism, and hence a more careful consideration of this interaction effect

is needed.

This research also contributes to our understanding of the effects of actor heterogeneity. By considering information processing on the actor-level, the information processing view allows for capturing the effects of actor behavior (and heterogeneity in this behavior). It is shown that the effects of heterogeneity are less straightforwards than previously assumed, and heterogeneity in different parts of the propagation mechanism affects the propagation dynamics in different ways.

The information processing view of the propagation, and the model capturing this view: the RTR-model of propagation provide a more detailed and realistic view of what drives the propagation dynamics. Given the multidisciplinary nature of the propagation process these contributions are applicable to a wide range of fields. The flexibility in the information processing framework allows it to be applied to a wide range of propagation settings, which can facilitate comparison among models and settings and hence can result in more interdisciplinary knowledge spill-overs with regards to propagation. In doing so this research contributes to the theory of propagation in its most general form. The lessons learned from this dissertation can, for example, be applied to improve intervention effectiveness in disease spreading studied in epidemiology, to improve targeting strategies for word of mouth or product adoption studied in marketing, to better understand the critical mass effects in the spread of innovation studied in business, to improve the understanding in ecosystem stability and the interactions between species studied in ecology, to improve network robustness towards cascading behavior studied in engineering or better understand the spread of information studied in business and informatics.

1.3 Structure of this dissertation

This dissertation consists of seven chapters. This first chapter has introduced the research topic, focus and research questions. The second chapter consists of a conceptual study, covering the concepts of propagation, networks and propagation dynamics in more detail. It will consider existing models of propagation and introduces the model incorporating the information processing view; the RTR-model of propagation. The third chapter provides the conceptual framework of this dissertation, introducing three distinct studies, each covering a chapter. The fourth chapter consist of the first study which focuses on the effect of differences in the propagation mechanism, the validation of the RTR-model and showcasing its relevance for interventions. The fifth chapter covers the second study on the

effects of heterogeneity in actor behavior, and the sixth chapter consists of the third study on the effects of the network structure. Finally, in the seventh chapter the conclusions, key findings, contributions of this research are synthesized, the limitations are addressed, and directions for future research are proposed.

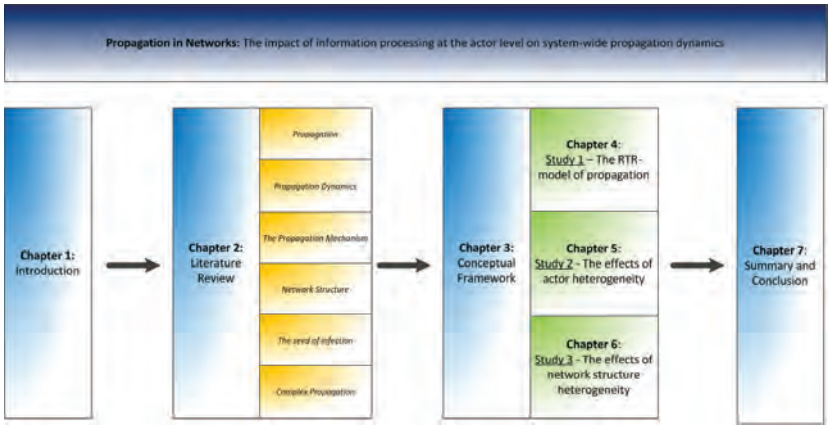


Figure 1.1: Graphical representation of the structure of this dissertation

Chapter 2

The concept of propagation in networks

This chapter considers the main concepts used in this dissertation: propagation, propagation dynamics, and network structure. Relevant literature is reviewed with respect to propagation dynamics. The chapter is structured as follows; the first section considers the concept of propagation and its dynamics. The second section focuses the propagation mechanism. The third section describes the network structure. The fourth section will focus on local heterogeneity. The fifth section will consider how these drivers of propagation interact.

2.1 What is propagation?

The term propagation stems from the Latin word *propagat* which according to the Oxford dictionary means 'multiplied from layers or shoots', and refers to the process by which new shoots grow from the parent plant. It hence refers to the process by which the plant evolves, and extends its presence. Other derivatives of the word *propagat* are *propeller*, which describes the tool used for moving a craft from one location to another, and *propaganda*, which means 'that which should move forward'. Both describe something moving from one location to another.

Similarly the term '*propagation*' is used to describe the process by which something moves from one place to another. It is used primarily in physics where, for example, under the label 'wave propagation' it describes the motion of a wave throughout a medium or the transfer of its energy. Under the label of 'crack propagation' it describe the motion of the crack tip or the crack front during the fracture of materials. As 'radio propagation' it refers to the behavior of radio-waves when they are transmitted, or propagated from

one point on the Earth to another. Propagation thus generally refers to a process of displacement; the spread of something in place and/or time.

The term propagation is not very commonly used outside the field of physics to describe such spreading processes. Phenomena fitting the description of propagation are however studied by scholars in many different fields. Throughout literature the propagation process has received different names based on the context in which is has been studied. Most commonly cited notions are those of contagion (of disease) (Dodds and Watts, 2004; Rahmandad and Sterman, 2008), social contagion (Burt, 1987; Galaskiewicz and Burt, 1991; Iyengar et al., 2011; Aral, 2011), social influence (Turner, 1991; Cialdini and Goldstein, 2004; Friedkin, 2006; Ma et al., 2014), cascading process (of disasters) (Buldyrev et al., 2010; Buzna et al., 2006; Watts and Dodds, 2007), diffusion of innovation (Coleman et al., 1966; Rogers, 1995; Guler et al., 2002; Valente, 1996, 2005) and information diffusion (Aral et al., 2007; Reagans and McEvily, 2003). While these notions do differ in the way they describe propagation, they all describe a process by which interaction among actors causes some sort of signal to flow from one place or person to another, and hence fit the general description of propagation.

In describing propagation four basic elements can be identified: a *source*, a broadcasted *signal*, a *medium* which transports this signal, and a *receiver* of the signal. For propagation to occur the sender will broadcast a signal. This signal can be anything ranging from energy, information, to a change in state or behavior. This signal is then transported through the medium to another place in the system where it eventually has an effect on the receiver(s). The labels 'sender' and 'receiver' indicate that there is some sort of behavior associated with propagation. While both the sender and receiver are expected to show behavior or act during propagation, for the purpose of this dissertation they are referred to as actors. Actors can be anything ranging from molecules, as is the case in the previously introduced examples of propagation, to people, firms or even species depending on the granularity of the process considered.

Borgatti (2005) defines propagation as a flow in a network of interacting actors. By doing so Borgatti (2005) explicitly mentions a network facilitating the propagation process, effectively stating that the network of interactions serves as the medium for transporting signals. This work implicitly argues that propagation is a network process, and should be defined as such. Following this notion, the propagation process is, for the purpose of this dissertation, defined as:

Definition 2.1. *Propagation: The process by which the (change in) state or behavior of one actor results in a change in state or behavior of one or more of its connected neighbors*

As the behavior of neighbors changes this can consequently propagate towards the neighbors' neighbors, and so on and so forth, until potentially the whole network is affected.

Apart from describing the propagation process as a network process Borgatti (2005) makes another contribution. In his paper he makes a clear distinction between two types of propagation; on one hand flows that occur in the form of a (physical) transfer from actor to a connected neighbor (the alter), and on the other hand flows that occur as duplication.
In the case of transfer the original owner is left empty handed after propagation, and hence the 'signal' being propagated is changing physical location, think for example of moving a box. The box can only be in one location at any time, so in order to move the box to another location it needs to be removed from the current location.
During duplication this is not the case, here the signal can in fact be at different locations at a single point in time. Therefore it does not need to leave the point of origin during propagation. An example of duplication could be knowledge sharing. The knowledge does not disappear at it's origin when it is transferred, it remains at the initiator while the signal (the information regarding this knowledge) moves towards to alter. In turn this signal can result in the duplication of the behavior or state at the alter, the neighbor becoming knowledgeable as well.
In this dissertation the focus is on propagation processes which take place by means of duplication. It aims to understand phenomena which have the potential to cause system wide change, which can cause emergent behavior. Such changes require large parts of the system to be affected simultaneously, and therefore consider a process which can have an effect on multiple actors at the same time. Such a spread throughout the system can only be achieved by the propagation processes which occur by means of the mechanism of duplication (Borgatti, 2005).

2.1.1 Propagation of shocks

The definition of propagation adopted is deliberately very broad. Propagation phenomena occur in a wide range of fields, this definition of propagation is designed to fit such a wide range of settings. It can cover any process in which interaction among actors occurs, even though not every process is equally relevant to study.
Strictly speaking any actor interaction affects this actor, be it only on a molecular level.

Therefore any interaction is likely to yield some sort of propagation. However not every interaction will have an impact which is significant. Therefore not all propagation dynamics might be equally relevant to study. For example, in order to understand the emergent behavior in groups of people it might not be relevant to consider the molecular change for each person. This suggests that some threshold exists for which kind of propagation effect should or should not be considered. The choice for such threshold will always be arbitrary and dependent on the phenomenon being studied, therefore this dissertation refrains from implementing an explicit threshold.
Rather than implementing a strict threshold or studying all propagation dynamics it will focus on the propagation of shocks. Note that while a shock has the connotation of being a negative and harmful event this is not the meaning of a shock in this dissertation. For the purpose of this dissertation a shock is defined as:

Definition 2.2. *A Shock: A rapid, unexpected and large change in the state or behavior of one or more actors in the network.*

In this definition 'rapid' implies that the time it takes for the shock to propagate is at least an order of magnitude smaller than the time it takes actors to strategically absorb such signals, therefore when considering the propagation of shock the actual propagation process is separated from strategic behavior actors might have. 'Unexpected' implies that being subject to a specific shock cannot be foreseen by an individual, and hence no last minute counter-measures can be taken to absorb such a shock. 'Large' implies that the behavior or state of an actor is significantly different than it was prior to the shock. Based on these characteristics studying propagation of shocks has three advantages; 1) The effects of shocks are likely to be seen on a relatively short time-span, 2) the effects of shock propagation are likely to be profound, potentially catastrophic, making the effects of propagation more easily identifiable and 3) strategic behavior of actors plays no role in the dynamics of the propagation process in the short term.

2.1.2 Existing models of propagation

Studying shock propagation in a system requires some sort of model to capture the behavior of the process. As literature on propagation is quite dispersed across disciplines this body of work has yielded a wide variety of models. They can be classified into four main model categories; SIS/SIR models (Kermack and McKendrick, 1927), Bass(-like) models (Bass, 1969), threshold models (Rogers, 1995; Valente, 1996) and cascade models (Kempe et al., 2003). A brief description of each of these models is provided below.

SIS/SIR models

By far most models of propagation are based on SIS/SIR type of models. These model stem from the field of epidemics. They owe their name to the states of actors in the model. In these models actors are either **S**usceptible, **I**nfected or **R**emoved, hence resulting name SIS/SIR model. These models describe the process by which actors are changing from Susceptible to Infected by means of an infection rate (λ) and a process by which actors change from Infected to Susceptible/Removed certain recovery rate (ρ) or death rate (γ), these processes are considered to be stochastic and dependent on the interactions among actors.

Even though many extensions of the traditional SIR/SIS models exist (most of them add extra state to the model, for example by allowing actors the be exposed, or temporary immune) for convenience the simple SIS model will be elaborated in order to introduce this type of propagation model. In this model the population is divided in different compartments of actors, each with a different state. These compartments interact at a certain rate which is based on the size of the population in each of the states available in the system. By considering the average rates by which the actors change from one state to another, such models allow writing the dynamics of propagation as a set of differential equations. Assume a set of N actors which are either susceptible (S) or infected (I) such that $N = S + I$. In this case the SIS model is described by:

$$\frac{\Delta I}{\Delta t} = -\rho I + \frac{\beta S I}{N} \tag{2.1}$$

$$\frac{\Delta S}{\Delta t} = -\frac{\beta S I}{N} + \rho I \tag{2.2}$$

In which β is the rate of interaction.

This model assumes random interactions among actors, essentially considering a scenario with homogenous mixing. More complicated extensions have been developed which include the notion that there is a network underlying the propagation process. The network causes actors to have a certain amount of connections, which affects their ability to both infect others, and be infect by others, resulting in the following formulation Pastor-Satorras and Vespignani (2001a):

$$\frac{\Delta I}{\Delta t} = -\rho I + I\langle k\rangle\lambda(N - I) \tag{2.3}$$

$$\frac{\Delta S}{\Delta t} = \rho I - I\langle k\rangle\lambda(N - I) \tag{2.4}$$

In which $\langle k \rangle$ is the average number of edges (connections) per vertex (actor).
A more complex variation of this model can also capture heterogeneity in the network structure (Pastor-Satorras and Vespignani, 2001a,b). What these models do is assign actors to a certain group, a compartment, based on their state and number of connections. It is assumed that all actors in the same compartment behave similarly. This model leverages the average behavior of actors in a compartment, for this reason it is an example of a mean-field model. Mean-field models can be roughly divided in three camps (Pastor-Satorras et al., 2014):

- Individual-based mean-field approach (IBMF): In which it is assumed that every actor belongs to compartment of the system with a certain probability, and that all actors within a compartment behave similarly.
- Degree-based mean-field approach (DBMF): Here compartments are based on the number of ties an actor has. It is assumed that every actor with the same number of ties, its degree, behaves statistically similar.
- Generating function approach: A special case method for scenarios in which infected actors are removed after infection. Which builds on the notion that the likelihood of finding a tie is related to the probability of transmission of the disease, which is constant for the complete population.

Pastor-Satorras et al. (2014) provide an mathematical representation of these models and an extensive overview of their characteristics and differences. Each of these approaches shares the same leveraging mechanism, they consider the propagation behavior averaged over a groups of actors or the system as a whole. The main argument for doing so is that this allows these models to step away from the apparent chaotic behavior of the individual actor level (NWO, 2014). Also combining set of actors, significantly reduces the complexity of the formulation of the propagation behavior. Because of this these so-called mean-field approaches enable using an analytical methodology to untangle the propagation process. This in turn has resulted in closed form solutions to many propagation problems Pastor-Satorras et al. (2014).

Cascade models

A second type of propagation models is the cascade models (e.g. Goldenberg et al. (2001)). This type of models similar to the SIS models considers a stochastic process in which an 'infected' actor will propagate its behavior towards it connected neighbors. Potentially resulting in the occurrence of cascades of such behavior. An important characteristic of

cascade models is that they are build on the notion of momentum. This means that once an actor changes state, it will sent out a signal towards a neighbor only once! Regardless of this result of the consequent propagation process it loses its momentum after this initial shock and will become inactive as a result.
As Kempe et al. (2003) puts it '*The basic cascade model therefore can be described by a process which starts with an initial set of active I_0 actors. The propagation process unfolds in discrete steps according to the following randomized rule. When node i first becomes active in time step t, it is given a single chance to activate each currently inactive neighbor j; it succeeds with a probability $p_{i,j}$ —a system-wide parameter— independently of the history thus far*'.

Threshold models

The third type of propagation models, the threshold models (e.g. (Valente, 1996)) assume a different type of propagation mechanism. These models assume that adoption of a certain state is a consequence of the states of its connected neighbors. In each time step all actors will therefore reconsider their state. Each actor will look at its direct neighbors and change state if a large enough proportion of the neighbors has adopted an alternative state. Whether the proportion of the neighbors is big enough to change state, depends on the adoption threshold of the focal actor.
Kleinberg (2007) describe the most basic threshold model, the linear threshold model, in which it is assumed that each actors has an individual threshold for changing its behavior. For each actor the proportion of neighbors needed differs, the threshold is chosen randomly. The model consists of two elements:

- A set of edges E with a positive weight w_{ij} on each edge from i to j, which indicates the influence of actor i on j . It is dictated that $\sum_{w \in N(j)} w_{ij} \leq 1$, where $N(j)$ is the set of nodes with edges to j.
- A set of thresholds Θ, containing a threshold θ_i for each node i. θ_i is chosen uniformly at random from $[0, 1]$

Due to some external event a set of nodes will initially adopt the alternate behavior, these nodes are claimed to be active. At any discrete time step $t = 1, 2, 3, \ldots$ any inactive node j becomes active if the fraction of active neighbors exceeds its threshold:

$$\sum_{A \in N(j)} w_{ij} \geq \theta_j \tag{2.5}$$

Where A is the set of active nodes. While far more complicated threshold models can be considered, for example by considering also the absolute number of neighbors (Kleinberg, 2007; Centola and Macy, 2007) all threshold models follow this fundamental mechanism of updating the state of actors based on the state of the connected neighbors.

Bass-like models

The traditional Bass model (Bass, 1969) was used to describe the adoption of innovation among actors. It consists of a differential equation describing the process of how such adoption occurs. The basic premise of the model is that adoption is driven by two forces, random adoptions, and influence from other actors. Each actor is classified as innovator or as imitator. Innovators are those which have a high probability to adopt a innovation at random, and imitators are those which are more likely to adopt due to influence. The speed and timing of adoption depends on their degree of innovativeness and the degree of imitation among adopters.
The basic Bass model is formulated as:

$$\frac{f(t)}{1-F(t)} = p + qF(t) \tag{2.6}$$

In which $f(t)$ is the change in the proportion of adopters, $F(t)$ is the proportion of adopters, p is the rate of innovation (random adoption) and q is the rate of imitation.
The Bass model can also be written as a discrete time model (Lilien et al., 2000), in which case it is formulated as:

$$x(t) = \left[p + q(\frac{X(t-1)}{m})\right][m - X(t-1)] \tag{2.7}$$

In which $x(t)$ is the number of adopters at time t, $X(t-1)$ is the cumulative adopters before time t, p is the coefficient of innovation, q is the coefficient of imitation and m is the number of eventual adopters. This version of the model has been extended to the generalized bass model (Bass, 1994), which captures the effect of marketing effort by including a multiplication factor $Z(t)$. resulting in the following formulation:

$$x(t) = \left[p + q(\frac{X(t-1)}{m})\right][m - X(t-1)]\, Z(t) \tag{2.8}$$

In which $Z(t)$ is oparationalized as:

$$Z(t) = 1 + \frac{\alpha\,[P(t) - P(t-1)]}{P(t-1)} + \beta \max\left\{0, \frac{[A(t) - A(t-1)]}{A(t-1)}\right\} \tag{2.9}$$

In which α is the coefficient for increase in diffusion due to price decrease, $P(t)$ is the price at time t, β is the coefficient for increase in diffusion due to marketing, and $A(t)$ is the advertising at time t. Again, many other variations of the Bass model have been proposed, but the overall form and dynamics remain the same. All these variation propose some way in which the initial bass model will be perturbed, and hence can be seen as models in which the operationalization of $Z(t)$ is changed.
It should be noted that the Bass model does not mention a structure of interactions, and hence this model ignores the impact of the network structure. It effectively assumes that actors have full information on the state (changes) of actors in the system, which can be translated into assuming a completely connected network, in which each actor is connected to each other actor.

2.1.3 Propagation dynamics

Describing the propagation of shocks highlights the potential of propagation to quickly spread among actors before for strategic action can be taken that absorbs the effects of the propagation. Consequently shock propagation processes have the potential to affect large parts of a system, which can consequently destabilize the system. Following this line of reasoning most models of propagation have adopted a perspective of measuring the impact of a propagation process on the system level. It should however be noted that propagation outcomes do not necessarily need to be considered on the system level. Especially in the process of scaling up, while propagation is turning into a global cascade, the outcomes might be interesting to consider. This suggest that, rather than capturing the propagation outcomes at a specific point in time, the timing/stage of propagation outcomes should be considered. Therefore for the purpose of this dissertation will be referred to propagation dynamics, which capture the propagation outcomes and their evolution in time. The propagation dynamics are defined as:

Definition 2.3. Propagation dynamics: The outcomes of propagation over a certain period of time, in a pre-defined part of the system.

This definition suggest that not only the timing, and evolution of the outcomes are critical, but also that location which is considered matters. While, in theory, any arbitrary part of the system could be selected, selections are often in one of three levels: Micro-, the meso- or the macro-level. The micro-level consider a dynamics in a dyad, the meso-level considers how it grows towards a sub-set of the network —a cluster or quadrant—, and

the macro-level (also called the system-level) considers the system as a whole.

Propagation dynamics not only capture how neighbors affect one another, but also how the system is affected, which actors in a system are impacted, and where such impact came from. Capturing the full complexity of the propagation dynamics therefore requires all three levels to be incorporated. However, such a multi-level notion of propagation dynamics makes it a lot harder to strictly define the dynamics. Therefore often a predefined choice is made on the level at which the dynamics are considered. This choice will be strongly driven by the research question(s) one wants to answer. To provide a comprehensive understanding of the different levels of dynamics the following sections will provide an overview of the levels at which propagation dynamics can be captured.

Propagation dynamics at the micro-level

The definition of propagation describes it as a process which takes place between actors, it's dynamics therefore by definition occur at the micro-level. The propagation dynamics on the micro-level should not be mistaken for the propagation mechanism. The local dynamics describes *whether* propagation has occurred between actors. The propagation mechanism in contrast describes *how* such dynamics are realized. The dynamics are thus the result stemming from the mechanism.
The local propagation dynamics have received relatively little attention in literature. Often it is assumed that there is a single rate or probability, be it the likelihood of transmission, the chance of adoption, the percolation probability or the rate of spreading. This simple notion might very well explain why local dynamics have received relatively little attention in propagation literature, there is simply not much to study about a single parameter. While processes can vary in the extend to which local dynamics occur, little is known about why such heterogeneity in dynamics occurs. Describing the local dynamics using a single parameter obscures the impact of differences in the mechanism of propagation. In this dissertation it is argued that these differences play a critical role in determining the local dynamics, suggesting that more complex view of local dynamics is required. In section 2.3 therefore a nuanced view of the propagation mechanism will be introduced.

Propagation dynamics at the meso-level

On the meso-level propagation dynamics capture how the propagation scales from a process with local impact towards one that impacts the system at the global scale. The main focus at the meso-level is therefore on describing the momentum and direction in which the propagation process is moving. In this dissertation a framework is proposed which synthesizes these motions. By using an analogy of a wave rippling over water, three dimensions underlying the meso-level dynamics can be identified; Strength, Width and Depth of propagation. A schematic overview of these three dimensions can be found in Figure 2.1.

- Strength, considers the extent to which a propagation effect affects the receiver (as compared to the sender). By doing so it captures whether the process is losing or gaining momentum during the process.
- Width, expresses the proportion of neighbors which are affected by propagation from the focal actor. By doing so it captures the direction in which the process is dispersing.
- Depth, describes the distance from the original source at which propagation is currently occurring. By doing so it both captures the rate and the reach of the propagation process.

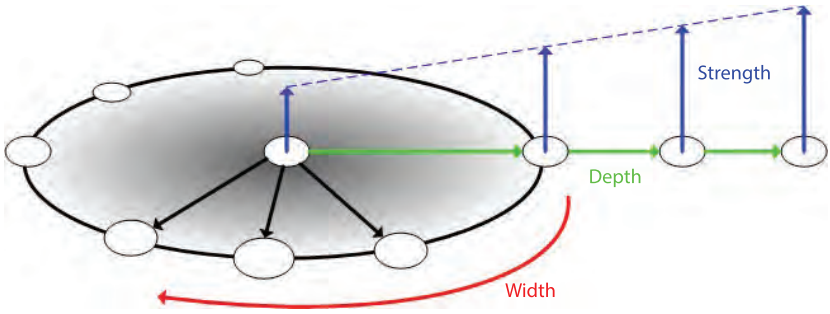


Figure 2.1: The three dimensions of propagation dynamics on the meso-level.

By tracking the propagation dynamics at the meso-level one can get insight in how the process is currently behaving and what kind of momentum it has, it therefore captures the evolution of the propagation process towards system-wide impact. This however does not provide insight into how the system as a whole is being affected by the propagation process. Measuring this kind of effect requires a different, a macro-level, perspective.

Propagation dynamics at the macro-level

Macro-level propagation dynamics capture the effect of the propagation process at the system level. The system-wide impact of propagation has received by far the most attention in literature. Therefore, there are quite some explicit measures capturing the macro-level propagation dynamics. These measures can be divided into three main categories; impact, speed and location.

Impact

The impact of propagation has obtained by far the most attention in literature. All previously introduced models aim at specifying the proportion of the population which is infected, essentially capturing the impact of the propagation process at the system level. The population of infected actors, the so-called prevalence (Pastor-Satorras and Vespignani, 2001a), is a dominant measure of describing the impact of propagation at the system level. It should be noted the prevalence of a process varies over time. In scenarios with an recovery process this results in a basin of attraction(s) for the process. At some point the number of newly infected actors, offsets the number of newly dis-infected actors, resulting in an (dynamic) equilibrium at the system level. The prevalence in this (semi)stable state, can be either positive or zero, indicating whether a propagation process in the long run is viable from prevailing in a population.

Speed

In scenarios without a recovery process there will be no new dis-infections, therefore such equilibrium state will not be present. Consequently rather than studying the extent of impact at any given stage in the process, often the speed by which it occurs is considered. The prime interest is on the time it takes for a process to reach (an arbitrary) critical mass, for example the time it takes for the 90% of the population to become infected.

Location

The last category of macro-level propagation dynamics measures focuses at the location of the propagation effects. The primarily aim for such measures is describing which part(s) of the network are affected (most). Here the system is divided into different sparsely connected groups, clusters, and one considers which clusters are affected and which ones are not.

The categories of system-wide propagation dimensions are not mutually exclusive; hybrid metrics can occur, and are receiving increasing amount of attention. An example of a hybrid method which has seen increasing use (e.g. (Stonedahl et al., 2010; Peres, 2014)) is the net present value (NPV) method put forward by Goldenberg et al. (2007). By assigning a value to each adoption and making this value time dependent —longer

adoption times yield diminishing gains— elements of both the impact and the speed are combined into a single measure.

2.1.4 Three drivers of propagation dynamics

Propagation literature has provided limited insight in what causes propagation dynamics. Generally variations in the propagation dynamics are attributed to one (or more) of three drivers:

- The starting point of the propagation process, the seed of the infection
- The process by which the behavior spreads between actors, the mechanism of propagation
- The structure of interactions in the population, the network structure

When considering the definition of propagation used in this dissertation, one can observe that each of these drivers is represented.
First, the '*connected neighbors*' part of the definition links to the network structure. Having connected neighbors implies some sort of connection among actors. An overview of these connections can be represented in a network or graph, in which the actors are nodes and the connections are edges. The resulting network graph depicts the structure of interactions, which serves as the medium facilitating propagation. As this medium is a necessity for propagation, the network structure capturing this medium provides a boundary condition for propagation, which will consequently affect propagation dynamics.
Second, the definition mentions '*the (change in) state or behavior of one actor*', which can be linked to the seed of infection. Although the definition considers the starting point of the dyadic process and the seed of infection refers to the start of the process as a whole, both suggest that there is a source of propagation. Linking the dyadic starting point to the process as a whole implies taking into consideration the network structure beyond the dyad. The inherent heterogeneity of these structures, is what makes the seed a crucial aspect in system-wide propagation dynamics. Having heterogeneous network structures means that the structure within a network will vary locally, and will be different depending on which part of the network is considered. As this local structure provides the infrastructure which can be leveraged the early stage of the propagation process it will determine the ability for the propagation process to gain critical mass and momentum. As the seed in turn dictates which local structure will be available this seed can strongly influence the process dynamics.
Third, the definition of propagation mentions '*The process by which*' which directly links

to the mechanism of propagation. While the definition does mention the mechanism of propagation, it does so on a high level of abstraction. It does not specify any characteristics of the mechanism, fundamentally treating the mechanism as a black box.

Each of these drivers seems to capture part of what constitutes propagation dynamics, therefore in the following sections each of the drivers will be considered in more detail.

2.2 The propagation mechanism

The notion that the mechanism of propagation is a driver of propagation dynamics has received relatively little attention in propagation literature. It is widely accepted that there is an effect of the mechanism, however this effect is often not explicitly mentioned. It has been recognized that models adopting different description of the propagation process, a different mechanism, can yield different propagation dynamics (Rahmandad and Sterman, 2008), yet the impact of the mechanism has to my knowledge not systematically been studied.

The lack of emphasis on the impact of the mechanism might, in part, be due to the design of mainstream propagation models. It seems intuitive to assume that some processes are more effective than others, and that consequently the way in which one actor affect its neighbor(s) will vary across processes. In line with this logic mainstream propagation models have captured and parameterized the local propagation dynamics, the extend to which propagation among actor occurs successfully. All mainstream models describe a certain rate or probability success —be it the likelihood of transmission, the threshold for adoption, the percolation probability or the rate of spreading— and therefore incorporate and parametrize the local dynamics of propagation. They however also ignore the mechanism by which such dynamics are caused.
Doing so allows for reducing the complexity of propagation models, as local dynamics can be captured using a single parameter. Which has resulted in the conclusion that variation in such a parameter yield very different behavior on the macro-level (Pastor-Satorras and Vespignani, 2001a). However, it also significantly reduces the extent to which one can make claims regarding the impact of the process itself. When a single parameter is used to describe a complex process little detail can be captured, and little insight can be gained on the impact of the process itself. Hence the quest for simplification migth very well be the reason why differences in the mechanism have received little attention in propagation

literature.

Building on management literature it is argued that this lack of emphasis might pose a problem. A commonly accepted notion in management literature, especially in the field of (business) process management (e.g. (Becker and Kahn, 2003; van der Aalst et al., 2003)), is that outcomes in organizations are the result of an underlying (production) process. It is argued that the best way to improve organizational outcomes is to streamline this process. However, in order to streamline the process there needs to be insight into the mechanism by which this process takes place. Therefore this body of management literature implicitly suggests that understanding the underlying mechanism is critical in order to effectively steer and manage its outcomes. In this dissertation a similar argument is made for the propagation process, it is suggested that in order to steer and manage propagation outcomes, its dynamics, one needs to be able to understand and describe the mechanism by which the process occurs.

2.2.1 The propagation mechanism in practice

The definition of propagation captures the propagation process at a high level, without explicitly characterizing the mechanism which makes up this process, effectively considering the mechanism of propagation to be a black box. While this allows for combining terminologies and capturing practical phenomena across different setting, it has little value in terms of understanding the effects of the mechanism. In order to make sensible claims about the effects of the mechanism one needs to open the black box of propagation and provide a detailed description of what happens during the process.

The best way to get a grasp of what happens during propagation is to observe and describe how this phenomena occurs in practice. Propagation phenomena occur in many different fields, which provide a rich source of potential insights. As links between these fields are not necessary strong, it is expected that each setting can yield different insights into the propagation mechanism. Therefore, to explore the behavior during the propagation process a set of six practical propagation phenomena is considered; the spread of influenza, giving a political speech, spreed of financial crisis, a cascade of breakdowns, diffusion of innovation and knowledge sharing.

Propagation in biology - The spread of influenza

The propagation of influenza starts with one (or more) infected individuals. These individuals start spreading germs by air by means of a sneeze. During the sneeze an actor sends out a signal, it releases germs into the air. Note that the number of germs send out during the sneeze, is not necessarily the same as the number which reaches an alter. There will be a certain dispersion of germs which causes the number of germs which reach the alters to be dependent on for example the proximity between actors. while germs reaching an alter is a necessary condition for infection, the alter getting sick will depend on how this alter responds to the germs which reach him/her. The immune system and the resistance of the alter will play a crucial role in determining how this signal (germs which reach the alter) will lead to him/her becoming sick.
Clearly, for influenza to spread a sneeze of the focal actor is not sufficient. Germs will need to travel from the sender to his/her alters, and these alters will need to respond to those germs by becoming sick. This indicates that the spread of influenza takes place in three consecutive steps.

Propagation in sociology - Giving a political speech

In a political speech a speaker aims to convey his/her standpoint to the crowd and convince them to change their standpoint. The behavior which is propagating is therefore the standpoint on a certain matter. This is propagated by sending words (actually soundwaves) towards the crowd. While this signal sent is singular, the manner in which it is received can vary based on the receiver, volume and clarity can vary across different locations at which alters are located. Even when the sounds reaches the alter in a similar manner this signal can be interpreted very different among alters. Messages bear a certain meaning, the extent to which such meaning will be deduced strongly depends on the understanding of the recipient. Understanding a message still is no guarantee the alter will change it standpoint, the interpretation and existing social context will affect the alters response to the new information.
Giving a political speech is a good example of a phenomena which captures social contagion. This process has many similarities to the mechanism observed in the contagion of disease; a signal is sent, transmitted and received. The main difference however lays the fact that there is a social context in which signals are received. While in disease spreading the signal being received (number of germs/virus load) is a tangible (although very small), in scenarios of social contagion this is not the case. This requires an additional step of

sense-making while processing signals.

Propagation in economics - Spread of the financial crisis

The financial crisis has recently received a lot of attention of scholars. It is often claimed to be a contagious process; A financial institution becomes insolvent (due to some external circumstance) and hence is legally required to obtain additional funds. It can do so by selling fixed assets, borrowing additional money or defaulting on a loan. Each scenario will be briefly considered.

In the first scenario, selling fixed assets, the distressed institution has to take a haircut due to the fire-sale of the fixed assets (accepting a lower sale-price for the fixed asset to liquidate it), effectively reducing the value and market price of that asset. While other financial institutions are likely to have some overlap in assets, such price drops can affect the financial status of these institutions. The market-mechanism in this scenario allows the behavior of the distressed actor (selling fixed assets) to affect the financial state of other actors in the system, which in turn can also become distressed.

In the second scenario, borrowing additional money, the propagation effect is two-fold; not only will the distressed institution (attempt to) borrow additional money, and by doing so send a direct signal to the counterpart of this transaction. Also the market for obtaining loans can be affected similar to the sales of fixed assets. As the distressed institution needs to fulfill its solvency criteria, it will be willing to incur additional costs for obtaining the required money (similar as in the case of haircuts) hence interest can increase. Again such increase in interest can influence the market price on loans, effectively affecting other institutions willingness to borrow/lend. This mechanism is what led to a liquidity freeze in the financial system in which institutions where unwilling to borrow/lend money to/from one another.

In the third scenario, defaulting on a loan, the focal firm lets the counterpart of the loan know that it is in a distressed state. Not only can this information become public indicating that there is an increased risk of transacting with this specific institution. Also, the counterpart of the loan is directly affected by the fact that it will not receive a the prospected income from this loan, directly affecting the financial status of this institution. Often the three scenarios go hand in hand when a financial institution is distressed, this makes propagation of the financial crisis in practice a complex matter to study.

While the details of each scenario differ in them three steps in the propagation mechanism can be identified. The distressed state of an institution results in it behaving out of the ordinary (sending out a signal), which reaches the alters (either directly or by means

of market mechanism), changes their financial state and can in turn render them in a distressed state. This setting is different in the sense that little processing of the signal during reception occurs, there simply is a change in the financial state, whether this makes the institution distressed depends on the existing financial status.

Propagation in engineering - Cascades of breakdown

An example of transport of energy is used to illustrate the cascade of breakdown. In this setting the network is used to transport current, which suggest a transport of physical matter, which by definition is bound by a location. This would suggest that propagation can only occur at one place at a time. It should however be noted that the behavior which is claimed to propagate is a form of breakdown. While such breakdowns are caused by the physical flow previously described, they are not bound to a single location and can occur at multiple places at once.
Consider the breakdown of a power station (due to some external event). As physical flows still need to occur in the network the system will attempt to reroute flows to remain maximally operational. this rerouting changes the remaining flows, which will affect the load at other stations, potentially causing them to overload and consequently break down. Again three steps in the mechanism of propagation can be identified. An station breaks down and stops processing the flow of current, the flows are redirected via other stations, which in turn are faced with a changed load, which might result in them becoming overloaded. This setting is different in the sense that the (change in) physical flow serves as the medium which allows a breakdown to be transferred to the alters.

Propagation in business - Diffusion of innovation

Diffusion of innovation/information is initiated by an actor (a firm or individual) adopting or developing an innovation or a piece of information. Information with regards to this adoption starts being sent out either voluntarily, the actor talks about the innovation/information, or involuntarily, when the adopter starts behaving observably different. By means of interaction other actors in the system will be able to observe this behavior, interpreting this as a signal of adoption. After being subject to such a signal the alter then has to respond by either adopting or not adopting the innovation/information itself. This scenario again clearly identifies three steps in the propagation mechanism. It is interesting to note that the transport of the adoption signal in this setting can both be caused by a push mechanism, in which case it is initiated by the adopter, and pull mechanism in which case it is initiated by the alter.

Propagation in management - Knowledge sharing

Knowledge sharing is a prime example of how propagation is considered in management. In order to map how groups of people behave and learn —be it teams or the organization as a whole— managers have tried to understand how knowledge is shared within (and between) groups. In this process there is commonly an individual who possesses the desired knowledge, this actor can be considered as the starting point of the propagation process. This actor communicates with the remainder of the group, by means of various communication media, allowing a flow between these individuals to occur. While this indicates that something can be shared, it is not knowledge which is flowing between them, the knowledge sharing process is slightly more complicated. Knowledge only exists as an interpretation of information. Therefore, a knowledgeable actor can share with its alters information (a codification of his/her knowledge), but not knowledge in itself. Similarly alters can receive information, but this information needs to be interpreted in order to result in (propagated) knowledge. Knowledge sharing therefore explicitly requires a flow of information but also actors to code and decode information. The extent to which this will effectively occur strongly depends on the capabilities of both sender and receiver.

2.2.2 The information processing view of propagation

In the real world examples of propagation two important characteristic of the propagation mechanism can be observed. First, the behavior which is propagating can be very different from the signal which is flowing between actors. And Second, the mechanism of propagation seems to be taking place in a structure with remarkably steps.

In many of the examples of propagation in the previous section a signal which is actually flowing between actors can be observed. To convince someone of a standpoint (which is the behavior that propagates) actors send out sound-waves, cascades of failures propagate by means of a changing current or load in the network, and spread of innovations and knowledge required a flow of information. This indicates that a 'signal' is transferred during propagation. This signal is often not the same as state or behavior which is propagating. Instead the signal captures some form of information regarding the state or a change in the state. This suggest that propagation is at its core a process by which an information signal is processed.

The real world propagation phenomena also appear to have significant overlap in their mechanism. Each process describes three distinct steps; An actor sends out a signal, this

signal reaches one or more alters and the alter(s) process the signal by (not) changing their state accordingly. This observation is in line with communication theory (Shannon, 1948), which is known for describing the information processing process, which similarly captures these three steps.

Based on these observations it is argued that the propagation process can be considered as an information processing process, and capturing this notion the information processing view of propagation is put forward. This view considers the propagation process to consist of three subsequent sub-processes by which an information signals flow between actors and consequently behavior/states can propagate. Rather than considering one overarching process and describing it propagation dynamics, this view describes the mechanism during propagation by which these dynamics occur.

2.2.2.1 Three sub-processes of propagation

All propagation processes require some sort of trigger, something which makes one actor change state, and consequently act as the seed of the propagation process. The initial change in state is considered to be due to some arbitrary external event. Due to this event the focal actor will change its state and starts affecting its alters. It will do so by sending out information regarding the (change in) state/behavior to those with whom it is interacting. This can be done by broadcasting this information, effectively sending a signal towards neighbors in all directions, or by narrow-casting, providing this information to a specific subset of neighbors. Because signals *can* be sent in many different directions this sub-process of propagation is referred to as the *Radiation*. Note that radiation links directly to the width dimension of propagation dynamics introduced in the previous section. Furthermore, radiation implies that information can be duplicated and sent in multiple directions, therefore it fits perfectly with the duplication Borgatti (2005) notion of propagation.
The next step that needs to be taken is that the 'radiated' signal needs to be transported from the focal actor to its neighbor(s). Think of this as sending a pulse over a wire; The pulse which is sent is not necessarily the same as the one which reaches the end of the wire, some loss or distortion can occur during this process. Similarly the during the transport the propagation signal distortion can occur, some part of the information might be lost or altered, communication theory refers to this as **noise**. The second sub-process of propagation, the process of transporting the signal over a tie (edge), is referred to as transmission.
The third step in the propagation process is referred to the as reception sub-process, it

considers the process by which the incoming information is processed and (potentially) result in the a change of state or behavior of an alter.
A schematic overview of the three sub-processes of propagation is provided in Figure 2.2 and 2.3. Note that none of the sub-processes by itself is sufficient for propagation to occur. In fact, only by going through all the three sub-processes a (change in) state of the focal actor can result in a change in state of one or more alters. Therefore these sub-processes should be considered as sequential processes, only if all three sub-processes are executed successfully there can be propagation.

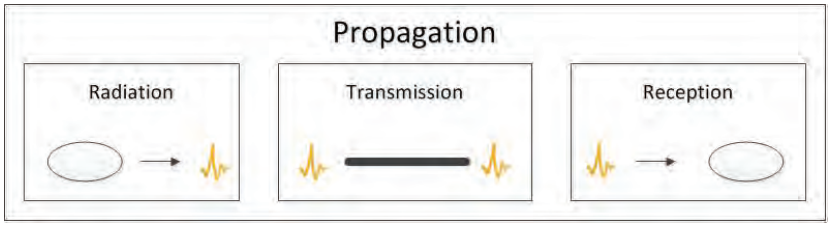


Figure 2.2: The three different steps underlying the propagation process

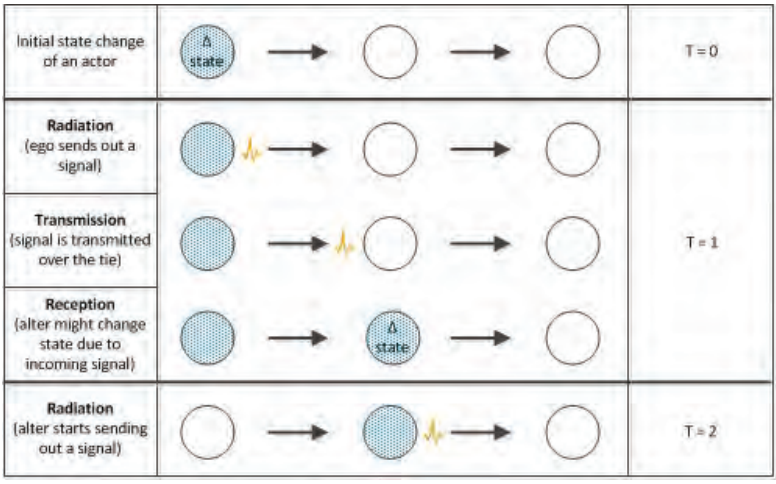


Figure 2.3: The mechanism by which the propagation process takes place

2.2.3 A model capturing the propagation mechanism

The information processing view of propagation describes the mechanism of propagation using three sub-processes; Radiation, Transmission and Reception (RTR). The propagation model designed to capture this view will consequently incorporate these sub-process.

Based on the names of these sub-processes this new model is labeled the RTR-model of propagation.
This model introduces three parameter-sets, one for each sub-process. In combination, these parameters describes the propagation mechanism. These three parameter-sets are claimed to each be embedded in a separate function, each of which describes a single sub-process of propagation. The RTR-model of propagation in its most generic form therefore consists of three distinct functions:

1. a Radiation function
2. a Transmission function
3. a Reception function

This model assumes that there exists an underlying network structure on which the propagation process will take place. This network structure can be considered as a graph $G = (V, E)$ with vertices $i \in V := \{1, \ldots, n\}$ and an edgeset $e \in E := \{1, \ldots, m\}$. All edges are assumed to be directed and weighted, where $w_{ij,t}$ denotes the weight of the edge from vertex i to vertex $j$on time t, and $[W_t]$ is the matrix containing all edge weights on time t. All vertices i at any point in time have a state $s_{i,t}$, and $[S_t]$ is the matrix containing the state all actors at time t ($s_{i,t} \in [S_t]$). The starting state of the system can thus be denoted as $[W_0]$ and $[S_0]$, this starting state is assumed to be stable.

The definition of propagation used in this dissertation considers it to be a process which is initiated by the change in the state/behavior of a seed, one or more actors which change state due to some external event. It can be measured by a change in the state of a set of actors on time $t = 0$, and hence a change in the starting state ΔS_0 ($\Delta S_0 = (\Delta s_{1,0}, ..., \Delta s_{n,0})$). This perturbation in turn can result in propagation and consequently destabilize the system. The term propagation is only used when the state of vertex set V changes during a single time-step in the model ($S_t \neq S_{t+1}$).
The propagation process is assumed to take place in discrete time. In every time unit ($t \rightarrow t+1$) the three aforementioned sub-processes occur. First a change in the state of an vertex i will be sent to the outgoing edges of this vertex in the form of a signal (radiation), second this signal will be transferred over the edge towards the alter (j) (transmission), and third the incoming signal(s) will reach alter j and (potentially) result in a change in the state of the alter j (reception).

The radiation sub-process

The radiation function describes how a change in the state of a vertex i ($\Delta s_{i,t}$) results in signal being sent out towards the outgoing edges of that vertex at time t ($E^{out}_{i,t} \subset E | w_{ij,t} > 0$), it can therefore generally be described by:

$$\Delta s_{i,t} = \sum_{e \in E^{out}_{i,t}} \alpha_{i,e,t} \times p^{out}_{i,e,t} = \sum A_{i,t} \times P^{out}_{i,t} \tag{2.10}$$

In which $A_{i,t}$ is a vector of radiation parameters ($\alpha_{i,e,t}$) of actor i at time t towards its outgoing edges ($e \in E^{out}_{i,t}$) at time t ($A_{i,t} = (\alpha_{i,e,t}) | e \in E^{out}_{i,t}$), and $P^{out}_{i,t}$ is a vector containing the signals sent from vertex i to all outgoing edges ($E^{out}_{i,t}$) at time t ($P^{out}_{i,t} = (p^{out}_{i,e,t}) | e \in E^{out}_{i,t}$). The radiation parameters ($\alpha_{i,t}$) can be influenced by many aspects, it makes sense to consider it as a function of a set of actor specific characteristics, which can consequently amplify or dampen the extent to which a change in state results in radiation.

One should note that this formulation assumes that a change in state results in a signal *only* in the consecutive time unit, and consequently the model considers only the current changes in state and has no memory of what has happened in previous time units. Following the notion put forward in previous work (Dodds and Watts, 2004, 2005) the generalized radiation process can be rewritten to also include memory in the system, effectively stating that a change in state can have an effect beyond the consecutive time unit.

$$\Delta s_{i,t} = \sum_{t'=t-T_{rad}+1}^{t} \sum (A_{i,t'} \times P^{out}_{i,t'} \times (\tau_{rad})^{(t-t')+1}) \tag{2.11}$$

In which T_{rad} is the memory duration for radiation, the number of time units that a change in state will yield signals ($T_{rad} \in \{1, \ldots, t\}$). And τ_{rad} is the memory inflation factor for radiation, the extent to which previous signals are remembered. The memory inflation factor can in theory have any positive value; although values ranging between 0 and 1 are most likely as they refer to a process in which signals are forgotten over time. Values of τ_{rad} higher than 1 suggest that signals have an increasing effect over time, and hence the effect of a change in state will be amplified over time.

This formulation has some build in boundary conditions; it assumes that any fluctuation in the state of i will fully radiate into one (or more) signals to the outgoing edges. As in many propagation models the vertices refer to social entities (which in itself are complex system with some inherent buffering capacity) it can be argued that vertices have some

absorptive capacity for changes in state. Therefore the assumption that all changes in state will result in radiation might be too strict to capture real-life radiation dynamics. In order to make the model more generally applicable the model should able to capture the notion that there can be some absorption in the system. This is done by stating that only if a change in state is large enough to pass the radiation threshold u ($\Delta s_{i,t} \geq u$) there will be any radiation. Including this notion into equation 2.11 yields:

$$\sum_{t'=t-T_{rad}+1}^{t} \sum \left(A_{i,t'} \times P_{i,t'}^{out} \times (\tau_{rad})^{(t-t')+1}\right) = \begin{cases} \Delta s_{i,t} & \text{if} \quad \Delta s_{i,t} \geq u \\ 0 & \text{if} \quad \Delta s_{i,t} < u \end{cases} \tag{2.12}$$

In which the radiation threshold (u) can have any positive value. Equation 2.12 describes the radiation sub-process in its most generic form. However radiation alone is not sufficient to model the propagation process. In order to model the propagation process both the other sub-processes (transmission and reception) need to be defined; therefore in the next section the transmission process will be considered.

The transmission sub-process

The second sub-process of propagation, the transmission process, considers how the signal coming *from* a vertex (i) is transmitted over an edge towards an alter (j). This process can therefore be formulated as follows:

$$p_{i,e,t}^{out} = \phi_{e,t} \times p_{e,j,t}^{in} \tag{2.13}$$

In which $p_{e,j,t}^{in}$ is the signal coming from edge e arriving at alter j at time t, and $\phi_{e,t}$ is the transmission parameter of edge e on time t. Similarly to the radiation parameter, the transmission parameter ($\phi_{e,t}$)can be considered as a function of edge characteristic which influence transmission.

It has been assumed that an edge also has a memory. When referring to an edge memory cannot occur due to abortive behavior (an edge does not describe an entity but a medium and hence cannot act or absorb) there can however be a certain capacity in for the medium. It can be best described by assuming an edge to be a pipeline which has a certain capacity to transport signals. If a signal does not get transported in a previous time unit, the signal should go somewhere. It can be assumed that it disappears (no memory), alternatively one can assume that it (partially) remains in the pipeline. This suggests that the transmission capacity at time t is influenced by previous signals. Including this notion of memory in the transmission sub-process yields:

$$p^{out}_{i,e,t} = \sum_{t'=t-T_{tra}+1}^{t} (\phi_{e,t'} \times p^{in}_{e,j,t'} \times (\tau_{tra})^{(t-t')+1}) \tag{2.14}$$

In which T_{tra} is the memory duration for transmission, and τ_{tra} is the memory inflation factor for transmission. Most existing propagation models do not describe the transmission process and hence implicitly assume a special case in which transmission has no effects ($\phi = 1$, $T_{tra} = 1$ and $\tau_{tra} = 1$). While this assumption in many cases makes sense, in this dissertation transmission is described in this more generic form for two reasons. First, it allows for capturing the effects where the transmission *does* causes signals to be dampened (or amplified), think for example of the inclusion of noise in communication, the notion of package loss in IT networks or the notion of resistive losses in power transport. Second, this formulation allows for easily incorporating the (potential) effects of edge-weights in the propagation model. The edge weights can be easily included in the transmission parameter $\phi_{e,t}$ hence this generic form allows for studying propagation also in weighted networks, something which is more difficult in traditional propagation models.
Equation 2.14 describes the generic formulation of the transmission sub-process, however for propagation to occur still the reception needs to be considered; this is being doing in the following section.

The reception sub-process

The third sub process of propagation, the reception process, describes how incoming signals translate into a change in the state of a vertex, in its simplest form it could be formulated as:

$$\eta_{j,t} \times \sum_{e \in E^{in}_{j,t}} (p^{in}_{e,j,t}) = s_{j,(t+1)} - s_{j,t} \tag{2.15}$$

In which $\eta_{j,t}$ is the reception parameter considering vertex specific attributes influencing the reception process of vertex j on time t, and $E^{in}_{j,t}$ is the set of incoming edges to actor j on time t. Note that this way of modeling assumes that all incoming signals are combined and have one single parallel effect on a vertex. This formulation therefore assumes that there is no discrimination on sources of a signal, a claim which especially in social systems might be dubious. Formulating the generic form of the RTR propagation model therefore requires a slightly more complex formulation. Consider a vector $\Psi_{j,t}$ which contains a set of $\eta_{j,t}$ for each incoming edge ($\Psi_{j,t} = (\eta_{e,j,t}) | e \in E^{in}_{j,t}$), in which all reception parameters

($\eta_{e,j,t}$) are dependent on the source, the edge, where the signal is coming from. This would yield the following formulation for reception:

$$\sum (P^{in}_{j,t} \times \Psi_{j,t}) = s_{j,(t+1)} - s_{j,t} = \Delta s_{j,t} \tag{2.16}$$

In which $P^{in}_{j,t}$ is a vector containing all the signals of all incoming ties of actor j at time t ($P^{in}_{j,t} = (p^{in}_{e,j,t})|e \in E^{in}_{j,t}$). Note that while this model assumes that incoming signal can be added up, however more complex function of aggregating the incoming signals could be used if one has indications that the process being studied has some none-linear dynamics. Also note that this formulation assumes that signals are processed simultaneously, rather than in sequence.

The RTR-model assumes that aggregated signals have one single combined effect on a vertex per time unit. One could make time units infinity small and thus allow actors to respond to (and send out) signals sequentially (rather then in parallel). This is for example done when considering the propagation process as a Markov-chain. Such an approach is however undesirable for three reasons:

- Processing signals sequentially requires an additional set of assumptions; it requires requires a very clear set of assumptions on the sequencing of signals (which in itself is so complex that the treatment is worse than the problem).
- It assumes that there are no interactions among multiple signals, as only one signal can be processed at each time unit. Therefore it will be unable to cope with more complex notions of propagation (Centola and Macy, 2007).
- Sequential processing assumes perfect responsiveness of the system.

While the first two factors can be a simple design choice, the latter poses a bigger problem. Perfect responsiveness assumes that actors are able to observe and act upon any change that occurs instantaneously. Especially in social systems this might not be a correct representation of reality, as it is more likely that it takes time to both to observe and respond to signals. This is suggesting that there is time of signals to aggregate before being processed.

Actors may not only be bounded in the speed of processing, but also in their capacity to do so. The section on the radiation process has put forward the notion that entities are likely to have some buffering capacity, and hence will not convert any change in state towards outgoing signals. A similar argument can be made for the reception sub-process; actors are unlikely to respond to any signal, it needs to be sufficiently large. Indeed such

a notion is the very foundation on which the previously introduced threshold models are build. A generic form of the RTR-model therefore should include such absorptive capacity also in reception sub-process. This is done by incorporating a reception threshold (q) into the reception function in a similar fashion as has been done in the radiation sub-process. Resulting in the following formulation:

$$\Delta s_{j,t} = s_{j,(t+1)} - s_{j,t} = \begin{cases} \sum (P^{in}_{j,t} \times \Psi_{j,t}) & \text{if} \quad \sum (P^{in}_{j,t} \times \Psi_{j,t}) \geq q \\ 0 & \text{if} \quad \sum (P^{in}_{j,t} \times \Psi_{j,t}) < q \end{cases} \tag{2.17}$$

Where q is the reception threshold which can vary from 0 to ∞. The reception threshold q is applied to the aggregate of signals for two reasons. First, the threshold is something which is bound to the entity 'actor' rather than its ties, as it describes an actor's bounded capacity. It is likely the result of entity characteristics, and hence should apply to the entity and not to its edges. One could argue that there may be different susceptibility depending on the source of a signal, which would be a valid claim, this however can already be captured by the vector $\Psi_{i,t}$ capturing the reception paramateres for different edges. The second reason for applying the threshold after aggregation builds on the notion that a small signal might not yield a state change but many of them combined might in fact do so. Capturing this aggregation effect can only be done by applying the threshold after aggregation.

Again, similar to the previous sub-processes also during reception memory can play a critical role. Like most commonly used models of propagation it can be assumed that entities respond to signals independently, and hence signals from the past play no role in the current reception process (no memory). But especially in social systems such claims might not be realistic as actors tend to learn. This suggests that what has happened in the past does in fact matter for their current decisions (memory). Including this notion of memory into the reception function yields:

$$\Delta s_{i,t} = \begin{cases} \sum\limits_{t'=t-T_{rec}+1}^{t} \sum (P^{in}_{j,t'} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}) & , \quad \text{if} \\ \sum\limits_{t'=t-T_{rec}+1}^{t} \sum (P^{in}_{j,t'} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}) \geq q & , \quad \text{otherwise} \\ 0 & \end{cases} \tag{2.18}$$

The generic RTR-model

Equations 2.12, 2.14 and 2.18 describe the information processing view of propagation in its most generic form. It introduces three parameters, one for each sub-process. These parameters describes the propagation mechanism. As it is the mechanism which causes the local propagation dynamics, the combination of the three sub-process captures that which is traditionally captured by a single parameter in traditional propagation models. While this model is far more complex than traditional models it allows for identifying the mechanism of propagation, provides a more detailed description of the propagation dynamics, and is applicable to nearly all types of propagation processes.

The binary state RTR-model

By making additional assumptions the complexity of the generic RTR-model can significantly be reduced. The generic RTR-model describes the state of vertex as a continuous variable. In many propagation settings this might however be a too complex representation of reality. Many studies have assumed a propagation processes in which the state of a vertex can be described by a binary variable, suggesting that actors can have one of two states ($s_{i,t} \in \{0,1\}$). For example when considering the spread of disease in which actors are either sick or not, an actor adopts an innovation or certain behavior or it does not. A binary state version of the RTR-model can be easily obtained by adjusting the reception sub-process and incorporating the notion that any change in state will be of size 1, and that once actors are in state 1 their state cannot further increase their state. The radiation and reception functions in binary state model can therefore be rewritten to:

$$\sum_{t'=t-T+1}^{t} \sum \left(A_{i,t'} \times P_{i,t'}^{out} \times (\tau_{rad})^{(t-t')+1}\right) = \begin{cases} 1 & \text{if} \quad \Delta s_{i,t} \geq u \\ 0 & \text{if} \quad \Delta s_{i,t} < u \end{cases} \tag{2.19}$$

In which u once again is the radiation threshold.
Similarly the reception sub-process consequently can be formulated as:

$$\Delta s_{i,t} = \begin{cases} 1 & \text{if} \quad s_{j,t} = 0 \quad \text{and} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) \geq q \\ 0 & \text{if} \quad s_{j,t} = 0 \quad \text{and} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) < q \\ 0 & \text{if} \quad s_{j,t} = 1 \end{cases} \tag{2.20}$$

As the transmission function does not take into account the state of the vertices it will remain as described in the generic RTR-model. Clearly, studying scenarios in which the only potential change in state does not pass the radiation threshold ($\Delta s_{i,t} = 1 < u$) is trivial as they will never result in propagation. However, to make sure the model mutually exclusive and collectively exhaustive this option is not to be excluded from the model description.

The stochastic binary state RTR-model

Taking a closer look at the propagation literature indicates that all commonly adopted models of propagation consider the propagation process to be stochastic rather than deterministic, as has been done so far when describing the RTR-model. The SIS/SIR models, commonly studied in epidemiology and underlying a large part of the propagation literature, consider a stochastic process and binary states. While the binary state RTR-model previously introduced is deterministic, this version of the model can easily be converted into a stochastic version very similar to the SIS/SIR models.
The binary nature of the state of the vertices makes it trivial to consider the size of a change in state or the size of the signal. What matters is a whether there is a signal or not. From this idea, the step towards considering the probability that such a signal is present is relatively small. Such a switch would effectively change the emphasis of the model from size of the signal (read deterministic) towards the probability that a signal occurs (read stochastic).

In a stochastic version of the RTR-model the radiation likelihood describes the probability that a change in state (which can only be of size 1) will be larger than the radiation threshold, and thus yields any signal towards the outgoing edge(s). This probability is only influenced by a function of the radiation properties ($A^*_{i,t} = f(A_{i,t}) = (\alpha^*_{i,e,t})|e \in E^{out}_{i,t} = f(A_{i,t})$) of the actor sending the signal(i) and the memory in the system. Therefore it can be formulated as:

$$p^{out}_{i,e,t} \sim Bern(p) \quad \text{in which} \quad p = 1 - \prod_{t'=t-T_{rad}+1}^{t} \left(1 - \left(\Delta s_{i,t'} \times \alpha^*_{i,e,t'} \times (\tau_{rad})^{(t-t')+1}\right)\right) \quad (2.21)$$

When the propagation process is assumed to be stochastic the transmission sub-process describes the chance that a radiated signal is transmitted over an edge. This chance

depends on the presence of an incoming signal, a function of the edge specific characteristic ($\phi^*_{e,t} = g(\phi_{e,t})$) and the memory in the transmission. It can therefore be formulated as:

$$p^{in}_{e,j,t} \sim Bern(p) \quad \text{in which} \quad p = 1 - \prod_{(t'=t-T_{tra}+1)}^{t} (1 - (\phi^*_{e,t'} \times p^{out}_{i,e,t'} \times (\tau_{tra})^{(t-t')+1})) \quad (2.22)$$

The reception sub-process in a stochastic propagation scenario refers to the chance that the sum of the incoming signals aggregates into a signal that in fact surpasses the reception threshold. It will consequently can result in a state change of the receiving actor. This clearly depends on the state of the receiving actor (as it need to be able to change state) and a function of the reception parameters ($\Psi^*_{j,t} = h(\Psi_{j,t'}) = (\eta^*_{e,j,t})|e \in E^{in}_{j,t})$) and the memory in the reception sub-process. Rewriting the binary reception formulation (equation 2.19) into a stochastic version yields:

$$\Delta s_{j,t} = \begin{cases} \sim Bern(p) & \text{if} \quad s_{j,t} = 0 \\ 0 & \text{if} \quad s_{j,t} = 1 \end{cases} \quad (2.23)$$

In which $p = 1 - \prod_{t'=t-T_{rec}+1}^{t} \prod (1 - (P^{in}_{j,t'} \times \Psi^*_{j,t'} \times (\tau_{rec})^{(t-t')+1}))$.

The SIS/SIR models can be denoted as a special case of this general stochastic form with two addition assumptions. First, it assumes no memory in the system. Second, it assumes that radiation is caused by the state itself rather than the change of this state (once an actor is sick it has a chance to radiate). Capturing SIR/SIR models by implementing these two assumptions allow us to reduce equation 2.21 to:

$$p^{out}_{i,e,t} \sim Bern(p) \quad \text{in which} \quad p = s_{i,t} \times \alpha^*_{i,e,t} \quad \Longrightarrow \quad (2.24)$$

$$p^{out}_{i,e,t} = \begin{cases} \sim Bern(\alpha^*_{i,e,t}) & \text{if} \quad s_{i,t} = 1 \\ 0 & \text{if} \quad s_{i,t} = 0 \end{cases} \quad (2.25)$$

Combining this with the notion that in stochastic processes the incoming signals are of size 1 or 0 ($p^{out}_{i,e,t} \in \{1, 0\}$) and that the latter case is a trivial as there is nothing to propagate allows for rewriting transmission in these settings as:

$$p^{in}_{e,j,t} \sim Bern(\phi^*_{e,t}) \quad \text{if} \quad p^{out}_{i,e,t} = 1 \quad (2.26)$$

And using the same logic ($p^{in}_{e,j,t} \in \{1, 0\}$) the reception sub-process for the SIS/SIR models can be rewritten as:

$$\Delta s_{j,t} = \begin{cases} \sim Bern(1 - \prod (1 - \Psi^*_{j,t})) & \text{if} \quad s_{i,t} = 0 \\ 0 & \text{if} \quad s_{i,t} = 1 \end{cases} \tag{2.27}$$

2.2.4 Generalizability of the RTR-model

The previous paragraph shows that the RTR-model can be easily adjusted to mimic SIS/SIR like propagation models. However, as previously indicated, there is a variety of models for studying propagation: The SIS type of models (discussed above), bass (Bass, 1969) type of models, threshold type of models (Rogers, 1995; Valente, 1996) and cascade models (Kempe et al., 2003). In this section it is shown that each of model types can be considered as special case of the binary RTR-model.

2.2.4.1 Bass-like RTR-model

Bass-like models (Bass, 1969) consider propagation to be a function of the previous adoption and a default adoption likelihood on a system level. It argues that a focal actors probability to adopt (read reception) is a function of the *amount* of previous adopters. This can be interpreted as a special case of the reception process in which the reception of actors is a function of the signals coming from *all* actors in the network. In order to be able to receive signals from all actors this effectively assumes a fully connected network graph underlying the propagation process.

The Bass model does not differentiate between the three sub-processes and hence does not make specific claims about radiation or transmission. From the way the model is described it can be substantiated that this type of model assumes that the state of all actors in the system is known. Effectively, this implies that any actor in the adopted (infected) state radiates a signal by default and that this signal is transmitted by default as well. Consequently all other actors are 'informed' about this state. In terms of the three sub-processes these Bass-like models hence assume radiation to be a function of the state, and a 100% chance to radiate. It can hence be formalized in a similar form as the SIS model:

$$p^{out}_{i,e,t} \sim Bern(p) \quad \text{in which} \quad p = s_{i,t} \times \alpha^*_{i,t} \quad \Longrightarrow \tag{2.28}$$

$$p_{i,e,t}^{out} = \begin{cases} \sim Bern(\alpha_{i,e,t}^*) & \text{if} \quad s_{i,t} = 1 \\ 0 & \text{if} \quad s_{i,t} = 0 \end{cases} \tag{2.29}$$

In which it is assumed that $\alpha_{i,e,t}^* = 1$.

Similarly the transmission process is the result of the signals radiated and is assumed to always be successful, consequently any signal coming in will result in a signal going out. Transmission can hence be formulated the same way as in the SIS model:

$$p_{e,j,t}^{in} \sim Bern(\phi_{e,t}^*) \quad \text{if} \quad p_{i,e,t}^{out} = 1 \tag{2.30}$$

In which it is assumed that $\phi_{e,t}^* = 1$.

The reception sub process is only in part similar to the SIS scenario. The first part of the bass model considers imitation, the influence of previous adopters. It is a function of the signals incoming, which given the completely connected graph underlying the propagation includes signals from all other actors in the system. This would imply a formulation can be used similar to the SIS model. However, as previously discussed bass-like models also capture innovation, which assumes there is also the default probability of adoption without these incoming signals.
There are two ways in which this secondary effect can be added. First, one could add a extra independent part to the reception function (this is what Bass did in his original model). This would however add an addition parameter to the model, which is undesirable given that there is a more elegant solution. One could also introduce an additional control vertex in the graph. This vertex, like all other vertices, would be connected to all other vertices in the system. By making the reception likelihood for all edges coming from this control vertex independent of the other signals an additional 'random' adoption force can be introduced. The benefit of this approach is that the logic and formulations applied in the SIS setting apply to the Bass model as well, and hence reception in bass-like models can be formulated as:

$$\Delta s_{i,t} = \begin{cases} \sim Bern(1 - \prod 1 - \Psi_{i,t}^*) & \text{if} \quad s_{i,t} = 0 \\ 0 \quad \text{if} \quad s_{i,t} = 1 \end{cases} \tag{2.31}$$

By adjusting the $\eta_{i,e,t} \in \Psi_{i,t}^*$ for the edges coming from the control vertex the influence of the default adoption effect can be varied. One could even vary this across alters,

hence such formulation directly increases the flexibility of the traditional bass-like models.

2.2.4.2 Threshold RTR-model of propagation

The threshold type of models (e.g. (Valente, 1996)) assume a somewhat different type of process dynamics. These models assume that the adoption occurs after a certain proportion of the connected alters have previously adopted. Literature on threshold models commonly limits itself to describing an adoption (read reception) process, implicitly assuming (similar to the previously introduced Bass models) that radiation and transmission will take place by default. Therefore the radiation and transmission functions can be assumed to be the same as in the previously considered Bass model (equation 2.28 and 2.30).
Unlike the previously discussed bass (and SIS) models threshold models are deterministic in nature. While this has no implication for the formulation of the radiation and transmission sub-processes (as they already are assumed to always occur) it does change the way the reception is formulated. This process should be deterministic rather than stochastic. Note that the general model of propagation was in fact deterministic, and hence the binary state version of this model (equation 2.20) describes such a process already. As the formulation of the general model already included the notion of limited susceptibility to signals and hence included a threshold the threshold model is already captured by the binary state version of the RTR-model. The only additional assumption being made is that the reception threshold of each actor (q_i) is a function of its number of neighbors ($E^{in}_{i,t}$)

2.2.4.3 Cascade RTR-model of propagation

The last general type of propagation model, the cascade models (Kempe et al., 2003), is different from the previously discussed models. It can be considered as a special case of the stochastic binary RTR-model. In cascade models an external perturbation is considered which consequently can result in a cascading dynamics. Like in the binary RTR-model this perturbation changes the state of one (or more) vertices to 'active' (or infected) and consequently this change in activity will cascade to all of its alters (read radiate) with certain probability of success. The logic in this process is similar to the one described in the stochastic binary radiation process (equation 2.21). Key assumption in the cascading models is however that a vertex will only try to activate its alters once, effectively incor-

porating the constraint of no memory in the radiation process. Therefore the equation 2.21 can be simplified, and the radiation for a cascade model can be described as:

$$p^{out}_{i,e,t} \sim Bern(p) \quad \text{in which} \quad p = \Delta s_{i,t} \times \alpha^{*}_{i,e,t} \tag{2.32}$$

As the cascade models assume a setting without recovery ($\Delta s_{i,t} \in \{0,1\}$) a constraint should be added to the equation, resulting in:

$$p^{out}_{i,e,t} = \begin{cases} p^{out}_{i,e,t} \sim Bern(\alpha^{*}_{i,e,t}) & \text{if} \quad \Delta s_{i,t} = 1 \\ 0 & \text{if} \quad \Delta s_{i,t} = 0 \end{cases} \tag{2.33}$$

The other sub-processes are not completely neglected in the cascade models. While there is no specific mention of transmission, it is assumed that each signal radiated will lead to a change in state of the alter. Consequently this type of model assumes that both transmission and reception will always occur. Transmission can therefore be formulated as:

$$p^{out}_{i,e,t} = p^{in}_{e,j,t} \quad \text{with} \quad e = (i,j) \tag{2.34}$$

While in cascades models the assumption is that radiation will result in propagation, neglecting the reception sub-process. These models do explicitly mention what happens when multiple changes in state would occur in a alters neighborhood (implicitly suggesting there is some reception sub-process). In such cases cascading models assume that each signal is processed sequentially (in a random order). For reasons discussed earlier (possible amplification, arbitrary ordering) the RTR-model however applies a parallel reception process. As long as signals do not affect one another, as is assumed in the general RTR-model, the reception sub-process of the threshold models and the RTR-model coincide. While the order of calculations is different —each incoming signal separately has a success chance, and then the product of success per signal is taken, versus, combining the signals (taking the product) and then consider the success chance of the combination— the fact that the general RTR-model assumes no distortion during aggregation (simply uses the product) means these functions yield the same results. However, strictly speaking the reception in the threshold models is formalized as:

$$\Delta s_{j,t} = Bern(1 - \prod_{e \in E^{in}_{i},t} (1 - (Bern(\eta_{e,i,t} \times p^{in}_{e,i,t})))) \tag{2.35}$$

The RTR-model - a general model of propagation

The previous sections have revealed that indeed the current propagation models can be linked to the RTR-model of propagation. The micro level dynamics of each of these models can be easily captured in the RTR-model, by imposing addition constraints. Indicating that the RTR-model can be used to reproduce any of these models. Therefore, these models can be considered as special cases of the RTR-model (with some (varying) additional constraints). Which not only suggests that the RTR-model of propagation subsumes these models, but also that is a model which has more flexibility. This flexibility enables it to capture a wider range of (more complex) propagation mechanisms which in turn allows for more realistic modeling of propagation phenomena.

2.3 The network structure

The network structure presents an overview of the interactions within a system. In it the vertices (nodes) are actors, and the edges are the links between these actors. This network structure serves as the infrastructure which facilitates propagation within the system. As such the network structure provides a set of boundaries to how propagation can occur, and by doing so it constrains the propagation dynamics.

On a local scale the propagation dynamics are often considered as the outcomes of the dyadic process, something which happens between two actors. This suggests that to determine local dynamics the presence of a tie provides sufficient information to distill how the dynamics are constrained by the structure. This would be true only when propagation is considered as dyadic process. Looking closely at the mechanism of propagation however suggests that propagation should be considered as a network process, rather than a dyadic one.
The mechanism of propagation identifies three sub-processes; Radiation, Transmission and Reception. The radiation sub-process describes how a shock results in the sending of a signals towards the outgoing edges, effectively making this a one-to-many process. And while the transmission process is dyadic by definition, the reception process once more captures a network effect. It considers how signals from incoming edges result in a (potential) change of state, it therefore describes a many-to-one effect. Both the one-to-many and many-to-one parts of the propagation process show that propagation is a network process. This dictates that studying the dynamics of propagation should be done whilst

incorporating the network structure rather than the dyadic presence.

While it is relatively easy to define what the network structure entails, it describes the interactions among actors, characterizing this structure is a far more difficult task. For this reason it remains hard to strictly determine the impact of the network structure on propagation dynamics. There is a large body of work which does address at least part the impact of network structure. This body of work can be roughly divided into two streams; the first taking a system level perspective and the second taking a micro level perspective.

2.3.1 The system level perspective of network structure

Describing the network structure in the system level is done by making use of network topologies. In his summarizing article on complex networks Newman (2003) identifies a set of four frequently used topologies; random networks, small world networks and scale free networks. These can be defined by means of a set of four characteristics; path length, clustering coefficient, degree-distribution, and in- (out-) degree.

- Path length refers to the number of steps it takes for an actor to reach any actor in the network, therefore this measure describes the width of the network.
- The clustering coefficient describes the extent to which the network can be divided into different groups (based on the structure).
- The degree distribution refers to how the number of degrees is distributed over the actors in the network (the degree refers to the number of ties an actor has), which is an indicator for how homogeneous and dense the network is.
- The degree (distribution) can be further specified in in-degree, which refers to the incoming ties of an actor, and out-degree which refers to the outgoing ties of an actor

Random Networks

Random networks were initially studied by Erdös & Rényi (1959) with the goal of understanding the properties of graphs as a function of the increasing number of random connections. Their method entailed taking a number of N nodes (actors) and creating K edges (ties) by randomly connecting pairs of these nodes with a likelihood of $P(K) = (1-p)^{N(N-1)/2-K}$ (Boccaletti et al., 2006). Consequently (for a large number of N) this resulted in the degree distribution following a Poisson distribution (figure 2.4), with a tail which drops slightly faster than exponential (Newman, 2003). Furthermore,

due to random assignment of ties there will be little clustering in these network and path lengths tend to be long. The previously posed network topologies are frequently related to the underlying evolution process. Clearly in the case of random network the evolutionary process is claimed to be random.
Studying real-world networks however revealed that despite their inherent differences, most of these networks are characterized by the same topological properties. For instance relatively small characteristic path lengths, high clustering-coefficients, fat-tailed shapes in the degree-distributions. All these features make most real world networks radically different from regular random graphs as studied by Erdös & Rényi (Boccaletti et al., 2006).

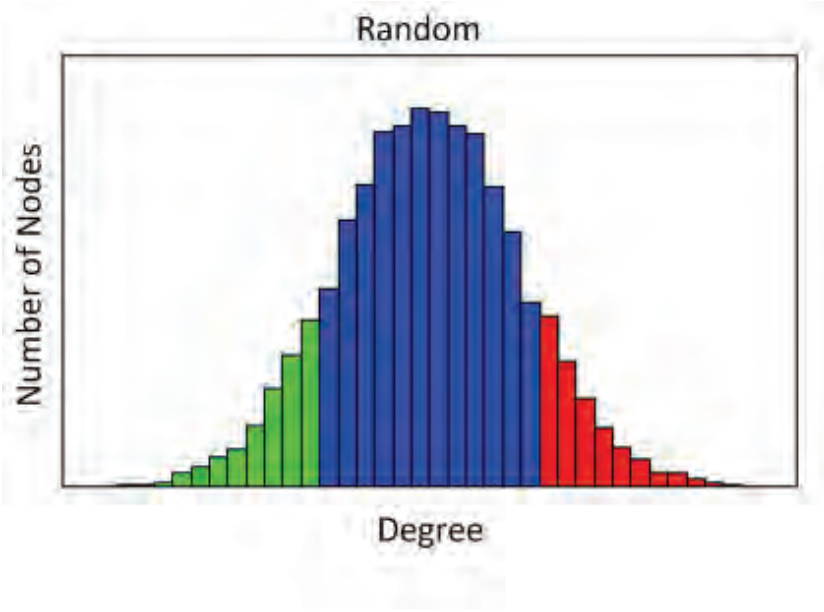


Figure 2.4: The degree (number of connections) distribution of a random network

Small world networks

The small world property refers to the idea that no actor in the network is more than a few steps away, Milgram (1967) referred to phenomenon as 'six degrees of separation'. This phenomenon is recently picked up by some websites such as the "theyrule" (http://www.theyrule.net/) showing that paths between corporate directors and firms are in fact very short. Watts and Strogatz (1998) further developed this network topology by proposing a model which has intermediate randomness in it. They start from a ring

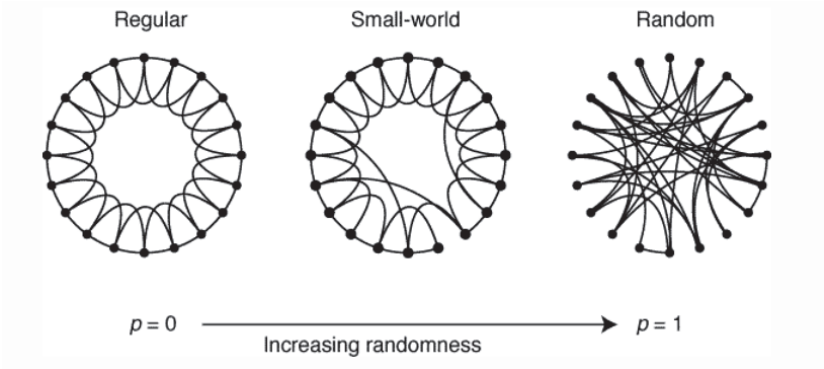


Figure 2.5: The creation of small world networks by increasing the the chance p to randomly rewire an edge. Figure from Watts and Strogatz (1998)

structure in which each node is connected to its $2m$ nearest neighbors with a total of $K = mN$ edges. Each edge then is rewired to a random node with a probability of P (and maintained with a probability of $1 - P$), resulting in a structured network for $P = 0$ and a random network for $P = 1$ (with the constraint that each node has a minimum connectivity $K_{min} = m$) (see figure 2.5). Intermediate values of P result in small world networks with a relatively high clustering coefficient (Boccaletti et al., 2006). In mathematical terms the small world property of networks (short path length) refers to the average node to node distance which is of the order $log(N)$, where N is the number of nodes in the network (Barabási, 2009). While sharing a similar degree distribution random networks lack this characteristic. On average the small world networks hence will be denser than random networks due to a number of boundary spanning links. With regard to network evolution, the small world network is claimed to evolve in a semi-random fashion, including both a random as well as a local attachment effect.

Scale free networks

Many real world networks show a structure of hubs and spokes. The previously introduced topologies are not suitable for capturing such structure. Therefore a third network topology is introduced, which are called scale free networks. Scale free networks refer to networks in which the degree distribution declines with a power law rather than exponentially Barabási et al. (2000). For these networks $P(K)$ decays as a power law, i.e. $P(K) \approx K^{-\gamma}$, free of a characteristic scale (Albert et al., 2000) (see figure 2.6. Studies with regard to the network structure of the world-wide web (www) (Barabási and Albert, 1999; Adamic and Huberman, 2000), Internet, and other large networks (Barabási

et al., 2000, 2001) indicate that many real world systems belong to this class of networks. This topology is further characterized by even shorter path lengths and higher clustering coefficients. The evolutionary process of scale free networks is related to what has been labeled as preferential attachment. This effect refers to the notion that actors entering the network are more likely to connect to other actors which have high prestige, in network terms this would be those which have a high centrality, explaining the hubs and spokes structure.

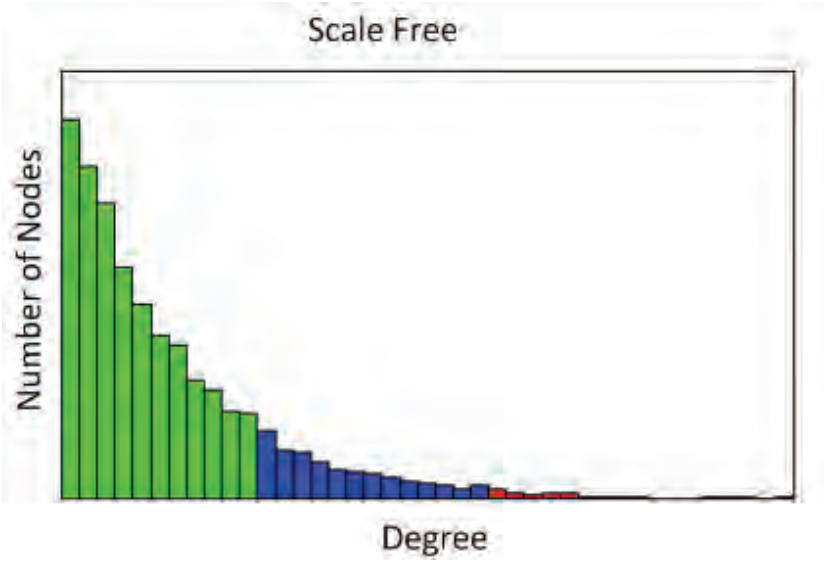


Figure 2.6: The degree (number of connections) distribution of a scale-free network

2.3.2 Topologies and propagation

By comparing among network topologies it has been shown that random and regular networks are poor at facilitating propagation, small-world networks by leveraging the short average path length are much better at doing so (Pastor-Satorras and Vespignani, 2001b; Pandit and Amritkar, 2001). Even more so scale-free networks by leveraging the presence of hubs (highly connected actors) yield the highest propagation effectiveness (Pastor-Satorras and Vespignani, 2001a). It has also been shown that scale-free networks are very robust at doing so (Albert et al., 2000). In the same body of work it has been shown that clustering facilitates propagation within the cluster but not between the clus-

ters(Rahmandad and Sterman, 2008).

These results indicate the relevance of the underlying characteristics of these topologies in facilitating propagation. Both the skewness of the degree distribution, the average path length and the clustering coefficient seems to play a facilitating role for propagation. While this does not yield a full overview of the effects of network structure, it certainly provides proof of the impact the system level network structure can have on propagation dynamics.

2.3.3 The micro level perspective of network structure

An argument can be made that the system level perspective of the networks structure ignores details of the network structure which can be critical in order to understand the propagation dynamics. Therefore the second stream of propagation literature has adopted a bottom-up approach and considers the effect of network structure to stem from local heterogeneity in the network. Local network structure captures network properties from the perspective of the actor. Note that this is not the same as looking at the ego network, but it does take the ego as the starting point of describing the network structure and its effects. The claim is that the characteristics of local network structure can be aggregated to describe properties of the complete network and hence are a fair representation of the network as a whole.

Dyads

A structure connecting two actors, the dyad, can be considered as the smallest level of local network structures. While the propagation literature mention the dyad is relatively limited, there are two fundamental propagating forces at play at this level; reciprocity and tie strength.

Reciprocity which is of interest in especially social network studies (Larson, 1992; Nowak, 2006) measures the extend to which actors respond to one another within the dyad. It suggests that once a flow of information has occurred in a one direction it is likely that at a later stage a similar flow will occur in the opposite direction. Suggesting that history within the dyad can stimulate propagation.

Tie strength (Granovetter, 1973) is a characteristic commonly studied at the dyadic level. It is argued that stronger ties are more likely to yield flow of information (even though the information might be less valuable). By aggregating these local measures one can gain insight into the characteristics of the network as a whole, and consequently make claims

regarding the propagation potential of the network on the system level.

Arguably, information on dyads in isolation should not be considered to describe *structural* properties of a network, as it does not provide any insight in how connections are related to each other.

Triads

By increasing the number of actors to be considered by one triadic structures can be created. These triads do capture the interrelated nature of connections and therefore are assumed to be the smallest unit of analysis for describing (local) network structure. As triads actually incorporate network effects, is a commonly studied local network structure. Triad can occur in potential forms (for argumentation sake we neglect the fact that ties can be directed); one of closure (Coleman, 1988) or one with a structural hole (Burt, 2005). The difference between these structures lies in the (lack of) presence of the third tie. In a brokerage situation actor A enables interaction between two other actors, B and C, which are not directly connected. In a situation of closure all three actors are connected to each other (Figure 2.7).

Being in a brokerage position has two benefits; First as a bride between to unconnected actors, a broker has control over the flows between these actors. Second, all flows go via the broker it is able the recombine such flows before any of the alters can, resulting in an information benefit.

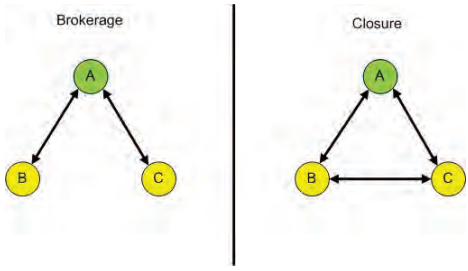


Figure 2.7: Brokerage versus Closure

From the propagation perspective broker are critical as they connect two otherwise unconnected parts of the network. This indicates that betweenness, the extend to which an actor is on the shortest path between other actors in the systems, is a key factor in propagation.

In a situation of closure all three actors are connected to each other, which results in them all having equal power. Actors who try to pressure their alters can be corrected for their behavior, as the alters are able to form coalitions. This results in a situation in which negative actions can be countered and hence will be less likely to occur. The benefits of such a situation are claimed to be closer collaboration, more trust between the actors and increased sharing (resulting in shared norms), indicating that closure situations are more

prone to propagation in the network.

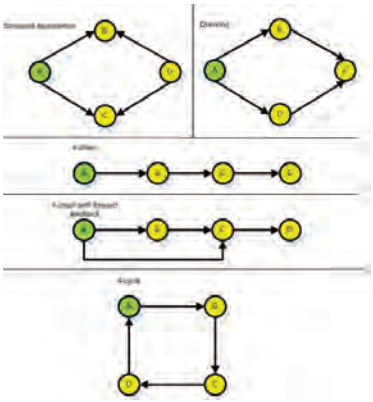


Figure 2.8: Examples of network motifs - note that in these examples the connections are assumed to be directed.

Motifs

Further increasing the size of the structure can be done by adding one or more connections to the network of an alter. Network structures which include the connections of the alters beyond distance 1 are called network motifs (Milo et al., 2002; Alon, 2007). These network motifs hold critical information regarding the local structure.

Consider an example (Figure 2.8, top structure) in which an actor A has a brokerage position over B and C. The extent to which this actor A is able to broker however strongly depends upon potential alternatives B and C have to interact. The presence of an actor D which is structurally equivalent to alter A will significantly reduce the influence of broker A.

Motifs can be used to characterize the network structure on the system level (Milo et al., 2002), but has not been linked to propagation dynamics, even though there seems to be an apparent connection between the two.

2.3.4 The impact of network structure on propagation

While the range of local structures is virtually limitless, most of them describe a sub-set of actors. This sub-set is bigger than that considered on the mirco-level, but smaller than the system as a whole. These sub-sets of actors constitute the meso-level structural measures. Most comonly these meso level measures revolve around clustering and communities in the network (Girvan and Newman, 2002), or different types of centrality such as betweenness centrality (Freeman, 1977) and eigenvector centrality (Bonacich, 2007).

Many meso-level structural characteristic are (to some extend) interrelated, making it difficult to consider them in isolation, especially in real world networks. In such networks there is no way to control for interactions, therefore most studies on the impact of network structure are done using synthetic networks. In these networks a specific structural characteristic can be manipulated while keeping other characteristics as stable as possible. By doing so it has for example been shown that, a shorter average path length

and the presence of long jumps (links connecting otherwise distant part of the network) (Pastor-Satorras and Vespignani, 2001b; Pandit and Amritkar, 2001), a scale-free degree distribution (Pastor-Satorras and Vespignani, 2001a), and an increased density (Burt, 2005) increase the speed and impact of propagation. On the other hand it has been shown that fully random or regular network are generally poor at facilitating propagation (Watts, 1999) and that clustering facilitates propagation within the cluster but not between the clusters(Rahmandad and Sterman, 2008).
A critical note should however be placed at the generalizability of such findings. They have been obtained assuming there is no impact of the mechanism of propagation. Consequently all effects found have been attributed to network structure. It could however be that the structure has different effect across different mechanisms of propagation.

2.4 The seed of infection

The third driver of propagation dynamics identified in literature is the seed of infection. This seed refers to the initial location (actor, or group of actors) at which the propagation phenomenon enters the system. While the seed is commonly mentioned and studied in literature (e.g. (Stonedahl et al., 2010)) as a driver of propagation this seems to be an incorrect assumption. Taking a pragmatic look at this driver indicates that the seed will only impact the propagation dynamics when heterogeneity in the can be leveraged. In a scenario in which the network is completely homogenous, the network will look exactly the same regardless of where the propagation process will enter the network, consequently the seed will cause no variation in the dynamics of the subsequent propagation process. Therefore the impact attributed to the seed of infection in fact stems from the assumption that networks are heterogeneous, suggesting that the heterogeneity in the system is the real driver of propagation dynamics.
In scenarios where the system is heterogeneous, the structure of the network will differ depending on which part of the network is considered. Depending on the choice of seed, a different structure can be faced in the early stages of propagation. As this local structure serves as the infrastructure underlying propagation in the earliest stages, it will restrict the initial propagation dynamics which in turn strongly affects the momentum a propagation process can gain. This indicates that in heterogeneous scenarios the seed certainly does matter.
Essentially it is the local dynamics which drive the system-wide dynamics. This suggests that things which can cause heterogeneity in the the local dynamics have the potential to affect the propagation dynamics on the system-level. The (local) network structure

can cause local variation in the dynamics, but also the actor behavior is likely to have a strong effect. As the mechanism of propagation is made up from the behavior of actors, the behavior by definition has an effect of the propagation dynamics. It is commonly accepted that actors are heterogeneous by nature, no two actors are exactly the same, and therefore their behavior will differ. This heterogeneity in actor behavior therefore will cause variations in how propagation occurs locally. Consequently, this behavior is likely to yield an effect on the propagation dynamics as a whole.
While the effects of heterogeneity have been studied in literature (Goldenberg et al., 2001; Rahmandad and Sterman, 2008; Young, 2009; Jackson and Lopez-Pintado, 2013), the results of these studies seem to be inconsistent at least. On the one had it is found that heterogeneity plays a role in determining the propagation effectiveness (Jackson and Lopez-Pintado, 2013), while other studied argue that such effect is neglectable (Rahmandad and Sterman, 2008). This suggest that the effect of heterogeneity on propagation dynamics are not completely understood in current propagation literature.

2.5 More complex propagation processes

In describing the drivers of propagation dynamics literature has adopted relatively simple propagation processes. In reality propagation processes might however be quite more complex. Thus far it has been assumed that each of the drivers (the mechanism, network structure and local heterogeneity) has an independent effect on propagation dynamics. This makes studying propagation far more easy, but might not be the most realistic assumption.

Independence among the drivers of propagation dictates that during the radiation of (a set of) signal(s) each of these signals is independently sent. Any signal being radiated will have no effect on the other radiated signals. It therefore does not matter how many signals are being radiated. Similarly during the reception process it is assumed that processing of signals is independent of the amount of signals received.
Centola and Macy (2007) however put forward a complicating argument; they suggest that in scenarios where the behavior being propagated is either costly, risky, or controversial the willingness to participate in this behavior and consequently a change in behavior/state of actors (reception) requires independent affirmation or reinforcement from multiple sources. What this effectively does is break the linear relationship between the amount of signals processed and the behavior. The reception behavior is significantly different when two signals are received as compared to a single signal. As multiple signals

can only be a result of multiple incoming ties, this implies that the network structure will affect the mechanism of propagation. Therefore the assumption that drivers are independent is abandoned. The authors refer to this type of propagation as 'complex contagion', and show that the interaction between drivers can yield propagation dynamics which are significantly different from traditional propagation processes.
While complex contagion is but a single way of relaxing the assumption of independence of drivers of the propagation dynamics, many similar notions of more complex propagation processes can be found in literature and practice. For example the notion of information overload refers to the idea that actors are bounded in their capabilities to process information (Simon, 1982). This bounded capacity will cause the efficiency by which actors respond to signals (reception) to either be capped or at least show a concave relationship with the incoming signals. Again as the amount of signals is dependent only on the network structure, this suggests an interaction of the the network structure and the mechanism of propagation.
Both the notions of complex contagion and bounded rationality refer to interaction among signals during the reception sub-process. But a similar complicating mechanism can be proposed for the radiation sub-process. An example is costly radiation. Costly radiation assumes that the are costs involved with sending signals and that actors are limited in the resources they have. Combined these constraints result in actors being restricted in the extent to which they can or will send out signals.

Each of these examples captures some form of a more complex propagation behavior. They all incorporate a notion of interaction among the drivers of propagation dynamics. This adds another level of complexity to the propagation process, of which the effect have not been structurally studied and hence remains poorly understood.
To explore this additional layer of complexity in a structured way, one needs to have method in place which is able to distinguish between all three drivers of propagation. Only when the the drivers can be identified the interactions among them can be considered. And only when the interactions among them are considered one can start to understand the complex propagation dynamics. While traditional models allow for incorporating the effects of the seed (as well as local heterogeneity which drives this effect), and the network structure, they generally lack a detailed description of the propagation mechanism. This makes traditional propagation models ill-equipped to address this layer of complexity. In contrast, the information processing view of propagation and the subsequent RTR-model of propagation are designed to capture and describe the propagation mechanism, therefore they are particularly well suited to study these more complex phenomena.

Chapter 3

The conceptual framework

In studying dynamics of propagation the literature chapter has introduced three drivers of propagation; the network structure, the seed of infection and the propagation mechanism. While there is literature describing the effects of network structure and the seeding locations, literature on the mechanism of propagation is largely missing. We aim to fill this gap in literature, and hence this dissertation will focus specifically on the mechanism of propagation. It aims to shed light on the impact of the mechanism of propagation on propagation dynamics, and by doing so increase the grip on these dynamics.

To describe the mechanism of propagation the information processing view of propagation, and the subsequent RTR-model of propagation, have been put forward. In this view (and model) the propagation process is considered as an information processing process, and consequently decomposed into three sub-processes; Radiation, Transmission and Reception. These sub-processes describe how information is processed on the actor level, and hence describe the mechanism of propagation.

In order to show the impact of the mechanism of propagation it is not sufficient to simply link the mechanism to propagation dynamics. As there are multiple drivers of propagation, the interactions between all three drivers will determine the eventual propagation dynamics. The impact of the mechanism should not be considered without also considering other drivers of propagation. Therefore, in this dissertation, the propagation mechanism is linked not only to the dynamics of the propagation process, but also to the two other drivers of propagation dynamics. Finally, the goal is to increase the grip on propagation dynamics, an increased understanding of these dynamics would allow for better intervention strategies, and it is argued to this research is a means to this end.
The resulting conceptual framework (depicted in figure 3.1) thus proposes four relation-

ships. The first three relate to understanding the propagation dynamics, and are consequently linked to three research questions. The latter focuses on the practical implications of such an increased understanding.

The first relationship describes the direct effect of the mechanism of propagation on the propagation dynamics. It can be linked to the first research question in this dissertation which reads: '*(How) do differences in the information processing at the actor level affect system-wide propagation dynamics?*'.

The second relationship describes the interaction effect of the mechanism of propagation and the network structure. It is assumed that the effects of network structure can be very different under different mechanisms of propagation, suggesting that the mechanism has a mediating effect on network structure. It can be linked to the second research question, which reads: '*How does information processing at the actor level affect the effects of network structure on system-wide propagation dynamics?*'

The third relationship, implicitly considers the third driver of propagation, the seed of infection. However, the seed of infection only has an impact on propagation dynamics when there is heterogeneity in either the network structure or the behavior of actors in the network. As heterogeneity in the network has already been covered in the second relationship and question, the third relationship describe the effect of actor heterogeneity. Rather than explicitly considering the seed of infection it thus focuses on that which makes the seed a driver of dynamics, and considers the interaction between heterogeneity in the actor behavior and the propagation mechanism. This relationship can be linked to the third research question which reads: '*What role does heterogeneity of actor information processing behavior play on propagation dynamics?*'.

The fourth and last relationship, does not link to a specific research question, instead it considers the practical value of the three questions posed before. Understanding the role of the propagation mechanism on the dynamics is critical in terms of knowledge creation, but the real value would be derived from that which can be done with such understanding. When increased understanding results in better intervention strategies, it will allow for increasing the grip on propagation dynamics.

To study the proposed relationships in the conceptual framework, each of the associated research questions will be addressed in a separate study.

- Study 1 (chapter 4) will be aimed at considering the direct effect of the propagation mechanism on propagation dynamics. It will describe the information processing view of propagation and the model of propagation capturing this view, the RTR-model of propagation. It captures the potential effects of difference in the propagation mechanisms and will provide validation with of the new propagation model.

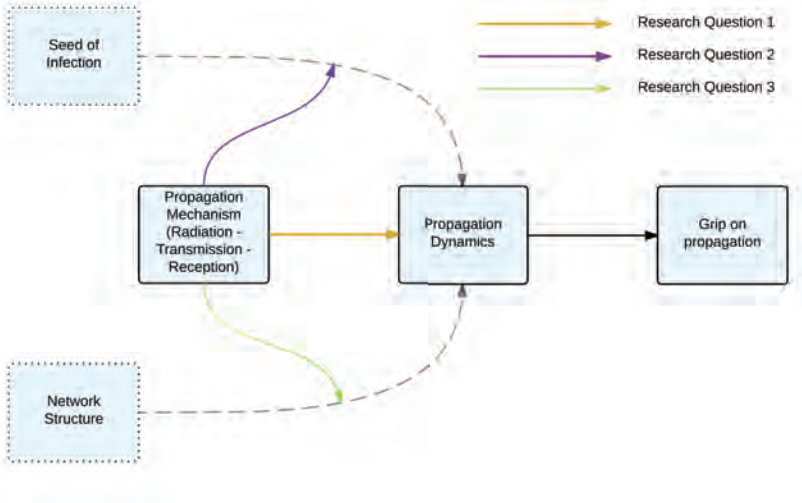


Figure 3.1: The conceptual framework of this dissertation

Emphasis is placed on validating both in the methodological and practical sense. Therefore this study also explicitly covers the impact on intervention strategies and relates directly to the fourth relationship in the conceptual model.

- Study 2 (chapter 5) will be aimed at considering the mediation effect of the mechanism of propagation on the impact of the actor behavior (actor heterogeneity). It will use agent based simulations with the RTR-model to capture the impact of differences in actor behavior and the consequent heterogeneity in local propagation dynamics. Once more these results are linked to potential lessons for interventions, specifically focused at seeding strategies.

- Study 3 (chapter 6) will be aimed at considering the mediation effect of the mechanism of propagation on the impact of network structure. Using agent based simulations with the RTR-model it will capture the effect of network structure under different mechanisms of propagation. By doing so it will test the notion that the characteristics of the network structure can have different effects depending on the mechanism being studied.

The combination of the three studies allow us to validate the importance of the propagation mechanism as a driver of propagation dynamics. First, the Agent-Based Modeling approach adopted is particularly suited for studying the propagation mechanism as a driver of propagation dynamics. It allows for describing what happens between actors

during propagation, and thus facilitates characterization of the local propagation mechanism. By doing so it allows system-wide dynamics to 'emerge' from local interactions without making a priori assumptions on how the system-wide dynamics will occur, which allows exploration of the effects on propagation dynamics.
Second, considering not only the direct effect of the mechanism but also the interaction with the other drivers of propagation dynamics, places the research within a larger context. While this might make settings very explicit, limiting the generalizability, it strongly increases the accuracy of the models and their findings, making them more suitable for being translated into practice.
Third, by considering also the practical relevance it not only considers whether the mechanism makes a difference, but also whether this is difference that makes a difference.

Chapter 4

Study 1: The RTR-model of propagation

Coauthors[1]: O.R. Koppius, P.H.M. Vervest

Abstract: *The study of network dynamics often focuses on the process of propagation; the process by which a change in the state of a focal actor results in a change of state in one or more of its connected neighbors. Propagation is studied in various fields under labels such as contagion, diffusion, spread, cascades or influence.*

Many previous studies of propagation have assumed propagation to refer to the process by which actors adopt a change in state. While this is an essential part of propagation, we claim that adoption of a state-change is only part of the propagation process and that propagation is actually a combination of three distinct sub-processes: Radiation, the sending of a signal, Transmission, the transport of the signal, and Reception, the response to a signal (RTR). We put forward a model for propagation based on these three distinct sub-processes.

We show that the sub-processes can be used to sub-divide potential interventions in the propagation process, in which each intervention can be mapped to a change in a particular sub-process. Using simulation with the RTR-model we show that understanding the different components of propagation is essential for assessing the effectiveness of interventions. Incorrect specification can lead to an over- or underestimation of the effectiveness of an intervention by almost an order of magnitude. This indicates that to manage the outcomes

[1]This chapter considers a working paper. Preliminary versions of this paper have been presented at Workshop Information in Networks (WIN) in 2012 and 2013 and the conference for complexity in business (CCB) in 2013.

of propagation and understand how to intervene effectively during propagation, it is vital to understand the role of the three distinct aspects of the RTR-model.

Propagation in networks refers to the process by which a change in the state or behavior of an actor results in a change of state or behavior in one or more of its connected neighbors. It covers processes such as spread of disease, diffusion of information or social influence. In literature the propagation process has been studied in various fields and is known under labels such as contagion (Centola and Macy, 2007; Dodds and Watts, 2004), diffusion (Reagans and McEvily, 2003; Rogers, 1995; Valente, 2005), cascades (Buldyrev et al., 2010; Buzna et al., 2006) or influence (Aral et al., 2009, 2013). In many of the propagation studies the structure of interactions among the actors, the network structure, has been the focus of attention. Studies that look at the role of network topology Dodds and Watts (2004); Albert et al. (2000); Pastor-Satorras and Vespignani (2001a), clustering (Centola and Macy, 2007; Dodds and Watts, 2004) or local network structures such as hubs (Boccaletti et al., 2006) or weak ties (Granovetter, 1973) have pointed out the importance of the network structure in determining the outcomes of the propagation process, suggesting it is the key to understanding the impact of interventions in the propagation process. In these studies however the role of the mechanism of the propagation process itself is often overlooked.

Previous work on propagation has adopted a variety of propagation dynamics (Bass, 1969; Valente, 1996; Kempe et al., 2003) depending on the field in which it is studied. In epidemics we speak of an infection rate, in management or social studies of an adoption threshold, and in physics of the percolation probability. While these dynamics assume a different mechanism of propagation they do share one commonality; the process by which the behavior or state of an actor (A) will result in a change of behavior or state of connected neighbor (B) is considered by means of a single rate or likelihood, and hence propagation is considered to be monolithic. Drawing on communication theory (Shannon, 1948), we however state that the mechanism of propagation is composed of three distinct sub-processes, Radiation, Transmission and Reception (RTR) (Figure 4.1), each with their own parameter (α, ϕ, η).

Radiation (α) refers to the process by which a change in the state or behavior of an actor results in that actor sending out signals to its outgoing ties. *Transmission* (ϕ) refers to the process by which a signal is transferred from the sender to the receiver (via a tie). *Reception* (η) refers to the process by which actors respond to the incoming signal(s) and change their behavior or state accordingly. Only when all three sub-processes have taken place can we speak of propagation.
For example, propagation of disease requires an actor to spread the virus/germ (radia-

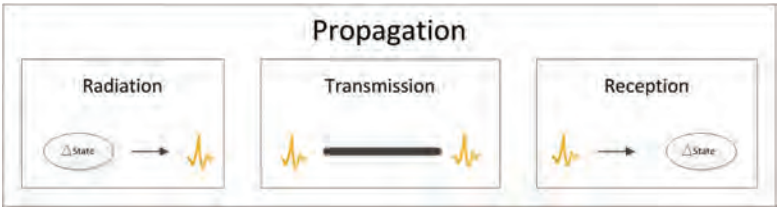


Figure 4.1: Propagation is divided in three distinct sub-processes; Radiation refers to the process with which an actor translates a change in state into an outgoing signal, Transmission to the process by which this signal is transferred (via a tie) towards the alter(s), and Reception to the process by which alters respond to the incoming signal and may change state accordingly.

tion), the virus/germs to be transferred to the alter(s) (transmission), and the alter(s) to get sick in response (reception). Similarly, the spread of a financial crisis requires banks to become insolvent and default on loans (radiation), this will affect the liquidity of the counterpart (transmission) but only result in propagation when the counter-part become insolvent as well (reception).

While current propagation models, using a single parameter, allow for variations in the rate of successful propagation, they make no distinction among the sub-processes in the propagation mechanism. Hardly ever claims are being made about which part of the mechanism is considered, and hence sub-processes are frequently used inter-changeably. Commonly literature refer to a process which resembles infection or adoption (Table 4.1) (mostly referred to as λ); they consider how actors respond to signals. In doing so how the signal reaches the actor is often ignored, and thus focus in literature has mainly been on the reception sub-process.

By not explicitly distinguishing the sub-processes of propagation and using them inter-changeably, it is assumed that each of they has the same effect on propagation, and that propagation mechanism thus can be considered as a homogeneous process. This assumption of homogeneity does reduce the complexity of propagation, but also limits the potential for describing the underlying mechanism of the process. Recent work (Centola and Macy, 2007; Aral et al., 2013; Banerjee et al., 2013) has found that neglecting these mechanics can have unforeseen and far-reaching consequences, suggesting that more attention needs to be paid to the mechanism underlying the propagation process. Centola and Macy (2007) in his study on complex contagion has shown that both the speed and pervasiveness of propagation are strongly influenced by claiming that for an actor to

Table 4.1: This table depicts the way in which propagation has been operationalized in recent literature. Different streams of literature focus on different sub-processes, and thus capture only part and not the whole propagation process.

Author	Year	Label of propagation	Sub-process considered		
			Radiation	Transmission	Reception
Granovetter	1973	Diffusion		x	
Rogers	1995	Diffusion			x
Reagens and McEvily	2003	Diffusion			x
Dodds and Watts	2004	Contagion			x
Valente	2005	Diffusion	x		x
Buzna et al.	2006	Cascades			x
Centola and Macy	2007	Contagion			x
Rahmandad and Sterman	2008	Diffusion	x		
Buldyrev et al.	2010	Cascades		x	x
Iyengar et al.	2011	Contagion			x
Aral et al.	2013	Influence	x		x
Banerjee et al.	2013	Contagion	x		x

change state, multiple stimuli are needed. Furthermore, the notion that actors can send out very different strengths of signals (Aral et al., 2013; Banerjee et al., 2013) and/or be susceptible to different magnitudes of signals (Aral et al., 2013) have been shown to have a strong influence on the outcomes of propagation. These results indicate that variations in process mechanism have a marked effect on the outcomes of the propagation process, suggesting a need to focus on the process itself rather than on the network structure.
The need to pay attention to the sub-processes of propagation becomes even more apparent when one considers changes in the propagation process. Such changes can be either unintentional or made in order to steer propagation, in which case we refer to them as interventions. Closely looking at such interventions reveals that each intervention is in fact aimed at changing a specific sub-process. The set of interventions described by Valente Valente (2012) can each be linked to a specific single sub-process: selecting leaders who are able to persuade others would be a radiation-based intervention; selecting low-threshold agents who easily change they state or behavior would be a reception-based intervention; and both induction and alteration which focus on the structure that facilitates transfer would be transmission-based interventions. Similarly, the interventions made to steer the outcomes of propagation processes in practice (Table 4.2), can be mapped to specific single sub-processes.

The notion that interventions are linked to a single sub-process rather than to the propagation process as a whole changes our perspective on how interventions work. Rather than changing the outcomes, the dynamics of the propagation process, interventions change the very mechanism that underlies them. These changes in mechanism in turn result in differences in the dynamics. Consequently, steering the outcomes of propagation now becomes a question of knowing how precisely interventions change the mechanism of the propagation process. Traditional single-parameter models adopt an oversimplified notion of the propagation mechanism, they assume homogeneity among the sub-processes, which makes them inadequate for dealing with variations in the mechanism and distinguishing between the effects of different types of interventions. Studying the impact of interventions therefore requires a new type of propagation model.

4.1 The Radiation-Transmission-Reception model

We put forward a new model, the Radiation-Transmission-Reception model of propagation or RTR-model. This model describes the propagation mechanism on the dyadic level (between two actors), and hence can be considered as an Agent-Based Model for propagation. In its general form, the RTR-model consists of functions for Radiation, Transmission and Reception (full description in the supplementary information[SI]) which consequently allows a change in state to propagate from one actor to its connected neighbors (alters). The radiation function describes how the change in state of an actor i ($\Delta s_{i,t}$), if higher than the radiation threshold (u), will result in a signals being sent ($p^{out}_{i,e,t} \in P^{out}_{i,t}$) to the outgoing ties ($E^{out}_i$).

$$\sum_{t'=t-T_{rad}+1}^{t} \sum \left(A_{i,t'} \times P^{out}_{i,t'} \times (\tau_{rad})^{(t-t')+1}\right) = \begin{cases} \Delta s_{i,t} & \text{if} \quad \Delta s_{i,t} \geq u \\ 0 & \text{if} \quad \Delta s_{i,t} < u \end{cases} \tag{4.1}$$

In which $A_{i,t}$ ($\alpha_{i,e,t} \in A_{i,t}$) is a vector of capturing the characteristics which influence the radiation per outgoing edge, T_{rad} is the radiation memory duration, the number of time units after a change of state that this change can cause radiation, and τ_{rad} is the memory inflation factor, the extend to which past changes are amplified or dampened.
The transmission function describes how the signal radiated ($p^{out}_{i,e,t} \in P^{out}_{i,t}$) is transformed into the signal which is received by the alter j ($p^{in}_{e,j,t}$)

$$p^{out}_{i,e,t} = \sum_{t'=t-T_{tra}+1}^{t} \left(\phi_{e,t'} \times p^{in}_{e,j,t'} \times (\tau_{tra})^{(t-t')+1}\right) \tag{4.2}$$

In which T_{tra} is the memory duration for transmission, τ_{tra} is the memory inflation factor for transmission, and $\phi_{e,t}$ is a vector of edge characteristics which influence transmission. The reception function describes how transmitted signals ($p^{in}_{e,i,t}$) received from the incoming ties (E^{in}_i) translate into a change of state of an alter if their combined effect passes the reception threshold (q).

$$\Delta s_{i,t} = \begin{cases} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P^{in}_{j,t'} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) & , \text{ if} \\ \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P^{in}_{j,t'} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) \geq q & , \text{ otherwise} \\ 0 & \end{cases} \tag{4.3}$$

Table 4.2: Examples of interventions in propagation in practical settings. For each setting it shows how the propagation process can be divided into three sub-processes and how different interventions are linked to specific sub-processes.

Setting	**Radiation**		**Transmission**		**Reception**	
	Process	Intervention	Process	Intervention	Process	Intervention
Spread of financial crisis	A bank becomes insolvent and defaults on a loan	*Capital injections* from central banks may change the state of the insolvent bank, making it solvent again before any propagation can occur. This prevents the radiation of a signal.	The loans between banks causes the default of one bank to result in a loss for a connected bank.	*Guarantees, securities and derivatives* can ensure defaulted loans are still (partially) covered, reducing the signal which reaches the alter.	The other bank will incur a loss from the defaulted loan and can absorb this loss or change state to become insolvent.	*Enforcing an increase in the liquidity buffers* results in banks being less likely to also become insolvent when incurring losses from other defaults.
Contagion of disease	A person becomes sick and spreads the viral load/germs in the direction of others, i.e. through sneezing.	*Quarantine interventions*, such as telling people to stay at home, reduce their interaction with friends and hence reduce the chance of spreading the disease to these friends.	The transfer of viral load/germs to other people (alters), i.e. being hit by a sneeze or touching a contaminated surface.	Getting those people who are infected to *wear protective masks, and/or wash their hands* extensively will reduce the viral load/germs an individual will spread and hence reduce the transmission of the virus to other people.	The alters respond to the viral load/germs being received. They attempt to fight off the infection, and either succeeds, so the disease does not propagate, or fail and gets sick.	*Immunization* decreases the susceptibility of actors, as immunized actors are considered to react differently to incoming signals. Through immunization, the reception process of these actors is changed.
Adoption of innovation	A firm adopts a new production technique and its employees start to talk about this adoption.	Communication or visibility is reduced or increased by means of *confidentiality or advertising agreements.*	Other firms becoming aware of the adoption, through interaction. This can be by communication or observation.	By *using a different means of communication or changing the transparency* the extent to which the signal reaches an alter can be changed.	Other firms make a decision on whether to also adopt the innovation, based on the signals they receive via their network.	By *changing the investment costs* (subsidizing or licensing) the other firms will become more, or less, prone to adopt the innovation.
Diffusion of information or knowledge	A person has a certain piece of novel information and decides to share this.	*Rewarding sharing activity with status* (as is often done on online forums) incentivizes the sharing of information.	Communication among individuals allows the information to be spread from one person to another.	By *using a different medium* (e.g., telegram vs. videoconferencing) the richness of communication can be changed, thus improving or reducing the extent to which the information is understood during transmission.	The alter receives the information and fits this to his or her existing knowledge and decides to accept or reject the information, in full or in part.	By *decreasing or increasing the utility* (novelty) of the information being shared, the willingness to accept such information can be increased.

In which T_{rec} is the memory duration for reception, τ_{rec} is the memory inflation factor for reception, and $\Psi_{i,t}$ ($\eta_{e,i,t} \in \Psi_{i,t}$) is a vector capturing actor specific characteristics influencing the reception per incoming edge. Using equations 4.1, 4.2 and 4.3, the RTR-model decomposes the single-parameter propagation process into three distinct sub-processes. What becomes clear when considering these sub-processes is that the each of them adopts a different logic. Radiation is a process from one actor to its outgoing ties, making it essentially a one-to-many process. Transmission occurs within a single tie and hence is one-on-one process. Reception is a process from many incoming ties to one single actor, making it a many-to-one process. Only by knowing what is actually happening during each step of propagation and describing the propagation process in terms of three distinct sub-processes one can understand the impact of interventions on the overall propagation likelihood and get a grip on the propagation dynamics.

4.2 Methods and Data

To indicate the relevance of using the RTR-model and the subsequent decomposition of propagation into three distinct sub-processes we will adopt a two-staged approach; first, we show that using this model allows for a better representation of propagation field data, and second, by means of simulation, we will show that knowing the three sub-processes is critical in order to effectively intervene in the propagation process.

While the sub-processes are conceptually different, data on propagation processes often only measures the outcomes of propagation (actors adopt or not), and hence lacks this distinction. Consequently, measurements of separate sub-processes are rare. As measuring the individual sub-processes is difficult, modeling the division into sub-processes will only yield relevant insights if these sub-processes can in fact be deduced from the observed outcomes in field data. Therefore in the first part of the analysis we justify dividing propagation into sub-processes by applying the RTR-model to field data relating to the adoption of a software application called Yahoo! Go. We show that applying the RTR-model not only enables us to parametrize the distinct sub-processes but also to generate a model that is a better fit with the actual propagation data.

In the second part of the analysis we focus on the implications of using the RTR-model. We show that only by dividing the propagation process into three sub-processes one can understand why interventions vary in effectiveness.

4.2.1 Field data

4.2.1.1 Yahoo! go 2.0 application adoption

In this section we apply the RTR-model to a real-world propagation setting: the adoption of a mobile software application. We use data that describes the adoption of the Yahoo! Go 2.0 application among approximately 27 million users of Yahoo messenger (Yahoo!, 2010) as collected by Aral et al. (2009). Drawing on instant messages among users we create a weighted network over which adoption can spread (more details on the data can be found in SI and Aral et al. (2009)) and track the spread over a period of four weeks. We assume this propagation to be based on the buzz created by adoption, suggesting that after adoption users will start sending out (radiate) signals towards other people, but will only do so for a limited period of time (in our case, one week). This has two implications for the way in which we study propagation in this setting. First, there is only a limited number of states that actors can be in; they are either able to adopt (*susceptible*) or have adopted already (*infected*). As we consider a propagation process driven by buzz, adopters will stop sending out signals and are permanently in an infected state (they are removed) a week after being infected, effectively making this model an SIR (*S*usceptible, *I*nfected, *R*emoved) model. Second, because actors send out (radiate) signals only while they are in the infected state, we consider only the interactions during this period as being important for propagation (we do not consider any previous interactions).

4.2.1.2 Applied Radiation-Transmission-Reception model

In our empirical setting we are considering a scenario in which actors can only have limited amount of states (they are susceptible, infected or removed) like the SIR models which traditionally used to model contagion of disease. Therefore, we restructure the general RTR-model, which is deterministic in nature, into a stochastic variant which resembles SIR model logic (for a detailed description see [SI]). This stochastic variant can be summarized as follows:

- Each actor in the system has a state ($s_{i,t}$): either Susceptible, Infected or Removed.
- Each infected actor in any time-step has a probability ($\alpha_{i,t}$) of sending out a signal to and activating an outgoing tie for transmitting a signal (all ties are considered separately).
- Each activated tie has a probability ($\phi_{e,t}$) of transmitting the signal which has been radiated at the same time-step.

- Each actor in the system has a probability ($\eta_{i,t}$) of changing to an infected state due to any single incoming signal (we assume no interaction among signals, therefore all signals are considered sequentially).

Our formulation dictates that the extent or likelihood of propagation (λ) is a product of the three sub-processes ($\lambda = \alpha\phi\eta$). Using a brute-force simulation approach (see [SI]), we retrieve the system-wide parameters (radiation, transmission and reception) for the stochastic RTR-model. We do so by simulating RTR-models with different parameters and picking the parameters which most accurately reproduce the real propagation found in the Yahoo! Go adoption data. We compare these results with those obtained from traditional single-parameter propagation models. We have chosen four single-parameter models; the first model, based on current literature, assumes no differences and interchangeability among sub-processes. All sub-processes play an equal part in the propagation process and hence always have the same value ($\alpha = \phi = \eta$). The latter three single parameter processes are traditional propagation processes mapped onto the different sub-processes, effectively assuming that it is a single sub-process —Radiation, Transmission or Reception respectively— which drives propagation (and that the other two sub-processes will always occur and thus do not play any role).

4.2.1.3 Results

Our results (Table 4.4 and Table 4.5) indicate two things: That we can distinguish the separate sub-processes and that by dividing the propagation process into sub-processes we have constructed a better fitting model of propagation (at least in this setting).
First and foremost, the optimal parametrization found using our brute force method (Table 4.4) shows variations between the sub-process parameters. This implies that there are differences between the three sub-processes in practice, contradicting the assumption of homogeneity and hence justifying the use of the RTR-model, and using three sub-processes to model propagation. Even more so it indicates that without gathering additional data we can re-analyze existing propagation processes to obtain the parameters for the specific sub-processes. We find that the optimal parametrization of sub-processes strongly depends on the fit measure applied, raising the question whether there is in fact a single stable parametrization for a propagation process. While this question is outside the scope of this research, it certainly poses an interesting direction for future research.
Second, the fitting results (Table 4.5) reveal that the RTR-model has a lower level of error and hence fits better than any single-parameter model, a result which has been found to be robust over all fit measures used. To further examine the robustness of our results (see

the [SI] for description robustness checks) also the Akaike information criterion (AIC) for model performance (Akaike, 1974) has been included. This measure penalizes the use of multiple parameters. Our results (Table 4.5) show that the RTR-model has the lowest AIC scores of all tested models (52.68 compared to respectively 54.26, 53.80, 98.86 and 53.67), indicating that even when penalized for using more parameters the RTR-model consistently outperforms the single parameter models in terms of reproducing real world propagation data.

4.2.2 Impact of interventions

In the previous section we have shown the methodological validity of the RTR-model, but in this section we will show the practical value of adopting the RTR-model. By means of simulation we show that the impact of interventions depends strongly on the decomposition of the propagation process. Hence effectively managing these processes requires a detailed view of the propagation mechanism.

4.2.2.1 Simulation using the Radiation-Transmission-Reception model

In our simulation we again use a model in which the state of active actors is limited to being either infected or susceptible, allowing the use of the probabilistic variant of RTR-model ($\lambda = \alpha\phi\eta$), similar to the one used while analyzing the field data. The simulation model differs from the empirical setting in three ways: First, we consider actors to recover rather than be removed, effectively making this a SIS model. We assume actors to recover with a given likelihood (ρ), rather than being recovered automatically after one time-step. Second, given that actors can be in the infected state for a longer period of time, we consider them to radiate during each time unit in which they are infected. Third, the network structure remains stable over the course of the simulation.
The simulation approach can be summarized as follows (a detailed description of the simulation method can be found in the IS). We run a set of 10,000 simulations on a given stable network structure (undirected and scale-free, with 10,000 actors generated using the Barabsi-Albert Barabási and Albert (1999) approach), and for each of these simulations we vary the seed of infection, effectively bootstrapping each scenario we simulate 10,000 times. We repeat this simulation process for multiple scenarios with the same overarching propagation probability (λ) but different decomposition into three sub-processes. In line with previous studies Albert et al. (2000); Pastor-Satorras and Vespignani (2001a, 2002) we consider the proportion of infected actors in the system —the so-called pervasiveness— to be the primary outcome variable.

We take a mean-field approach when considering the simulation results, averaging the pervasiveness found over the bootstrapped set of simulation runs. Averaging these results over all potential seeds implies we lose the individual dynamics stemming from the initial seed; this enables us to capture the effects of the variation in process dynamics without having to worry about effects of the network structure. This approach does hide many of the differences between the various scenarios studied, but even when using this (over)simplified method and studying propagation outcomes at the mean-field level the impact of studying the sub-processes of propagation becomes apparent when interventions in the propagation process are considered.
We measure the effects of interventions by simulating a propagation process and intervening in this process halfway through the simulation (at $t = 50$). As has been shown, an intervention can be linked to a single sub-process, and consequently an intervention is operationalized as a change in one of the sub-process parameters. As we consider the sub-process parameters in a stochastic model —meaning they should be interpreted as probabilities of success in each step in the propagation process—, any absolute change made to a sub-process parameter translates into a change in the probability success for that specific sub-process. While an intervention of any size could have been applied in our simulations, we chose to use an intervention that would reduce the success chance of a sub-process by 20%, effectively reducing either α, ϕ or η by 0.2. Generally we assume that an intervention decreases (or increases) the likelihood that a sub-process will successfully take place, and therefore an absolute parameter change is the most suitable form of modeling interventions.

4.2.2.2 Results

Our simulation results (Figure 4.2, Figure 4.3, Figure 4.4) show that the same intervention can have very different effects depending on the decomposition of the propagation process, which is not surprising when we consider that $\lambda = \alpha\phi\eta$. This formulation dictates that the effect of an absolute change in either of the sub-processes on the propagation probability (the effect on λ) will depend on the parameters of the other sub-processes. Therefore the effect of an interventions can differ drastically, depending on the decomposition in to sub-processes.

When considering the effects of adopting the RTR-model in this setting it is interesting to compare the scenarios with heterogeneous sub-processes (scenarios 1-3: Figure 4.5, Figure 4.6, Figure 4.7) to the ones in which the sub-processes are assumed to be homogeneous

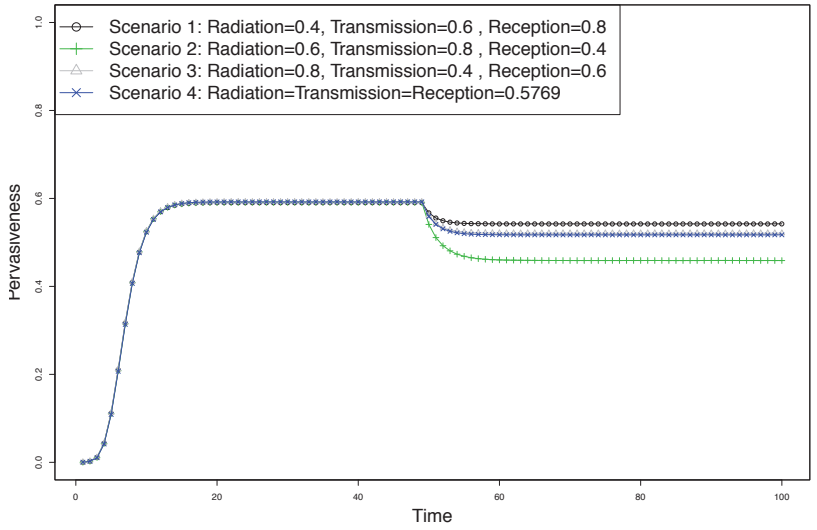


Figure 4.2: The pervasiveness over time is depicted for scenarios with the same overarching propagation likelihood ($\lambda = 0.192$) but with different decompositions into radiation (α), transmission (ϕ) and reception (η). The black open circles represent a process with $\alpha = 0.4$, $\phi = 0.6$ and $\eta = 0.8$. The green pluses represent a process with $\alpha = 0.6$, $\phi = 0.8$ and $\eta = 0.4$. The (grey) triangles represent a process with $\alpha = 0.8$, $\phi = 0.4$ and $\eta = 0.6$. The blue crosses represent a process with homogeneous sub-processes ($\alpha = \phi = \eta = 0.5769$). During the simulation at t = 50 an intervention (reduction of 0.2) in the reception is applied (figures of interventions in radiation and transmission, are included in the SI

(scenario 4, Figure 4.8). In the homogeneous scenario any intervention will yield the same effect regardless of which sub-process is targeted. In contrast, in the heterogeneous scenarios the decomposition into sub-processes allows the same intervention to result in very different effects. Clearly in such heterogeneous scenarios the decomposition of propagation will affect the outcomes of an intervention, making the question of which sub-process to target a crucial one for effective interventions.

In our example scenarios we have assumed a given propagation probability (λ) and network structure. In this setting we have shown that under the assumption of homogeneity of sub-processes the intervention we enforce (a 0.2 reduction in either single sub-process) would yield a 12.7% reduction in pervasiveness. We use this as a baseline scenario and compare it to two extreme scenarios (Table 4.3, Figure 4.9). In the first extreme scenario —a worst case scenario (the likelihood of success pre-intervention equals 1.0)— the

same intervention has an effect of reducing the pervasiveness by 6.1%, indicating the lower bound of the intervention effect. In the second extreme scenario —a best case scenario (likelihood of success pre-intervention equals 0.2)— the same intervention yields a 100% reduction in pervasiveness (and hence eradicated the infection), indicating the upper bound of the intervention effect. The differences in lower and upper bounds show that assuming homogeneity among sub-processes (not decomposing propagation into sub-processes), can lead to an overestimation of the effect of an intervention by a factor of 2 or an underestimation by a factor of 8. Even more so these bounds indicate that incorrect decomposition in sub-processes can even yield an over- or underestimation by a factor of 14.

While these factors will clearly vary across scenarios being studied, variance of an order of magnitude does pose a problem. It indicates that different mechanisms (decomposition into three sub-processes) can yield different intervention effects, suggesting that in order to effectively intervene in the propagation dynamics one needs to know how the propagation process is decomposed into sub-processes.

4.3 Conclusion

In this paper we put forward the notion that the propagation process is not monolithic and that the mechanism by which it occurs should be decomposed into three sub-processes: Radiation, Transmission and Reception. We propose a model incorporating these three sub-processes (the RTR-model of propagation) which allows the capturing of differences in the propagation process dynamics. We show that each of the sub-processes covers a distinct part of propagation, therefore dividing the mechanism into sub-processes gives us the ability to describe the process in more detail, and model behavior which can not be modeled by using a single-parameter model. We corroborate this by showing that the RTR-model is better able to reproduce real propagation phenomena than any single-parameter model.

In our paper we also show that interventions are linked to specific single sub-processes, therefore in order to evaluate the effectiveness of these interventions the sub-processes need to be identified. We show that failing to correctly represent the sub-processes can yield an under- or overestimation of intervention effects by more than an order of magnitude. Our findings have important implications for decision-makers trying to steer propagation processes. The use of the RTR-model provides a detailed description of the propagation mechanism and effectiveness of interventions. As interventions in different sub-processes require a different set of actions, this allows decision-makers to make more focused efforts

Table 4.3: This table compares three scenarios with equal overarching propagation probability (λ), a homogeneous scenario where each sub-process is equally strong, and the two most extreme scenarios, a worst case scenario which is extremely resilient, and a best case scenario which is extremely volatile. It considers the effects of the same type of intervention in each of these scenarios.

Scenario	**Pre-intervention pervasiveness**	**Post-intervention pervasiveness**	**Intervention effect**	**Comparison with homogeneous scenario**	
				Absolute Error	**Relative Error**
Homogeneous sub-processes: $\alpha = \phi = \eta = 0.5796$	0.592	0.517	12.7 %		
Most resilient Scenario: likelihood of success pre-intervention equals 1	0.593	0.557	6.1 %	-0.041	-52.0%
Least resilient Scenario: likelihood of success pre-intervention equals 1	0.592	0.000	100 %	0.517	687.4 %

at intervention and hence to become more effective in steering the propagation process outcomes.

Our results suggest some interesting directions for future research. First, as previous literature has often been unclear on which sub-process has been considered, it would be valuable to reanalyze data sets used in existing literature to get an overview of the impact of each sub-process on process outcomes. Second, while we have explored the impact of differences in the propagation mechanism, we currently assume that all actors behave similar. In many real-life situations we find that actors in fact differ in terms of their contagiousness, transmission medium, and susceptibility, making this an interesting extension to our model. Third, we have assumed a stable network structure, ignoring the probable interaction between the structure of the network and process mechanism. Studying this interaction would be a valuable avenue for future research.

4.4 acknowledgments

We would like to acknowledge Yahoo! Labs (http://webscope.sandbox.yahoo.com) for providing access to the G7 - Yahoo! Property and Instant Messenger Data use for a Sample of Users, v.1.0 database used in this study.

4.5 Supplementary Information

This section contains a detailed description of the methods used in the paper and additional information to support the readers understanding of the article: "The three sub-processes of propagation in networks: The Radiation-Transmission-Reception (RTR) model of propagation". This section will contain the following tables, figures and methods:

Tables:

- Table 4.4: The optimal operationalization of the RTR-model
- Table 4.5: Model fit for RTR and single-parameter models

Methods:

- S1: The Yahoo! Go service application
- S2: The general RTR-model of propagation
- S3: The stochastic RTR-model
- S4: Brute-force fitting
- S5: Simulation using the RTR-model

Figures:

- Figure 4.3: Interventions in the Radiation process
- Figure 4.4: Interventions in the Transmission process
- Figures 4.5 - 4.8: Effectiveness of interventions in per scenario (1-4)
- Figure 4.9: Intervention in extreme scenarios
- Figures 4.10 - 4.13: Degree per week (Yahoo! data)
- Figures 4.14 - 4.17: Degree correlations per week (Yahoo! data)
- Figure 4.18: The steps in the propagation process
- Figure 4.19: Propagation fitting data
- Figures 4.20 - 4.22: RTR-model validation

Table 4.4: This table shows the parametrization of the RTR-model (into the three sub-processes) which best fit the actual propagation numbers found in the Yahoo! Go field data. A different fit measure has been applied for each model, reporting the total error of the model for that measure.

Optimal parametrization of RTR					
Radiation	Transmission	Reception	**RMSE**	**LRMSE**	**RRMSE**
0.1	0.3	0.1	684.2		
0.1	0.1	0.2		0.982	
0.2	0.2	0.1			0.855

Table 4.5: This table shows the errors (and errors relative to the RTR-model) obtained from the models which best fit the actual propagation numbers found in the Yahoo! Go field data. The results of the three fit measures are shown for the RTR-model (which has been fitted using single-decimal parameters), and four single-parameter models. The first single-parameter model assumes all sub-processes are homogeneous and have the same effect and parameter ($\alpha = \phi = \eta$) (and has been fitted using single-decimal parameters), the second single-parameter model is driven by radiation and assumes the other sub-processes always occur ($\phi = \eta = 1.0$) (and has been fitted using two-decimal parameters), the third single-parameter model is driven by transmission and assumes the other sub-processes always occur ($\alpha = \eta = 1.0$) (and has been fitted using two-decimal parameters) and the last single-parameter model is driven by reception and assumes the other sub-processes always occur ($\alpha = \phi = 1.0$) (and has been fitted using two-decimal parameters).

	RTR-model	**Single-parameter models**			
Fit measure		*Homogeneous sub-processes*	*Driven by Radiation*	*Driven by Transmission*	*Driven by Reception*
RMSE	684.2	1374.0	1297.0	362381.2	1276.0
(relative error)		*(200.8%)*	*(189.6%)*	*(52964.2%)*	*(186.5%)*
LRMSE	0.982	3.986	2.113	10.659	1.929
(relative error)		*(405.9%)*	*(215.2%)*	*(1085.4%)*	*(196.4%)*
RRMSE	0.855	1.680	1.300	1.966	1.321
(relative error)		*(196.5%)*	*(152.0%)*	*(229.9%)*	*(154.5%)*
AIC	52.68	54.26	53.80	98.86	53.67

S1: The Yahoo! Go service application

In the first section of this paper, we analyzed the feasibility of using three sub-processes to model propagation. In order to do so we need to be able to distinguish the three sub-processes not only conceptually but also in practice. Therefore in this part of the paper we consider a real-life propagation phenomenon: the adoption of the Yahoo! Go 2.0 application Yahoo! (2010). Yahoo! Go can best be described by quoting the authors who initially collected this data (Aral et al., 2009): "*The Yahoo! Go is a mobile software application designed to deliver personalized content to users devices and to enhance mobile search capabilities. Yahoo! Go provides personalized information across several domains, including news, sports, weather, and financial information, as well as access to e-mail, calendaring, location based services, photo sharing, and web search services.*" The Yahoo! Go dataset includes detailed demographic and daily usage behavior data for approximately 27 million users, including geographic location and demographic data, instant messaging (IM) usage behavior, PC usage behavior, mobile usage behavior, Go usage behavior, and product adoption date for October 2007. A detailed description of the data of this dataset can be found in the supplementary information of the work of Aral et al. (2009). Only a sub-set of this information is used in our study; in order to capture the propagation process in this data set we solely use the daily number of instant messages among Yahoo! users (aggregated weekly) to construct a network of interactions, and the adoption of the Yahoo! Go 2.0 application as the 'behavior' which spreads across this network.

In our study a distinction is made between adoption and propagation. Whereas adoption can refer to starting to use the Yahoo! Go application —which can be driven by any form of external stimulus— we consider adoption as propagation only where it is potentially driven by direct influence from someone else in the actor's immediate neighborhood. Therefore propagation relates to only those changes of state (adoptions) which occur when an alter of the adopter has the infected state.

Descriptive Information for the Yahoo! Go dataset

The Yahoo! Go 2.0 dataset consists of a total of approximately 27 million unique users: the descriptives of this dataset can be found in Table 4.6. As we are considering both the behavior of users and the structure of the user community over a longer period of time, a check for structural changes during that observation period is required. The descriptive analysis of the data shows that not all actors are active during each of the time frames

(weeks). We also find a relatively stable growth in the number of active actors during each time frame, suggesting that during the period covered by the data set the Yahoo community, i.e. the number of actors who actively used the Yahoo Instant Messenger (IM), has been growing. The same trend can be found when considering the total number of IM messages sent during each time frame. The increase in terms of the number of messages sent, however, seems to be far larger than that found in the number of users. As a consequence the average number of messages per user is also found to increase over time.

Table 4.6: Descriptive information from the Yahoo! Go 2.0 dataset by week

Variable	Week 1	Week 2	Week 3	Week 4
Active IM users	10,431,939	11,185,637	11,918,764	12,018,406
Total IM messages	1,160,240,264	2,041,763,217	2,757,450,647	3,023,659,831
Avereage IM messages per user	111.2200	182.5344	231.3537	251.5858
Average in-degree	4.514386	4.891909	5.015574	5.052724
Average out-degree	4.510625	4.844500	4.975948	5.011638
Average total degree	3.784815	4.078306	4.196741	4.233276
Average edge weigth	29.38585	44.75740	55.12700	59.43051
Mode edge weight	6	11	14	15
Mode in-degree	1	1	1	1
Mode out-degree	1	1	1	1
Mode total degree	1	1	1	1
New adoptions	186832	70757	62962	63916
Propagation	5368	984	404	319

Looking at the average degrees over time once again corroborates the trend of slight growth, as positive trends are found in average in-degree, average out-degree and average total degree. Similarly we find trends of growth in the average and mode of the weight of edges (number of messages per edge), suggesting that not only has the community been growing but also that the intensity of use has increased during the observation period.
While we consider the messages of users as the vehicle for propagation, this would suggest that simply because of an increase in activity in the community the absolute numbers for propagation in the later time-steps might be inflated. While the adoption number might be affected by the increasing network size, it is assumed that the network size has no impact on the mechanism of propagation. The chance of propagation are independent of the network size therefore increase in network size is expected to influence the propagation process characteristics (parameters).
When we look at the mode of the degree measures, we see that their values remain stable at the value of 1, suggesting the degree distribution to be highly skewed. Plotting the degree on a log-log plot (see Figures 4.10, 4.11, 4.12 and 4.13), indeed reveals strongly

skewed distributions in each period. These distributions seem to follow a power-law, suggesting that the IM network has in fact a scale-free structure. Scale-free networks are often associated with large hubs. These hubs play a crucial role in the propagation process as they are more likely to be connected to infected actors (high in-degree), making them prone to be infected, and have many alters (high out-degree), allowing them to cause widespread propagation once infected.

In the Yahoo IM network, however, there seem to be no such hubs. When one examines the correlation between in- and out-degree (Figures 4.14, 4.15, 4.16 and 4.17 show the log-log plot of the degree correlations) it reveals that the upper-right quadrant is completely empty, indicating that there are no actors with both extremely high in- and out-degree. Hence there are no actors who seem to fit the previously described hub role, a characteristic which might hamper rapid propagation in this network.

S2: The general RTR-model of propagation

We put forward a generic model for studying propagation incorporating three sub-process; Radiation, Transmission and Reception. Based on these sub-processes we formulate a model of propagation consisting of three distinct functions; one for each of the sub-processes. Hence the generic RTR-model of propagation consists of:

1. a Radiation function
2. a Transmission function
3. a Reception function

The propagation process has been defined as *the process by which the change in state of one (or more) vertices causes the change in the state of one (or more) of its alters.* The general RTR-model allows for modeling the propagation mechanism in a simple but straightforward way. In this model we assume that there exists an underlying network structure on which the propagation process will take place. This network structure can be considered as a graph $G = (V, E)$ with vertices $i \in V := \{1, \ldots, n\}$ and an edgeset $e \in E := \{1, \ldots, m\}$. All edges are assumed to be directed and weighted, where $w_{ij,t}$ denotes the weight of the edge from vertex i to vertex $j$on time t, and $[W_t]$ is the matrix containing all edge weights on time t. All vertices i at any point in time have a state $s_{i,t}$, and $[S_t]$ is the matrix containing the state all actors at time t ($s_{i,t} \in [S_t]$). The starting state of the system can thus be denoted as $[W_0]$ and $[S_0]$, this starting state is assumed

to be stable.

The definition of propagation used in this paper considers it to be a process which is initiated by the change in the state/behavior of a certain actor A. When the system is perturbed this can be measured by a change in the state of (part of) the set of vertices V on time $t = 0$ ($\Delta S_{V,0}$). This perturbation in turn can destabilize the system and consequently result in a propagation. We speak of propagation only when the state of vertex set V changes during a time-step in the model ($S_{V,t} \neq S_{V,t+1}$).
The propagation process is assumed to take place in discrete time. In every time unit ($t \rightarrow t + 1$) the three aforementioned sub-processes occur. First a change in the state of an vertex i will be send to the outgoing edges of this vertex in the form of a signal (Radiation), second this signal will be transferred over the edge towards the alter (j) (Transmission), and third the incoming signal(s) will reach alter j and (potentially) result in a change in the state of that alter (reception) (see Figure 4.18).

The radiation sub-process

The radiation function describes how a change in the state of a vertex i ($\Delta s_{i,t}$) results in signal being sent out towards the outgoing edges of that vertex at time t ($E^{out}_{i,t} \subset E | w_{ij,t} > 0$), it can therefore generally be described by:

$$\Delta s_{i,t} = \sum_{e \in E^{out}_{i,t}} \alpha_{i,e,t} \times p^{out}_{i,e,t} = \sum A_{i,t} \times P^{out}_{i,t} \tag{4.4}$$

In which $A_{i,t}$ is a vector of radiation parameters ($\alpha_{i,e,t}$) of actor i at time t towards its outgoing edges ($e \in E^{out}_{i,t}$) at time t ($A_{i,t} = (\alpha_{i,e,t}) | e \in E^{out}_{i,t}$), and $P^{out}_{i,t}$ is a vector containing the signals sent from vertex i to all outgoing edges ($E^{out}_{i,t}$) at time t ($P^{out}_{i,t} = (p^{out}_{i,e,t}) | e \in E^{out}_{i,t}$). The radiation parameters ($\alpha_{i,t}$) can be influenced by many aspects, it makes sense to consider it as a function of a set of actor specific characteristics, which can consequently amplify or dampen the extent to which a change in state results in radiation.

One should note that this formulation assumes that a change in state results in a signal *only* in the consecutive time unit, and consequently the model considers only the current changes in state and has no memory of what has happened in previous time units. Following the work of Dodds and Watts (2004, 2005) we generalize this simple version of the

radiation function to also include memory, effectively stating that a change in state can have effects beyond the consecutive time unit. This leads us to extend equation 4.4 into:

$$\Delta s_{i,t} = \sum_{t'=t-T_{rad}+1}^{t} \sum \left(A_{i,t'} \times P_{i,t'}^{out} \times (\tau_{rad})^{(t-t')+1} \right) \tag{4.5}$$

In which T_{rad} is the memory duration for radiation, the number of time units that a change in state will yield signals ($T_{rad} \in \{1, \ldots, t\}$). And τ_{rad} is the memory inflation factor for radiation, the extent to which previous signals are remembered. The memory inflation factor can in theory have any positive value; although values ranging between 0 and 1 are most likely as they refer to a process in which signals are forgotten over time. Values of τ_{rad} higher than 1 suggest that signals have an increasing effect over time, and hence the effect of a change in state will be amplified over time.

This formulation has some build in boundary conditions; it assumes that any fluctuation in the state of i will fully radiate into one (or more) signals to the outgoing edges. However, social actors are often bound by limited responsiveness, meaning that the their capacity to process and observe changes in their state are limited. As in many propagation models the actors refer to social entities (which in itself are complex system with some inherent buffering capacity) it can be argued that vertices have some absorptive capacity for changes in state.Therefore the assumption that all changes in state will result in radiation might be too strict to capture real-life radiation dynamics. In order to make the model more generally applicable the model should able to capture the notion that there can be some absorption in the system. This is done by stating that only if a change in state is large enough to pass the radiation threshold u ($\Delta s_{i,t} \geq u$) there will be any radiation. Including this notion into equation 4.5 yields:

$$\sum_{t'=t-T_{rad}+1}^{t} \sum \left(A_{i,t'} \times P_{i,t'}^{out} \times (\tau_{rad})^{(t-t')+1} \right) = \begin{cases} \Delta s_{i,t} & \text{if} \quad \Delta s_{i,t} \geq u \\ 0 & \text{if} \quad \Delta s_{i,t} < u \end{cases} \tag{4.6}$$

In which the radiation threshold (u) can have any positive value. Equation 4.6 describes the radiation sub-process in its most generic form. However radiation alone is not sufficient to model the propagation process. In order to model the propagation process both the other sub-processes (transmission and reception) need to be defined; therefore in the next section the transmission process will be considered.

The transmission sub-process

The second sub-process of propagation, the transmission process, considers how the signal coming *from* a vertex (i) is transmitted over an edge towards an alter (j). This process can therefore be formulated as follows:

$$p^{out}_{i,e,t} = \phi_{e,t} \times p^{in}_{e,j,t} \tag{4.7}$$

In which $p^{in}_{e,j,t}$ is the signal coming from edge e arriving at alter j at time t, and $\phi_{e,t}$ is the transmission parameter of edge e on time t. Similarly to the radiation parameter, the transmission parameter ($\phi_{e,t}$)can be considered as a function of edge characteristic which influence transmission. Also similar to the radiation sub-process it can be assumed that an edge also has a memory. While this memory does not stem from behavior (an edge does not describe entity but a relationship and hence cannot act) it relates to the notion of capacity. It can be best described by assuming an edge to be a pipeline which has a certain capacity to transport signals, if a signal does not get transported in a previous time unit, the signal should go somewhere. We can assume that it disappears (no memory) or that it (partially) remain in the pipeline suggesting that the transmission at time t is influenced by previous signals. Including this notion of 'memory' in the transmission sub-process yields:

$$p^{out}_{i,e,t} = \sum_{t'=t-T_{tra}+1}^{t} (\phi_{e,t'} \times p^{in}_{e,j,t'} \times (\tau_{tra})^{(t-t')+1}) \tag{4.8}$$

In which T_{tra} is the memory duration for transmission, and τ_{tra} is the memory inflation factor for transmission. Most existing propagation models do not describe the transmission process and hence implicitly assume a special case in which transmission has no effects ($\phi = 1$, $T_{tra} = 1$ and $\tau_{tra} = 1$). While this assumption in many cases makes sense, in this dissertation transmission is described in this more generic form for two reasons. First, it allows for capturing the effects where the transmission *does* causes signals to be dampened (or amplified), think for example of the inclusion of noise in communication, the notion of package loss in IT networks or the notion of resistive losses in power transport. Second, this formulation allows for easily incorporating the (potential) effects of edge-weights in the propagation model. The edge weights can be easily included in the transmission parameter $\phi_{e,t}$ hence this generic form allows for studying propagation also in weighted networks, something which is more difficult in traditional propagation models.

Equation 4.8 describes the generic formulation of the transmission sub-process, however

for propagation to occur still the reception needs to be considered; this is being doing in the following section.

The reception sub-process

The third sub process of propagation, the reception process, describes how incoming signals translate into a change in the state of a vertex, in its simplest form it could be formulated as:

$$\eta_{j,t} \times \sum_{e \in E^{in}_{j,t}} (p^{in}_{e,j,t}) = s_{j,(t+1)} - s_{j,t} \tag{4.9}$$

In which $\eta_{j,t}$ is the reception parameter considering vertex specific attributes influencing the reception process of vertex j on time t, and $E^{in}_{j,t}$ is the set of incoming edges to actor j on time t. Note that this way of modeling assumes that all incoming signals are combined and have one single parallel effect on a vertex. This formulation therefore assumes that there is no discrimination on sources of a signal, a claim which especially in social systems might be dubious. Formulating the generic form of the RTR propagation model therefore requires a slightly more complex formulation. Consider a vector $\Psi_{j,t}$ which contains a set of $\eta_{j,t}$ for each incoming edge ($\Psi_{j,t} = (\eta_{e,j,t}) | e \in E^{in}_{j,t}$), in which all reception parameters ($\eta_{e,j,t}$) are dependent on the source, the edge, where the signal is coming from. This would yield the following formulation for reception:

$$\sum (P^{in}_{j,t} \times \Psi_{j,t}) = s_{j,(t+1)} - s_{j,t} = \Delta s_{j,t} \tag{4.10}$$

In which $P^{in}_{j,t}$ is a vector containing all the signals of all incoming ties of actor j at time t ($P^{in}_{j,t} = (p^{in}_{e,j,t}) | e \in E^{in}_{j,t}$). Note that while this model assumes that incoming signal can be added up, however more complex function of aggregating the incoming signals could be used if one has indications that the process being studied has some none-linear dynamics. Also note that this formulation assumes that signals are processed simultaneously, rather than in sequence.

The RTR-model assumes that aggregated signals have one single combined effect on a vertex per time unit. One could make time units infinity small and thus allow actors to respond to (and send out) signals sequentially (rather then in parallel). This is for example done when considering the propagation process as a Markov-chain. Such an approach is however undesirable for three reasons:

- Processing signals sequentially requires an additional set of assumptions; it requires requires a very clear set of assumptions on the sequencing of signals (which in itself is so complex that the treatment is worse than the problem).
- It assumes that there are no interactions among multiple signals, as only one signal can be processed at each time unit. Therefore it will be unable to cope with more complex notions of propagation (Centola and Macy, 2007).
- Sequential processing assumes perfect responsiveness of the system.

While the first two factors can be a simple design choice, the latter poses a bigger problem. Perfect responsiveness assumes that actors are able to observe and act upon any change that occurs instantaneously. Especially in social systems this might not be a correct representation of reality, as it is more likely that it takes time to both to observe and respond to signals. This is suggesting that there is time of signals to aggregate before being processed.

Actors may not only be bounded in the speed of processing, but also in their capacity to do so. The section on the radiation process has put forward the notion that entities are likely to have some buffering capacity, and hence will not convert any change in state towards outgoing signals. A similar argument can be made for the reception sub-process; actors are unlikely to respond to any signal, it needs to be sufficiently large. Indeed such a notion is the very foundation on which the previously introduced threshold models are build. A generic form of the RTR-model therefore should include such absorptive capacity also in reception sub-process. This is done by incorporating a reception threshold (q) into the reception function in a similar fashion as has been done in the radiation sub-process. Resulting in the following formulation:

$$\Delta s_{j,t} = s_{j,(t+1)} - s_{j,t} = \begin{cases} \sum (P^{in}_{j,t} \times \Psi_{j,t}) & \text{if} \quad \sum (P^{in}_{j,t} \times \Psi_{j,t}) \geq q \\ 0 & \text{if} \quad \sum (P^{in}_{j,t} \times \Psi_{j,t}) < q \end{cases} \tag{4.11}$$

Where q is the reception threshold which can vary from 0 to ∞. The reception threshold q is applied to the aggregate of signals for two reasons. First, the threshold is something which is bound to the entity 'actor' rather than its ties, as it describes an actor's bounded capacity. It is likely the result of entity characteristics, and hence should apply to the entity and not to its edges. One could argue that there may be different susceptibility depending on the source of a signal, which would be a valid claim, this however can already be captured by the vector $\Psi_{i,t}$ capturing the reception paramateres for different edges.

The second reason for applying the threshold after aggregation builds on the notion that a small signal might not yield a state change but many of them combined might in fact do so. Capturing this aggregation effect can only be done by applying the threshold after aggregation.
Again, similar to the previous sub-processes also during reception memory can play a critical role. Like most commonly used models of propagation it can be assumed that entities respond to signals independently, and hence signals from the past play no role in the current reception process (no memory). But especially in social systems such claims might not be realistic as actors tend to learn. This suggests that what has happened in the past does in fact matter for their current decisions (memory). Including this notion of memory into the reception function yields:

$$\Delta s_{i,t} = \begin{cases} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) & , \text{ if} \\ \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) \geq q & , \text{ otherwise} \\ 0 \end{cases} \tag{4.12}$$

Equations 4.6,4.8 and 4.12 combined describe the RTR-model in its most general form. Describing propagation processes using this generalized RTR-model not only allows for the modeling of a multitude of different types of propagation mechanisms, each with their own characteristics, it also gives researchers a clear overview of what the overlap and differences are between these different propagation processes, and hence facilitates (potential) comparison between different processes.

S3: The stochastic RTR-model

The general RTR-model describes the state of vertex as a continuous variable, however, in many propagation settings this might be a too complex representation of reality. Many studies have described processes in which the state of an actor in fact can be described by a binary variable. In these models an actor can have only one of two states ($s_{i,t} =\in \{0, 1\}$). Examples are the spread of disease in which actors are either sick or not (e.g. (Rahmandad and Sterman, 2008)), the adoption of a innovations or certain behavior, again actors either have adopted or have not (e.g. (Aral et al., 2013)) or break down or stay operational (e.g. (Buldyrev et al., 2010)). A binary state version of the RTR-model can be easily obtained by adjusting the radiation and reception sub-processes and incorporating the notion that

any change in state will be of size 1, and that once actors are in state 1 their state cannot increase further. Incorporating this notion we can rewrite the radiation and reception functions to fit a binary state model; radiation becomes:

$$\sum_{t'=t-T+1}^{t} \sum \left(A_{i,t'} \times P_{i,t'}^{out} \times (\tau_{rad})^{(t-t')+1}\right) = \begin{cases} 1 & \text{if} \quad \Delta s_{i,t} \geq u \\ 0 & \text{if} \quad \Delta s_{i,t} < u \end{cases} \tag{4.13}$$

In which u once again is the radiation threshold.
Similarly the reception sub-process consequently can be formulated as:

$$\Delta s_{i,t} = \begin{cases} 1 & \text{if} \quad s_{j,t} = 0 \quad \text{and} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) \geq q \\ 0 & \text{if} \quad s_{j,t} = 0 \quad \text{and} \sum_{t'=t-T_{rec}+1}^{t} \sum \left(P_{j,t'}^{in} \times \Psi_{j,t'} \times (\tau_{rec})^{(t-t')+1}\right) < q \\ 0 & \text{if} \quad s_{j,t} = 1 \end{cases} \tag{4.14}$$

As the transmission function does not take into account the state of the vertices it will remain as described in the generic RTR-model (Equation 4.8). Clearly, studying scenarios in which the only potential change in state does not pass the radiation threshold ($\Delta s_{i,t} = 1 < u$) is trivial as they will never result in propagation. However, to make sure the model mutually exclusive and collectively exhaustive this option is not to be excluded from the model description. Also doing so facilitates the conversion into a stochastic version of this model, which will be done below.

Taking a closer look at the literature studying binary state models reveals that nearly all of them consider the propagation process to be a stochastic process rather than the deterministic approach we have adopted thus far. Commonly they model the propagation process by (a variation on) the traditional SI models commonly studied in epidemiology. In these models actors are either **S**usceptible, **I**nfected (or **R**emoved), hence resulting name SIS/SIR model. These models describe a process by which actors are changing from Susceptible to Infected with a certain infection rate (λ) and a process by which actors change from Infected to Susceptible/Removed by certain recovery rate (ρ) or death rate (γ), these processes are considered to be a stochastic and dependent of the interactions. While thus far the RTR-models discussed are deterministic in nature, we can easily convert the the binary state version of the model into a stochastic version similar to the SIS/SIR models. The binary nature of the state of the vertices makes it trivial to consider the size of a signal, the only relevant criteria becomes whether it passes the threshold or not.

Therefore rather than considering size of a signal we can consider the probability that it passes the threshold. This effectively changes the emphasis of the model from size (read deterministic) to probabilities (read stochastic), facilitating the comparison of the RTR-model to traditional SIS/SIR models.

This stochastic RTR-model resembles traditional SIS models but differs in two respects; First it considers the local interaction, rather than the mean field level dynamics, and second it includes the three sub-processes for propagation rather than a single process.

We consider there to be a probability of success for each of the sub-processes, and thus decompose the propagation probability (λ) into three distinct probabilities (α, ϕ, η), one for each sub-process. For each sub-process a likelihood that the signal is being forwarded to the next stage of the propagation process can be included. Propagation can consequently be modeled by means of three Bernoulli distributions. The first describes the probability of successful Radiation (α), the second of successful Transmission (ϕ), and the third of successful Reception (η). The general RTR-model in cases of binary vertex states can thus be easily transformed into a stochastic variant in which we assume that some function of the radiation/transmission/reception parameters, will yield a probability of it successfully occurring.

In a stochastic version of the RTR-model the radiation likelihood describes the probability that a change in state (which can only be of size 1) will be larger than the radiation threshold, and thus yields any signal towards the outgoing edge(s). This probability is only influenced by a function of the radiation properties ($A^*_{i,t} = f(A_{i,t}) = (\alpha^*_{i,e,t})|e \in E^{out}_{i,t} = f(A_{i,t})$) of the actor sending the signal(i) and the memory in the system. Therefore it can be formulated as:

$$p^{out}_{i,e,t} \sim Bern(p) \quad \text{in which} \quad p = 1 - \prod_{t'=t-T_{rad}+1}^{t} \left(1 - \left(\Delta s_{i,t'} \times \alpha^*_{i,e,t'} \times (\tau_{rad})^{(t-t')+1}\right)\right) \quad (4.15)$$

The SIS/SIR models can be denoted as a special case of this general stochastic form. The traditional SIS models make two assumptions; first it assumes no memory in the system, second it assumes that radiation is caused by the state itself rather than the change of this state (once an actor is sick it has a chance to radiate). These two assumptions allow us to reduce equation 4.15 to:

$$p^{out}_{i,e,t} = \begin{cases} \sim Bern(\alpha^*_{i,e,t}) & \text{if} \quad s_{i,t} = 1 \\ 0 & \text{if} \quad s_{i,t} = 0 \end{cases} \quad (4.16)$$

When the propagation process is assumed to be stochastic the transmission sub-process describes the chance that a radiated signal is transmitted over an edge. This chance depends on the presence of an incoming signal, a function of the edge specific characteristic ($\phi^*_{e,t} = g(\phi_{e,t})$) and the memory in the transmission. It can therefore be formulated as:

$$p^{in}_{e,j,t} \sim Bern(p) \quad \text{in which} \quad p = 1 - \prod_{(t'=t-T_{tra}+1)}^{t} (1 - (\phi^*_{e,t'} \times p^{out}_{i,e,t'} \times (\tau_{tra})^{(t-t')+1})) \tag{4.17}$$

Once more the SIS type of models can be considered as a special case of this generic stochastic transmission process, in which there is no memory. Combining this with the notion that in stochastic processes the incoming signals are pulses either of size 1 or 0 ($p^{out}_{i,e,t} \in \{1, 0\}$) and that the latter case is a trivial one as there is nothing to propagate, enables us to write transmission in these settings as:

$$p^{in}_{e,j,t} \sim Bern(\phi^*_{e,t}) \quad \text{if} \quad p^{out}_{i,e,t} = 1 \tag{4.18}$$

The reception sub-process in a stochastic propagation scenario refers to the chance that the sum of the incoming signals aggregates into a signal that in fact surpasses the reception threshold. It will consequently can result in a state change of the receiving actor. This clearly depends on the state of the receiving actor (as it need to be able to change state) and a function of the reception parameters ($\Psi^*_{j,t} = h(\Psi_{j,t'}) = (\eta^*_{e,j,t})|e \in E^{in}_{j,t})$) and the memory in the reception sub-process. Rewriting the binary reception formulation (equation 2.19) into a stochastic version yields:

$$\Delta s_{j,t} = \begin{cases} \sim Bern(p) & \text{if} \quad s_{j,t} = 0 \\ 0 \quad \text{if} \quad s_{j,t} = 1 \end{cases} \tag{4.19}$$

In which $\quad p = 1 - \prod_{t'=t-T_{rec}+1}^{t} \prod (1 - (P^{in}_{j,t'} \times \Psi^*_{j,t'} \times (\tau_{rec})^{(t-t')+1}))$.

The SIS models again are a special case of this formulation in which there is no memory. To rewrite the reception sub-process following the SIS model logic the same logic as applied for the transmission process ($p^{in}_{e,i,t'} \in \{1, 0\}$) can be used. Consequently the reception sub-process following the SIS logic can be written as:

$$\Delta s_{j,t} = \begin{cases} \sim Bern(1 - \prod (1 - \Psi^*_{j,t})) & \text{if} \quad s_{i,t} = 0 \\ 0 & \text{if} \quad s_{i,t} = 1 \end{cases} \tag{4.20}$$

S4: Brute-force fitting

During the first part of our study we use a brute-force simulation approach to reproduce the actual propagation number found when considering the propagation of the Yahoo! Go 2.0 application. We distill the actual propagation and the network structure at the start of our observation period from the data and use this information as the input for our set of simulations. Given this starting scenario, we run a series of simulations in which we vary the propagation mechanism, namely the radiation, transmission and reception parameters ((α, ϕ, η)). While many arbitrary heuristics could be used to find the optimal set of parameters (radiation, transmission and reception probabilities), all of these heuristics require sequential fitting, which in turn means that we have to include a prioritization in the sequence in which each of the parameters is fitted. This prioritization might cause the heuristic to get stuck in a local minimum, strongly biasing the fitting outcomes. To circumvent this problem a brute-force simulation approach is used in this study.
The brute-force approach implies doing a complete sweep of the parameter space in order to find the global minimum. As the RTR-model uses three distinct parameters, each decimal added to the parameter estimation will increase the number of scenarios in the parameter space by a factor of 1,000 (10^3). Therefore the brute-force approach has considerable implications for the processing time, forcing a trade-off between scope and level of detail. For this reason in the initial set of simulations a single decimal detail level has been chosen. Resulting in 1,000 scenarios with different mechanisms (division into Radiation, Transmission and Reception).
We simulate the stochastic propagation process on the given network structure (drawn from the actual Yahoo! Go data) for each scenario with varying propagation parameters. Using bootstrapping we run each scenario multiple times (5) and measure the average amount of propagation (number of new adopters) per week in each scenario. We compare these numbers to the numbers for adoption distilled from the actual data, and calculate the fit between the simulated and actual propagation.

Fit measures

There exist many different potential fitting measures, three of them are used to calculate fit in our study. First, we use the traditional root-mean-squared error (RMSE), and then, because there are big variations in the absolute propagation numbers (the propagation in the first week of observation is an order of magnitude bigger that the propagation in the last week), we also include both the logged root-mean-square error (LRMSE) and the relative root-mean-square error (RRMSE). These measures are formulated as follows:

$$\text{RMSE} = \sqrt{\sum_n (\hat{Y} - \bar{Y})^2} \tag{4.21}$$

$$\text{LRMSE} = \sqrt{\sum_n (\log \hat{Y} - \log \bar{Y})^2} \tag{4.22}$$

$$\text{RRMSE} = \sqrt{\sum_n (\frac{\hat{Y} - \bar{Y}}{\bar{Y}})^2} \tag{4.23}$$

For all scenarios in the parameter space (10^3), we calculate the fit measures. We select the best fitting scenario by minimizing the fitting error, and consider this scenario to be the optimal scenario given this fit measure.
What becomes apparent when considering the fitting results (Table 4.4) is that each fit measure results in a different optimal decomposition into sub-processes. Clearly each measure emphasizes a different aspect of the propagation curve (Figure 4.19), suggesting that finding the optimal decomposition depends very much on the criteria for fitting. As we have no preference for either of these criteria or measures and are interested in making a general statement in the remainder of the study we keep using all of the fit measures.

What is more important is that we can in fact parametrize the different sub-processes and the decomposition of the propagation based on simple adoption data. As we are interested in the performance of the RTR-model (in fitting the data), we should compare its results with traditional single-parameter propagation models, as these are the current standard in literature. We can take multiple approaches to doing so as current literature is often unclear on the type of sub-process considered. We chose an approach which is twofold and results in four single-parameter models. In the first single-parameter model we assume homogeneity in the sub-processes, with each process being equally important and equally strong ($\alpha = \phi = \eta$), while this model still assumes three sub-process the properties of all sub-process are linked, enabling us to capture all three processes by a single parameter. The other three single-parameter models assume that a single sub-process (Radiation, Transmission or Reception) is responsible for the propagation process, and that the other sub-processes are occurring by default, setting their probability parameters to 1.0, effectively this maps the RTR-model onto a single parameter (and sub-process) model. This results in single-parameter model 2 which is driven by Radiation ($\alpha = ?, \phi = 1.0, \eta = 1.0$), single-parameter model 3 which is driven by Transmission ($\alpha = 1.0, \phi = ?, \eta = 1.0$), and single-parameter model 4 which is driven by Reception ($\alpha = 1.0, \phi = 1.0, \eta = ?$). For each

of these models again the same brute-force-fitting approach has been used, resulting in the initial comparison in Table 4.7.

Table 4.7: This table shows the errors obtained from the models which best fit the actual propagation numbers found in the Yahoo! Go field data (all model parameters estimated at the single decimal level). The results of the three fit measures are shown for the RTR-model, and four single-parameter models. The first single-parameter model assumes all sub-processes are homogeneous (have the same effect and parameter), the second single-parameter model is driven by radiation (and assumes the other sub-processes always occur ($\phi = \eta = 1.0$)), the third single-parameter model is driven by transmission and assumes the other sub-processes always occur ($\alpha = \eta = 1.0$), and the last single-parameter model is driven by reception and assumes the other sub-processes always occur ($\alpha = \phi = 1.0$).

	RTR-model	**Single-parameter models**			
Fit measure		*Homogeneous sub-processes*	*Driven by Radiation*	*Driven by Transmission*	*Driven by Reception*
RMSE	684.2	1374.0	199941.5	2329788.0	200157.3
LRMSE	0.982	3.986	9.539	13.900	9.540
RRMSE	0.855	1.680	1.945	1.988	1.945

Robustness of fitting results

The initial results (Table 4.7) show that the RTR-model is able to significantly outperform any single-parameter model in terms of fitting propagation data. However, with exception of the homogenous scenario, all single-parmeter models have only one sub-process and thus one parameter which can change. Therefore, in part, these results can be attributed to the increase in parameters used in the RTR-model. This increase has two effects: the degree of freedom in the RTR-model is higher than for the single-parameter models and the level of detail to which the overarching propagation parameter (λ) can be estimated is increased. In order to cope with these effects we take two separate actions.

First, as the increase in the number of parameters translates directly into an increase in the degree of freedom in the model, one would expect the fit to increase as well. Generally one would argue that using more parameters is always better in terms of fitting a model to data. The trade-off being made is the risk of over-fitting by using too many parameters in the model versus not having a sufficient number of parameters to draw the correct conclusions. While the latter would be primarily a conceptual argument which is generally hard to check, the former has received some attention among statisticians. There are several measures controlling the model fit for the number of parameters used.

The Akaike information criterion (AIC) (Akaike, 1974) is one of the most commonly used measures in this area. It penalizes for the number of parameters used to generate the fitting error, and by doing so provides a more balanced way of comparing models with varying numbers of parameters. As we are in fact comparing models with different numbers of parameters we apply the AIC in order to give a more fair comparison and reduce the risk of over-fitting.
Second, the increase in the number of parameters has a second effect: it increase the level of detail. We divide the traditional propagation parameter into three distinct parameters, and measure each on a single decimal detail level. Consequently we are comparing models which describe the overall propagation parameter on a single decimal (which is the case when we use a single-parameter model) with a model which can estimate the propagation parameter at three decimals (one for each sub-process, 0.1^3). While AIC already controls for this aspect, on top of this correction we increase the level of detail for the single-parameter benchmark models (with exception of the homogenous scenario) by a factor of 10. Consequently these models are estimated on the 0.01 parameter detail level when compared to the RTR-model. The results of these corrections can be found in Table 4.5.

S5: Simulation using the RTR-model

In the second section of the paper the simulation model is used to show the impact of dividing the propagation mechanism into the three sub-processes. To allow propagation to be modeled some constraints are required, and a network has to be generated on which to run the propagation process. In our simulations the following constraints are used:

- The state of an actor is either 1 (infected) or 0 (susceptible); the default is that actors are susceptible
- The weight of all edges is equal and one, $w_(ij,t) = 1$
- The edges are undirected, resulting effectively in a unweighted undirected network
- The number of nodes in the network is fixed, $n = 10,000$
- Each node has on average three edges, $m = 3$
- The probability of recovering from a disease is fixed, $\rho = 0.2$
- The overarching propagation probability is fixed, $\lambda = 0.192$
- The propagation is initiated from a single seed

- The simulation will run for 30 time-steps

As the starting point for the simulation, a single undirected scale-free network based on the Barabasi-Albert Barabási and Albert (1999) approach is generated, in which each vertex has on average three ($m = 3$) edges. This structure remains constant over all simulations, effectively removing any influence of network structure on the outcomes of the simulation. An infection is introduced to the network by randomly selecting a seed node and changing its state to Infected. This node starts radiating and thereby serves as the seed of the infection, and consequently the propagation process with given mechanism (pre-defined sub-process parameters) is simulated while the pervasiveness —the proportion of the population which is infected— is tracked over time.
In order to generate robust results we use bootstrapping, effectively repeating the process described above 100 times. The results are then averaged over this bootstrapped set of simulations, resulting in a single pervasiveness curve for each propagation scenario.

Validating the RTR simulation model

In the first set of scenarios we investigate the notion that a propagation process can be decomposed into sub-processes. While the chance of propagation has been conceptually claimed to be a product of three consecutive stochastic processes, in the initial stage we use simulation to test this claim. To do so we use a sample set of four scenarios where the propagation probability is equal for in each case ($\lambda_1 = \lambda_2 = \lambda_3 = \lambda_4 = 0.192$). These scenarios do differ in their mechanism, way in which they are decomposed into sub-processes. Scenario 1 is a process with low radiation, medium transmission and high reception ($\alpha = 0.4, \phi = 0.6, \eta = 0.8$); scenario 2 is a process with medium radiation, high transmission and low reception ($\alpha = 0.6, \phi = 0.8, \eta = 0.4$); scenario 3 is a process with high radiation, low transmission and medium reception ($\alpha = 0.8, \phi = 0.4, \eta = 0.6$); and scenario 4 is a process in which the sub-processes are homogeneous ($\alpha = \phi = \eta = 0.5769$). The initial results (Figure 4.20) show variations in propagation outcomes for these different scenarios; based on the conceptual decomposition argument, one would, however, expect there to be no differences between these models.

Further investigation (Figure 4.21) revealed that the variations found are in fact insignificant, which indicates that the limited amount of the bootstrapping might have caused the differences in the initial results. As these results were generated using a set of 100 bootstrapped simulations it is likely that variations have risen from either stochasticity or

sampling, causing differences in local structure surrounding the seed of infection. Therefore a second set of simulations has been run in which each node in the population is used as seed nodes. In this set of simulations each node is chosen once as the seed of infection. This resulted in a bootstrapped sample of 10,000 simulations —the increased sample size should reduce the stochastic problems— in which each actor has been used as seed node, removing all potential sampling bias. The simulation results (Figure 4.22) show that the pervasiveness curves for the three scenarios lie exactly on top of each other, confirming two things. First, it indicates that variation in propagation process outcomes can occur due to sampling and/or stochasticity, which has important implications for the generalization of propagation process outcomes, and supporting the notion that the seed plays a role in the eventual outcomes of propagation process (something which outside the scope of this paper). Second, it shows that on the mean-field all four scenarios yield a similar pervasiveness, supporting the notion that a propagation process can indeed be decomposed into three distinct sub-processes.

Simulating interventions using the RTR-model

We have shown that decomposing the propagation process into three sub-processes is both methodologically desirable, and conceptually valid. This allows us to use the RTR-model to simulate the impact of interventions in the propagation process. Once more the same four scenarios are used during the simulation of the interventions. We compare scenarios with different mechanisms (and corresponding decompositions) but similar propagation probabilities (λ). An intervention is included in the simulation by changing a single propagation parameter (either α, ϕ or η) during the simulation. We extend the number of time-steps (t) to 100, and at $t = 50$ a parameter of one of the sub-processes is reduced by the arbitrary amount of 0.2, which effectively means that the chance that this sub-process will take place successfully (i.e. result in a signal) is decreased by 20%. To avoid potential stochasticity or selection bias, the scenarios are once more bootstrapped 10,000 times, once for each potential starting seed.

Our simulations show the results of interventions in each of the sub-processes: radiation (Figure 4.3), transmission (Figure 4.4), and reception (Figure 4.2). Two things become apparent from these simulations.

First and foremost, these results support the notion that the mechanism of the propagation process plays a critical role in determining the effect of an intervention on the dynamics of the propagation process. As interventions are aimed only at changing the parameter of a single sub-process, our simulations show that to model the effect of such

interventions effectively one needs to be able to break down the propagation process into the sub-processes. Only once this decomposition is known can one estimate the impact of an intervention.

Second, the effectiveness of interventions varies a great deal, depending on the decomposition into sub-processes. This becomes even more apparent when we considering Figures 4.5,4.6,4.7 and 4.8 which capture the same adoption curse, but represent it per mechanism, consequently each line represents a distinct intervention (in either Radiation, Transmission or Reception). Scenario 1 is depicted in Figure 4.5, scenario 2 can be found in Figure 4.6, scenario 3 in Figure 4.7 and scenario 4 in Figure 4.8. When we consider scenario 4 (Figure 4.8), we find all interventions (in any single sub-process) to have an equal effect. Therefore in this specific scenario —which assumes homogenous sub-processes— any intervention will have the same effect, and consequently the distinction in sub-processes seems to bear no additional value. The remainder of the figures however show that there are differences between different decompositions when it comes to the effectiveness of interventions.

To get a feel for the extent to which these differences lead to over- or underestimation of intervention effects we take a closer look at some extreme scenarios. We again take the scenario with homogeneous sub-processes (scenario 4) as our benchmark, as this scenario fits the current state of most propagation research. We compare this scenario to two extreme scenarios: scenario 5, the 'worst case' most resilient scenario in which the parameter under intervention is initially maximized (1.0), and scenario 6, the 'best case' most volatile scenario in which the parameter under intervention is equal to the intervention size (0.2). The results of these simulations (Table 4.3, Figure 4.9) show that both under- and overestimation of the intervention effect are possible. In this specific example setting, based on the default scenario (scenario 4) one would expect a reduction in pervasiveness of 12.7%. In the most resilient scenario (scenario 5), one can see that pervasiveness is, however, only reduced by 6.1%, so by assuming sub-processes to be homogeneous one could overestimate the effectiveness of this intervention by 52.0%. In contrast, in the most volatile scenario (scenario 6), the infection is completely removed. The pervasiveness is thus reduced by 100%, indicating that in this case if one had used the default scenario the intervention effect would have been underestimated by 687.4%.

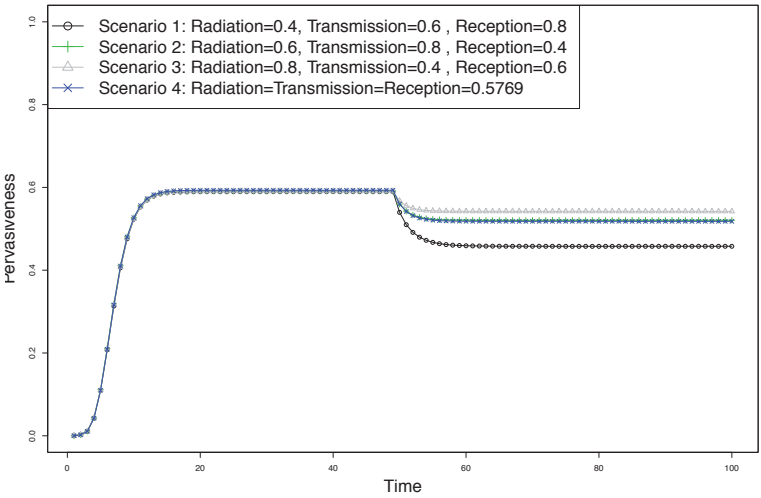


Figure 4.3: The pervasiveness over time is depicted for scenarios with the same overarching propagation likelihood ($\lambda = 0.192$) but with different decompositions into radiation (α), transmission (ϕ) and reception (η). The black open circles represent a process with $\alpha = 0.4$, $\phi = 0.6$ and $\eta = 0.8$. The green pluses represent a process with $\alpha = 0.6$, $\phi = 0.8$ and $\eta = 0.4$. The (grey) triangles represent a process with $\alpha = 0.8$, $\phi = 0.4$ and $\eta = 0.6$. The blue crosses represent a process with homogeneous sub-processes ($\alpha = \phi = \eta = 0.5769$). During the simulation at t = 50 an intervention (reduction of 0.2) in the **radiation** is applied

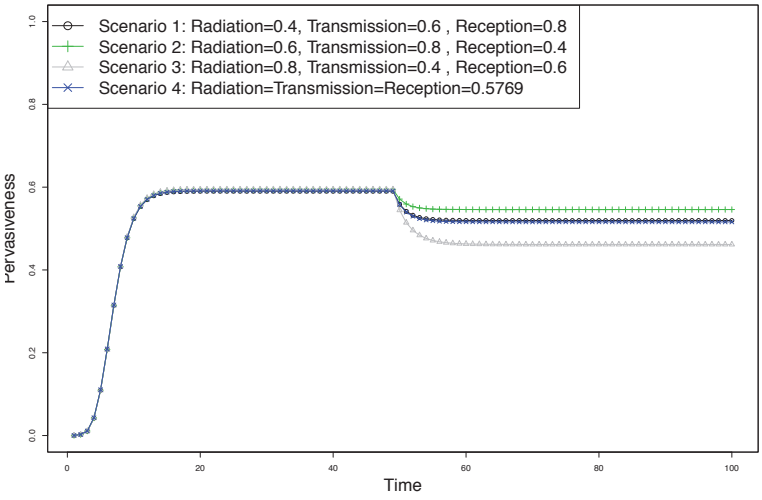


Figure 4.4: The pervasiveness over time is depicted for scenarios with the same overarching propagation likelihood ($\lambda = 0.192$) but with different decompositions into radiation (α), transmission (ϕ) and reception (η). The black open circles represent a process with $\alpha = 0.4$, $\phi = 0.6$ and $\eta = 0.8$. The green pluses represent a process with $\alpha = 0.6$, $\phi = 0.8$ and $\eta = 0.4$. The (grey) triangles represent a process with $\alpha = 0.8$, $\phi = 0.4$ and $\eta = 0.6$. The blue crosses represent a process with homogeneous sub-processes ($\alpha = \phi = \eta = 0.5769$). During the simulation at t = 50 an intervention (reduction of 0.2) in the **transmission** is applied

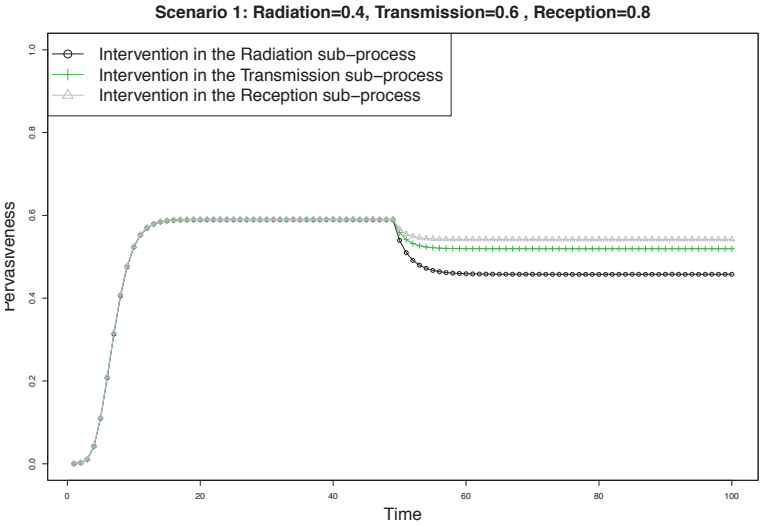


Figure 4.5: The pervasiveness over time is depicted for scenario 1 which is characterized by a propagation process with $\alpha = 0.4$, $\phi = 0.6$ and $\eta = 0.8$. Different interventions (-0.2) are applied in this scenario; the black open circles represent an intervention in the radiation parameter, the green pluses represent an intervention in the transmission parameter and the grey triangles represent an intervention in the reception parameter.

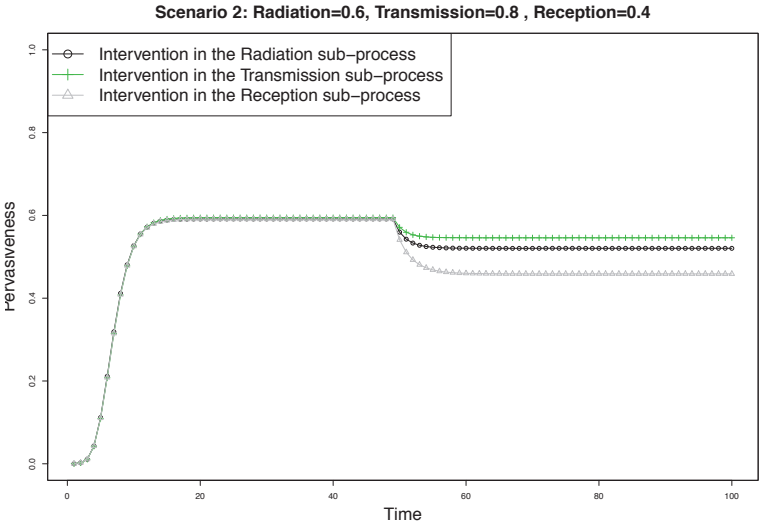


Figure 4.6: The pervasiveness over time is depicted for scenario 2 which is characterized by a propagation process with $\alpha = 0.6$, $\phi = 0.8$ and $\eta = 0.4$. Different interventions (-0.2) are applied in this scenario; the black open circles represent an intervention in the radiation parameter, the green pluses represent an intervention in the transmission parameter and the grey triangles represent an intervention in the reception parameter.

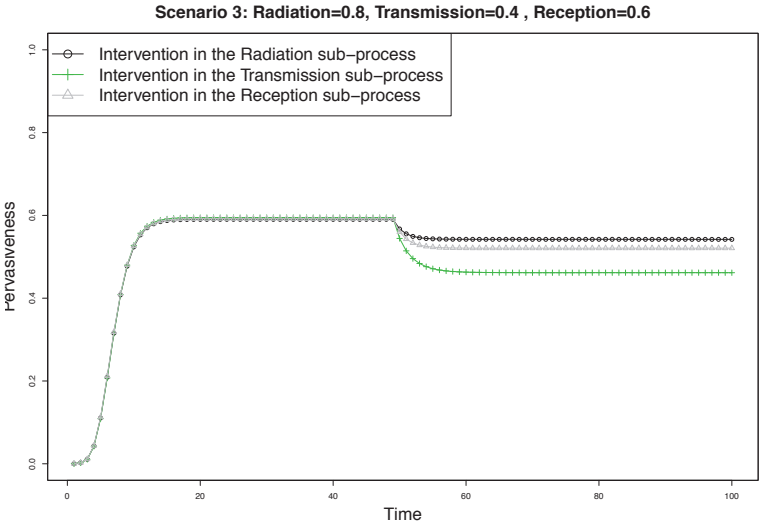


Figure 4.7: The pervasiveness over time is depicted for scenario 3 which is characterized by a propagation process with $\alpha = 0.8$, $\phi = 0.4$ and $\eta = 0.6$. Different interventions (-0.2) are applied in this scenario; the black open circles represent an intervention in the radiation parameter, the green pluses represent an intervention in the transmission parameter and the grey triangles represent an intervention in the reception parameter.

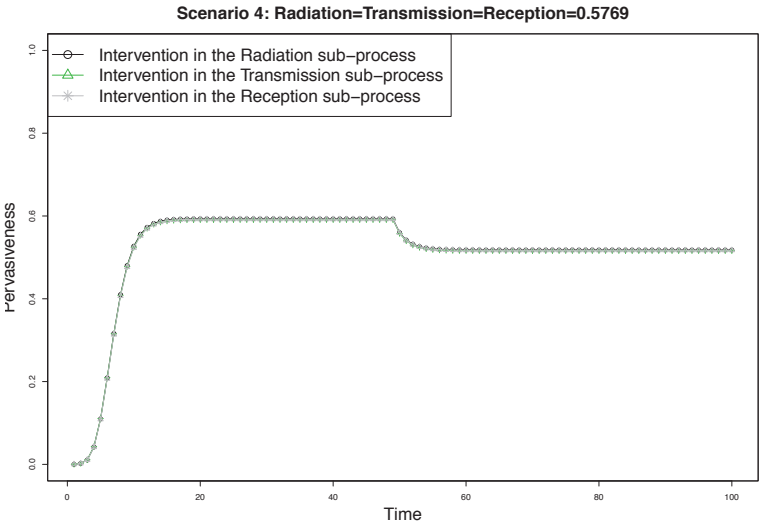


Figure 4.8: The pervasiveness over time is depicted for scenario 4 which is characterized by a propagation process in which sub-processes are homogeneous ($\alpha = \phi = \eta = 0.5769$). Different interventions (-0.2) are applied in this scenario; the black open circles represent an intervention in the radiation parameter, the green pluses represent an intervention in the transmission parameter and the grey triangles represent an intervention in the reception parameter.

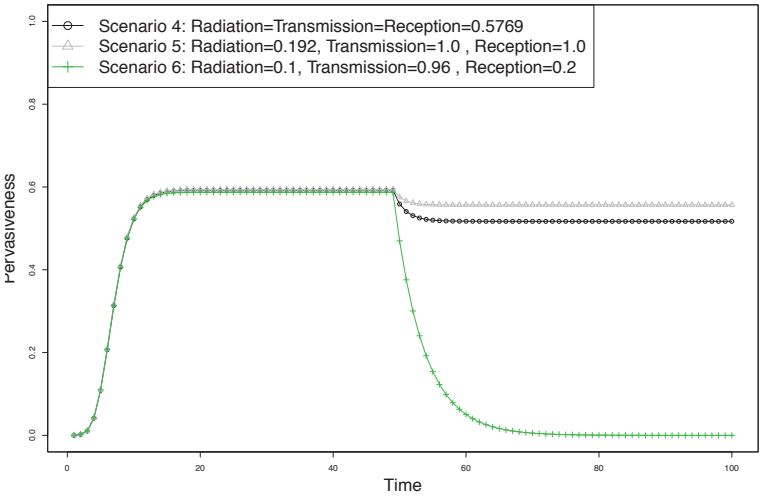


Figure 4.9: The pervasiveness over time is depicted under an intervention in the reception (η). Three scenarios are depicted: scenario 4 (black open circles) which has homogeneous sub-processes ($\alpha = \phi = \eta = 0.5769$), scenario 5 (grey triangles) in which the intervened parameter was initially maximized(pre-intervention: $\eta = 1.0$), and scenario 6 (green pluses) in which the intervened parameter was initially equal to the intervention size (pre-intervention: $\eta = 0.2$)

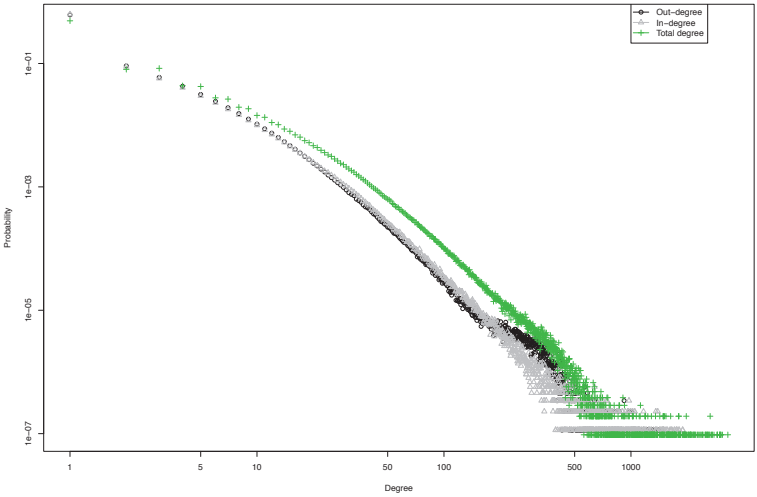


Figure 4.10: Log-Log plots of degree versus probability for week 1. The open black circles represent the out-degree; the open grey triangles represent the in-degree and the green pluses represent the total degree.

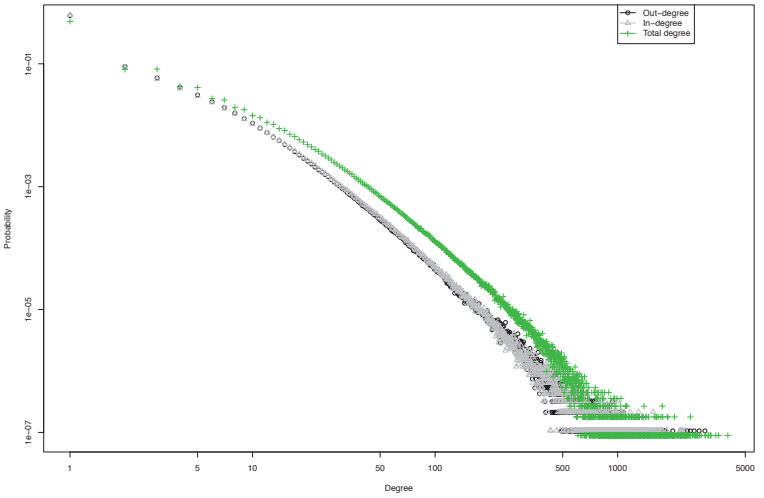


Figure 4.11: Log-Log plots of degree versus probability for week 2. The open black circles represent the out-degree; the open grey triangles represent the in-degree and the green pluses represent the total degree.

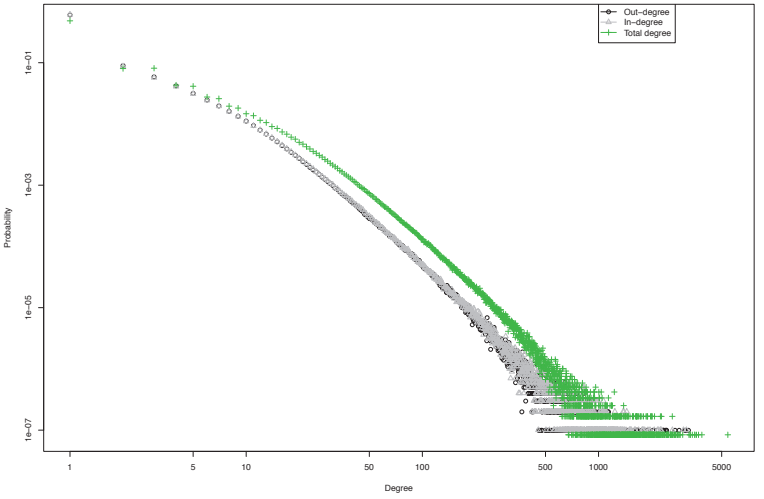


Figure 4.12: Log-Log plots of degree versus probability for week 3. The open black circles represent the out-degree; the open grey triangles represent the in-degree and the green pluses represent the total degree.

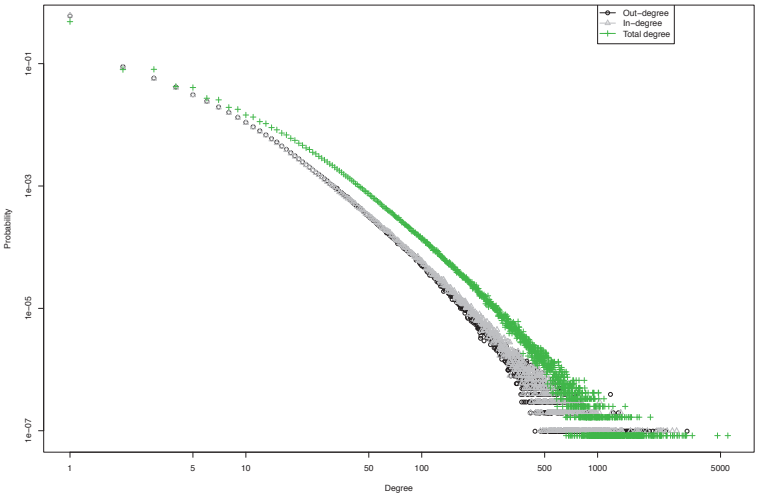


Figure 4.13: Log-Log plots of degree versus probability for week 4. The open black circles represent the out-degree; the open grey triangles represent the in-degree and the green pluses represent the total degree.

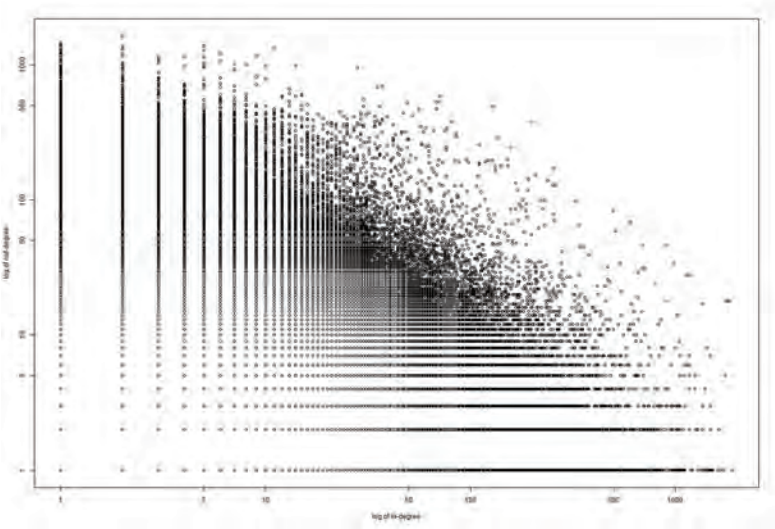


Figure 4.14: Log-Log plot of the In-Out degree correlations for week 1.

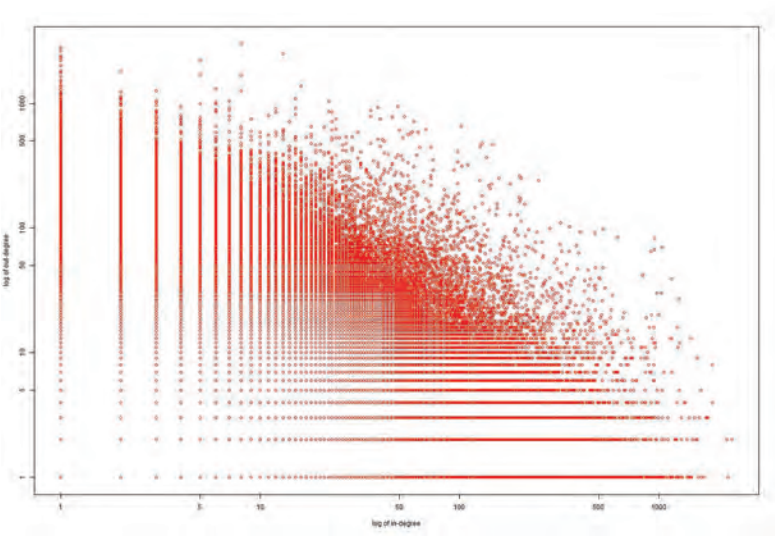


Figure 4.15: Log-Log plot of the In-Out degree correlations for week 2.

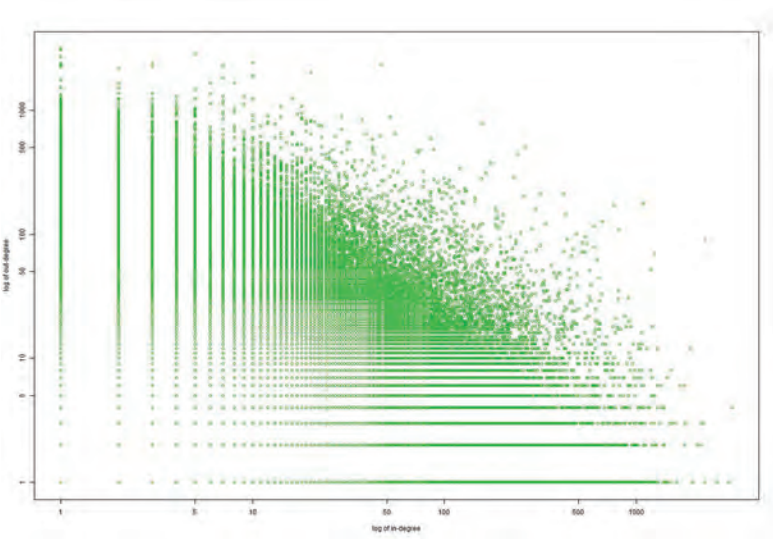


Figure 4.16: Log-Log plot of the In-Out degree correlations for week 3.

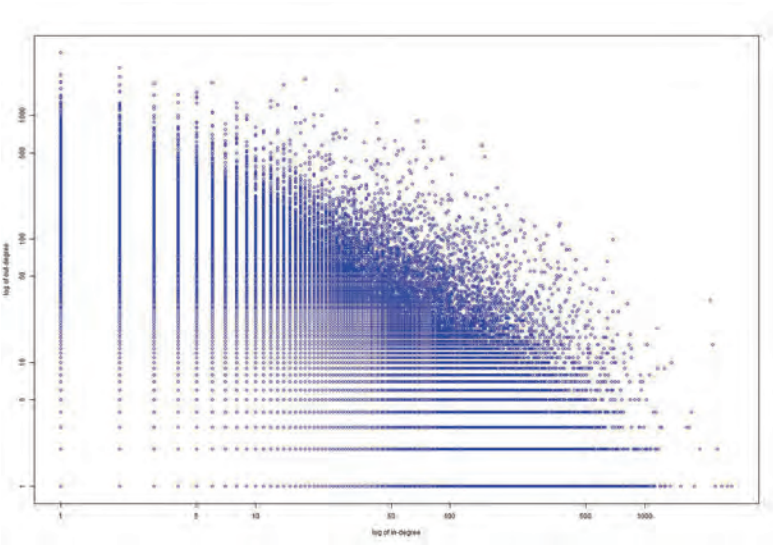


Figure 4.17: Log-Log plot of the In-Out degree correlations for week 4.

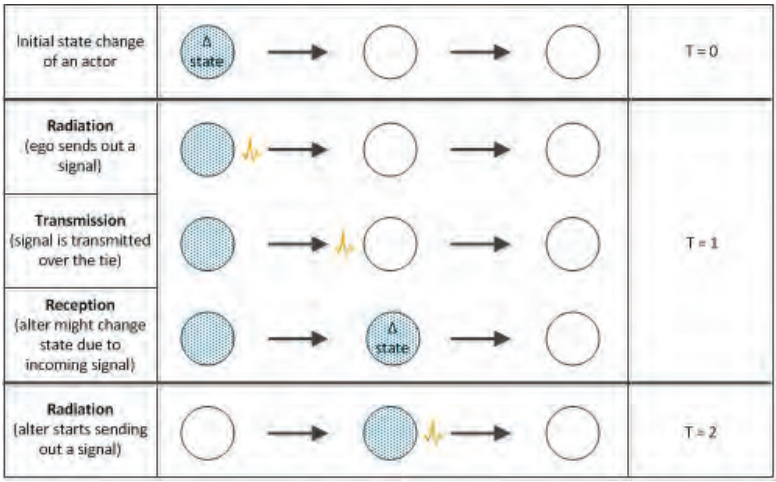


Figure 4.18: The steps in the propagation process.

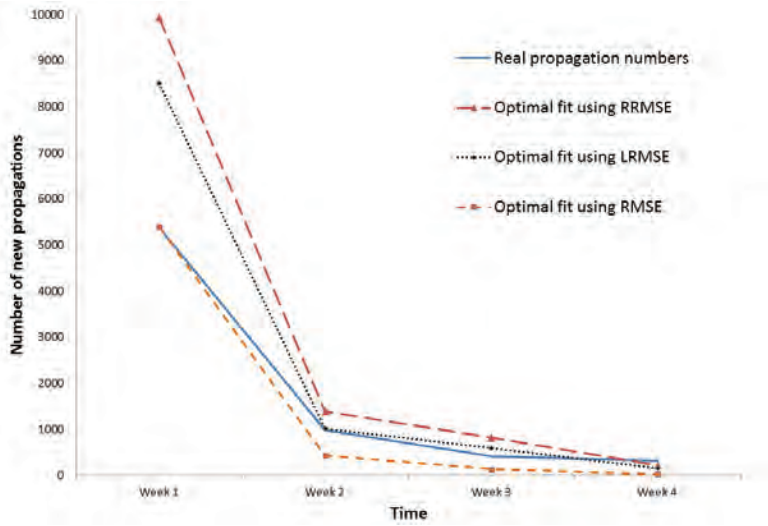


Figure 4.19: This figure shows the number of new adopters over time due to propagation for the actual data (solid blue line), the best fitting decomposition using the Root-Mean-Squared- Error (RMSE) fit measure (short-dashed green line), the best fitting decomposition using Logged-Root-Means-Squared-Error (LRMSE) measure (dotted black line), and the best fitting decomposition using the Relative-Root-Mean-Squared-Error (RRMSE) measure (long dashed red line)

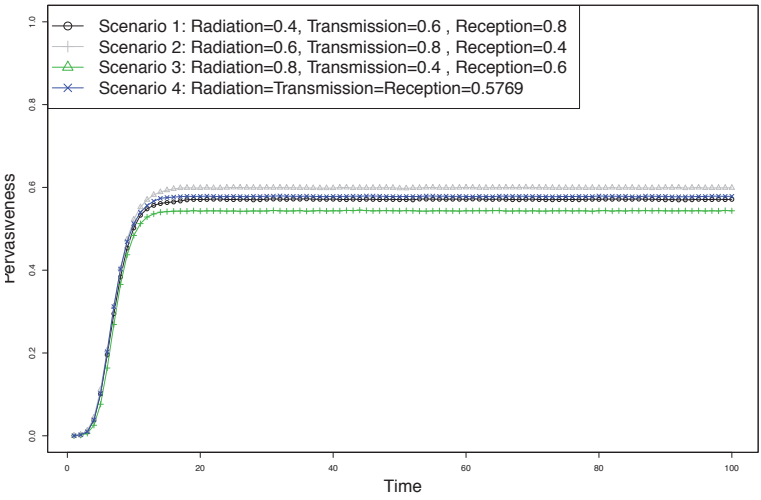


Figure 4.20: The pervasiveness over time is depicted for scenarios with the same propagation likelihood ($\lambda = 0.192$) but different divisions into radiation (α), transmission (ϕ) and reception (η). Scenario 1 (black open circles) represent a process with $\alpha = 0.4, \phi = 0.6, \eta = 0.8$. Scenario 2 (grey triangles) represent a process with $\alpha = 0.6, \phi = 0.8, \eta = 0.4$. Scenario 3 (green pluses) represent a process with $\alpha = 0.8, \phi = 0.4, \eta = 0.6$. Scenario 4 (blue crosses) represent a process with $\alpha = \phi = \eta = 0.5769$. Results are generated using a bootstrapping sample (100 times) with a randomly selected seed node.

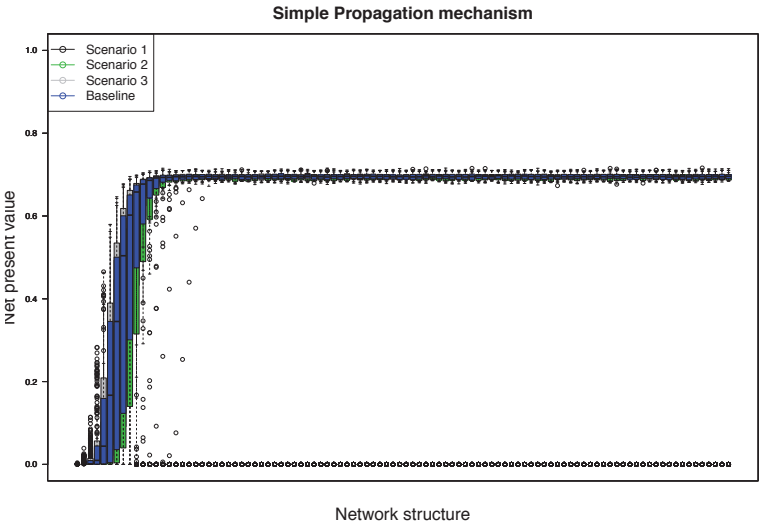


Figure 4.21: The boxplot of the pervasiveness over time is depicted for the same propagation scenarios, Scenario 1 (black) represent a process with $\alpha = 0.4, \phi = 0.6, \eta = 0.8$. Scenario 2 (grey) represent a process with $\alpha = 0.6, \phi = 0.8, \eta = 0.4$. Scenario 3 (green) represent a process with $\alpha = 0.8, \phi = 0.4, \eta = 0.6$. Scenario 4 (blue) represent a process with $\alpha = \phi = \eta = 0.5769$. Results are generated using a bootstrapping sample (100 times) with a randomly selected seed node.

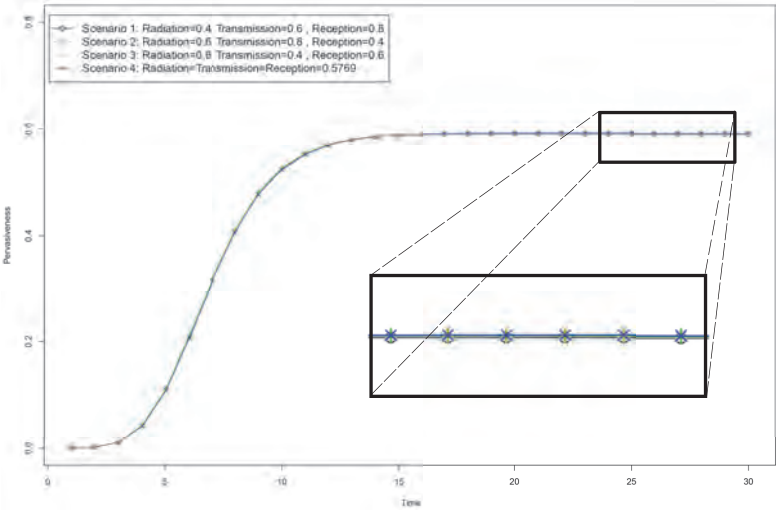


Figure 4.22: The pervasiveness over time is depicted for scenarios with the same propagation likelihood ($\lambda = 0.192$) but different divisions into radiation (α), transmission (ϕ) and reception (η). Scenario 1 (black open circles) represent a process with $\alpha = 0.4, \phi = 0.6, \eta = 0.8$. Scenario 2 (grey triangles) represent a process with $\alpha = 0.6, \phi = 0.8, \eta = 0.4$. Scenario 3 (green pluses) represent a process with $\alpha = 0.8, \phi = 0.4, \eta = 0.6$. Scenario 4 (blue crosses) represent a process with $\alpha = \phi = \eta = 0.5769$. Results are generated using bootstrapping (10,000 times) on the full population of seed nodes.

Chapter 5

Study 2: The effects of actor heterogeneity

Coauthors[1]: -

Abstract:
In this paper we explore the effects of local heterogeneity in the mechanism of propagation on propagation dynamics. We extend current research on heterogeneity by controlling for the effects of network structure while incorporating heterogeneity in actor behavior. By applying the notion that propagation exists of three sub-processes (Radiation, transmission and reception) we link actor behavior to radiation and reception, and allow for heterogeneity in local mechanism of propagation by altering these parts of the propagation process. We find that on average heterogeneity in radiation and reception have similar effects, both reduce the propagation outcomes, however, the manner in which they achieve such effect differs radically. Whereas heterogeneity in radiation reduces the chance of successful propagation, heterogeneity in reception reduces the size of a successful propagation. Furthermore, we show that interaction between heterogeneity in radiation and reception has a strong impact on both the speed of propagation and extent to which processes propagate.

[1]This chapter considers a working paper. A preliminary version of this paper has been presented at the NWO complexity day 2014.

5.1 Introduction

Growth in the amount and strength of interactions within and between systems, has lead to a world in which the spreading phenomena have in increasing impact on our society. Global trends such as the 2008 financial crisis, the 2014 Ebola outbreak and the 2014 ice-bucket challenge are only a tip of the vast amount of global propagation processes shaping our world. As a consequence the literature on propagation, the process by which a change in behavior or state of one actor results in the change in one or more of its alters, is steadily increasing. And while there is prevalent consensus that heterogeneity causes variations found in propagation outcomes, there is a divide in what is assumed to be the source of such heterogeneity.

Heterogeneity within a propagation process can effectively yield propagation mechanisms which are varying in space or time (Strang and Tuma, 1993). The variance in the mechanism will consequently cause variance in the propagation dynamics. Clearly, considering mechanisms that vary in time implies just that; mechanism can be different depending on at which moment in time they are considered, suggesting there is some form of path dependance in the mechanism and dynamics of propagation. Considering mechanism that vary in space, implies that at a single point in time the mechanism and consequent dynamics might be different depending on which location of the system is considered, suggesting that there is no single dynamic which applies to the system as a whole. In this study we will focus on the latter, variations in space, as a source of heterogeneity in the propagation dynamics.

5.1.1 Two views on local heterogeneity in propagation

Within this domain that considers local heterogeneity in the propagation dynamics two commonly studied drivers of heterogeneity are identified; network structure and actor behavior. The first driver mentions the structure of interactions as a driver of local heterogeneity; it argues that the network structure will vary locally. As the propagation process is constrained by this structure its dynamics will consequentially vary per location in the network. The second describes actor behavior as a source of local heterogeneity; it argues that actors are different and as the mechanism of propagation is based on actor behavior this will impact the local mechanism of propagation and consequently its dynamics. While each of them separately only covers part of heterogeneity in propagation, in fact both are needed to fully understand propagation dynamics on the system level. While we are interested primarily in role of actor behavior. we in this study will capture

both aspects in a single model, but control for the impact of heterogeneous network structure.

5.1.1.1 The network structure as a source of heterogeneity

The structural view of heterogeneity focuses on the structure of interactions, the network structure, as a source of heterogeneity in the propagation process dynamics. It builds on the notion that the network provides the infrastructure over which processes can propagate, and hence differences in this structure will impact the dynamics of propagation. In support of this notion research focusing on the impact of variations in the structure of the system as a whole (and the consequent network topology) has show the network structure to be critical in determining the robustness of a network to propagation (Albert et al., 2000; Dodds and Watts, 2004). While this suggest the to hold at the system level, the same can be argued to be true at the local level or anything in between. Indeed, support has been found for effects of structural holes (Burt, 1987, 2005) and clustering (Rahmandad and Sterman, 2008), suggesting that the structure impacts propagation dynamics at all levels.

On the local level the claim is that variations in the local structure can cause the same propagation mechanism to result in locally different propagation outcomes, and consequently affect the global propagation process. It should be clear that the mechanism of the process itself does not change in such scenarios. In fact in this type of study it is assumed that all actors behave similar, and hence (apart from their network structure) are homogeneous. As assuming such homogeneity implies that the effect of the actor characteristics on propagation dynamics are the same everywhere in the system, the propagation dynamics are assumed to be independent of the actors characteristics. While this assumption does simplify the modeling of propagation significantly, we know that in practice actors are very seldom homogenous (Jackson and Lopez-Pintado, 2013; Young, 2009) and that their characteristic do play a role in the propagation process (e.g. (Myers, 2000; Stein, 2011)).

5.1.1.2 Actor behavior as a source of heterogeneity

The behavioral view on heterogeneity focuses on the notion that no two actors are the same, and actors thus are inherently heterogeneous. It is claimed that the actor characteristics influence an actor's behavior, and hence influences their role during propagation (e.g. (Myers, 2000; Stein, 2011)), suggesting that actor characteristics and consequent behavior, create heterogeneity in local propagation dynamics. When zooming in on propagation,

and considering the mechanism of propagation, the argument can be better explained. On the dyadic level the propagation process can be considered as interaction much like described in traditional communication theory (Shannon, 1948), being build up from three steps; radiation transmission and reception. An actor sends out a signal (Radiation), it is transferred to an alter (Transmission) and the alter does (not) respond to the signal (Reception). What becomes evident from this description of the mechanism of propagation is; both the radiation and the reception sub-process are driven by actor's activity. Therefore, these sub-processes are likely linked to the characteristics of that actor. Indeed, previous work has indicated the influence of actor characteristics on an individual's propensity to; adopt an innovation (Aral et al., 2013), to be susceptible for respectively influenza (Boon et al., 2011), HIV (Hardie et al., 2008) and Hantaan (Nakamura et al., 1985), and to spread SARS (Stein, 2011), supporting the notion that actor characteristics indeed can change the local mechanism of propagation. Consequently, these local mechanisms will yield local dynamics, which in turn affect the dynamics of propagation on the system-level. Consequently, the notion of incorporating actor heterogeneity into the modeling of propagation has received some attention in literature (Goldenberg et al., 2001; Rahmandad and Sterman, 2008; Young, 2009; Jackson and Lopez-Pintado, 2013). However, these studies have adopted a simplified notion of the mechanism of propagation, not distinguishing between the sub-processes of propagation.

We claim that both sources are not mutually exclusive and in fact cover two distinct effects of local heterogeneity. Therefore, both should be studied simultaneously in order to get an understanding of propagation and its outcomes. Existing literature however usually addresses either one of them, and not both. Exceptions can be found in the body of work covering influence versus homophily (Aral and Walker, 2014; Fang et al., 2013; Jackson and Lopez-Pintado, 2013; Ma et al., 2014). This body of work addresses in particular the tension between the two, and builds on the notion that network positions of actors and actor characteristics are often correlated. By doing so this body of work raises awareness of the fact that both effects often co-occur and hence the cause of propagation are hard to determine. This work is certainly a step towards integration of the two sources (even though the conclusion is that they are hard to distinguish), but even in these studies the propagation mechanism is oversimplified and often a single sub-process is considered rather than the whole propagation process.
Knowing that each of the sub-processes plays a crucial role in propagation, and that the dynamics of propagation can strongly depend on the decomposition of propagation into sub-processes (see chapter 4) suggests that studying influence of local dynamics requires

a more nuanced view of propagation. The information processing view of propagation and the consequent RTR-model up forward in this dissertation build on the notion that propagation is not a single process but consists of three distinct sub-processes: Radiation, transmission and Reception. We adopt this view on propagation incorporating the mechanism of propagation in this study of the effects of local heterogeneity on propagation dynamics.

5.2 Modeling propagation

Consider a propagation process, for example a behavior (or disease) spreading in a population of interacting actors. The question we aim to address is: Will introducing heterogeneity of local propagation dynamics significantly change the outcomes of this propagation process? In order to study this question we simulate a stochastic propagation process on a finite stable network structure.

5.2.1 States in the system

Actors ($iinN$) in the system can have one of two states; 0 or 1 ($s_i = (0, 1)$). In which the 0 state refers to being susceptible (to infection) and 1 state refers to being infected. We assume that actor characteristics do no influence the state of an actor directly, and that actors in isolation are homogenous in their likelihood of being in either of the two state. Actors can change state once in each time unit, and will do so based on the number of signals they receive from infected actors in their environment. The more signals an actor receives the more likely it will be for this actor to be in state 1. We assume that there is a default recovery process which steers infected actors to the susceptible state (effectively making this a propagation model which resembles an SIS model), hence reducing the amount of signals an actor receives will increase the likelihood of this actor to be in the 0 state.

5.2.2 The network of interactions

We are interested in the effect of heterogeneity in local dynamics on propagation outcomes. In line with previous work (e.g. (Myers, 2000; Hardie et al., 2008; Boon et al., 2011)) we assume that a set of latent actor characteristics (for example: age, race, gender, extroversion or affinity towards technology) that influence the mechanism of propagation. As

these actor characteristics are heterogeneously spread among the population, such heterogeneity will cause heterogeneity in individual parameters which describe the mechanism of propagation. In order to circumvent the homophily/influence discussion we assume that the location of actors is independent of actor characteristics (see section 2.1) and hence only local propagation mechanisms are affected by actor characteristics. Furthermore, we need to correct for potential local heterogeneity in the network structure. Rather than assuming no structure (homogeneous mixing) as often done in previous work Young (2009); Jackson and Lopez-Pintado (2013), we control for the influence of the structure by making sure there is no heterogeneity in the network structure, by assuming the network structure to be regular. In a regular network all actors have the same degree and ego-network structure, therefore the network does have an effect on propagation dynamics, but this effect is similar everywhere in the system. Consequently any heterogeneity in local dynamics can be attributed to the heterogeneous actor behavior rather than the network in which it is embedded.

5.2.3 The propagation process

We assume a propagation process with a propagation rate which is above the epidemic threshold, and hence based on the mean-field estimations we expect a widespread propagation will occur for such a process (Pastor-Satorras and Vespignani, 2001a). Following chapter 4, we consider a (stochastic) propagation process which is decomposed into three distinct sub-processes; Radiation, Transmission and Reception. Each of these sub-processes has an arbitrary chance of success. During each time unit all actors in state 1 (infected) will attempt to radiate a signal towards all their alters over their outgoing ties, for every outgoing tie this attempt will be successful with the chance α, the radiation likelihood. Within the same time unit the successfully radiated signals will be transmitted with likelihood ϕ, the transmission likelihood. Last, also still within the same time unit any signal which is successfully transmitted has a chance of η to result in a change of state of the alter, the reception likelihood. On a dyadic level we hence state that $\lambda = \alpha\phi\eta$. After radiation, and before reception, all actors in state 1 (infected) change towards state 0 (susceptible) with a chance ρ, the recovery likelihood.

5.2.4 Propagation outcomes

Propagation process outcomes can be measured in many different ways, there is no one single correct way of defining propagation outcomes. Depending on the question being addressed the variable of interest can change; one could for example consider the speed

of propagation (Centola et al., 2007), the chance of widespread propagation (Jackson and Lopez-Pintado, 2013) or the proportion of infected actors (Albert et al., 2000; Pastor-Satorras and Vespignani, 2001a, 2002). In scenarios with propagation rates above the epidemic threshold, which we have assumed in section 5.2.3, it seems most fitting to consider the proportion of the population which becomes 'infected', and the speed by which this is reached. The tradition way to capture these measures is to consider the adoption curves during propagation. These curves depict on the y-axis the number of infections (as a percentage of the complete population) as the propagation process unfolds, and hence tracks them over time which is depicted on the x-axis. These adoption curves are used as the primary outcome of propagation in this study.

5.2.4.1 Prevalence

The number of actor which at any point in time is in the infected state captures the how prevalent such an infection is on the system level, it is therefore often referred to as prevalence or pervasiveness. The prevalence in the the network at any time $t \geq 0$ is hence defined as: *the proportion of actors in the system which is at state 1 at any point in time*. Previous work (Pastor-Satorras and Vespignani, 2002; Jackson and Lopez-Pintado, 2013) has using mean-field approaches shown that, under the assumption of homogenous mixing, the number of actors that gets infected at any point in time (propagation) can be described as the chance that an actor is in the infected state, multiplied by the number of signals it is able to send out, multiplied by the chance that the alter is in fact in the susceptible state. If one subtracts from this the number of actors which recover during that same time unit one can calculate the proportion of infected actors in the system. Hence the prevalence can be described as:

$$\frac{\Delta I}{\Delta t} = -\rho I + I \langle k \rangle \lambda (1 - I) \tag{5.1}$$

In which $\langle k \rangle$ is the average number of edges per vertex, I is the proportion of actors in state 1 (infected). This function effectively takes a radiation perspective, assuming that any signal being send out (radiated) will be transmitted and received by the alter(s), and considers propagation to be the sum of interactions between two groups (compartments) of actors. Imposing a network structure (rather than homogeneous mixing) requires reevaluation of this formulation. For starters we have to acknowledge that their are multiple ties which can facilitate propagation of signals. This implies that while the state of an actor can only change once, this actor might receive multiple signals from its ego network in

the same time unit. Consequently the chance of changing state (reception) is conditional not on the dyadic states alone but on the state of all neighbors in the ego-network. Introducing the network structure hence forces adoption of a nuanced view of propagation incorporating three sub-processes. It becomes evident that we cannot simply replace the propagation likelihood (λ) by the function of the three sub-processes ($\alpha\phi\eta$). Instead we build the propagation process as the function of the three sub-processes:

Sub-process	Logic	Partial function
Radiation	The rate at which an actor in the infected state will successfully radiate a signal	$I\alpha \langle k \rangle$
Transmission	The chance that the radiated signal is successfully transmitted	ϕ
Reception	The chance that any the incoming signals leads to reception	$(1-I) \sum_{k=1,\ldots,\langle k \rangle} \binom{n}{k} p^k (1-p)^{n-k}$

Combining them the propagation process can be described as:

$$\frac{\Delta I}{\Delta t} = -\rho I + I\alpha \langle k \rangle \phi \sum_{l}^{1} \binom{n}{l} p^l (1-p)^{n-l} (1-I) \tag{5.2}$$

In which $l = (1, \ldots, \langle k \rangle)$, n is the number of trials which is equal to $\langle k \rangle$ and p is the likelihood that a given tie will yield reception. The chance of reception per tie is equal to the chance that actor is infected, this actor generates a signal, this signal is transported, and received by the alter ($p = I\alpha\phi\eta$).

When $\frac{\Delta I}{\Delta t} = 0$ the system is in equilibrium and the formula in a (dynamic) stable state (I_e), clearly $I = 0$ yields a stable state in which none of the actors are infected. The other none-zero solution could simply be obtained by solving equation 5.2. However, as I is part of p (the binomial chance of successful propagation over a tie, $p = I\alpha\phi\eta$), one can get an exact analytical solution only for scenarios with small $\langle k \rangle$. For higher number of $\langle k \rangle$ the binomial part of the equation becomes infeasible to solve analytically. As there is no elegant analytical solution for equation 5.2 we will adopt a Agent-based simulation approach to determine the non-zero equilibrium state. Such an approach is not bound by $\langle k \rangle$ to determine I_e and hence provides us with less constraints to do our analysis.

The big difference between the mean-field and agent-based approach is that the in the latter the behavior of the model is defined on the micro level. In agent based models only the behavior rules of actors (agents) are defined, without making assumptions of the system-wide dynamics. Therefore system-wide behavior is claimed to 'emerge' from the

micro-level interactions rather than being predefined in the model. Additional benefit of this type of method is that as behavior is defined on the actor level (rather than the system level) it allows for easily incorporating heterogeneity in actor behavior.

5.2.5 Heterogeneity of actor behavior

We include the notion of actor heterogeneity into our model by assuming that each actor has a (latent) vector of characteristics c which determines its propensity to send out (radiate) and respond to (receive) propagation signals; c can vary for each actor and over time and thus $c_{i,t}$ describes the characteristics of actor i on time t. In order to focus on heterogeneity in space rather than time we will assume that the characteristics are stable over time ($c_{i,t} = c_{i,t+1}$). As we assume that actor characteristics influence the local propagation mechanism, including the actor heterogeneity requires replacing the global radiation and reception parameters (α, η) with local parameters per actor $\alpha_{i,e}$ and $\eta_{e,i}$ respectively. For simplicity we will assume that actors do have a preference for ties during radiation and reception ($(\alpha_{i,1} = \alpha_{i,2}... = \alpha_{i,e} = \alpha_i)$ and $(\eta_{i,1} = \eta_{i,2}... = \eta_{i,e} = \eta_i)$), resulting in a vector A ($A = (\alpha_1, ..., \alpha_i)$) capturing radiation probabilities and a vector Ψ ($\Psi = (\eta_1, ..., \eta_i)$) capturing reception probabilities. When comparing different propagation processes we pose that the average radiation (and reception) remains equal in all (x) settings ($\alpha = \overline{A^1} = \overline{A^x}$ and $\eta = \overline{\Psi^1} = \overline{\Psi^x}$). Having a model with local propagation parameters, we now can introduce heterogeneity into the system by assuming heterogeneity in the characteristics (c_i) of part population, as this would yield only a change in an individual's radiation and/or reception probabilities we can also simply change these parameters directly. Consequently, we start from the simplest scenario of heterogeneity in which we create two groups of actors, each with different radiation (reception (or both)) and compare the propagation outcomes to those of the unperturbed (default) scenario.

5.3 Simulation

We explore the influence of actor heterogeneity and the consequent heterogeneity in propagation mechanism by means of Agent-based simulation. As input for this simulation we generate a fixed regular network structure with N actors, on which we will let a propagation process run from a single seed. We let this process run until it reaches the approximate dynamic equilibrium, at which point we can determine the prevalence of the process and the time it took to reach this (semi) stable state. As results of a single

simulation run would be very susceptible for variations due to stochasticity we increase the robustness of our results by bootstrapping each simulation N times. The results are averaged over this bootstrapped sample, any simulation which converges into the stable state in which the propagation process dies out ($I_e = 0$) will be ignored. As using a simulation approach requires fixing some of the parameters at arbitrary values, the next section will address the parametrization of the simulation in more detail.

5.3.1 Parametrization of the simulation setting

First the simulation requires a network structure on which the process can propagate. We generate a regular three-dimensional lattice network, in which each actor is connected to its two nearest neighbors on each dimension. We assume the network is wrapped and hence that each dimension is circular, consequently each actor has a degree of 6, and the lattice has no boundaries and is fully regular. Each dimension has 10 actors resulting in a total population of 1.000 actors ($N = 1.000$).

Second we fix arbitrary values for the propagation process. The default scenario assumes all three sub-processes play an equally strong role in propagation ($\alpha = \phi = \eta = 0.5$), resulting in an overall propagation likelihood (λ) equal to 0.125 ($0.5 \times 0.5 \times 0.5$). The recovery likelihood is fixed at an arbitrary value ($\rho = 0.2$) such that the epidemic threshold is surpassed and widespread propagation is to be expected.

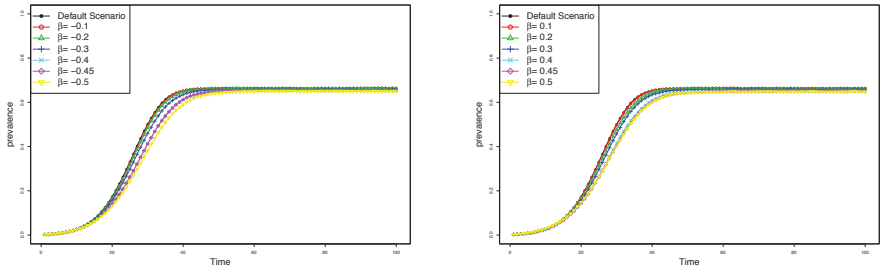
Third an arbitrary amount of heterogeneity has to be introduced into the actor behavior. We select a group of arbitrary size g, and hence divide the population into two groups of size g and $1 - g$ respectively. In our default scenario $g = (\frac{1}{3}N)$ and hence group 1 holds $\frac{1}{3}$ ($g_1 = \frac{1}{3}N$) and group 2 holds $\frac{2}{3}$ of the population ($g_2 = \frac{2}{3}N$). Actors are randomly assigned to either one of these groups. For the first group we adjust the radiation and/or reception likelihood with an arbitrary amount β. We will refer to this adjustment to group 1 as a 'perturbation', which will effectively will mean a *reduction* of the given likelihood by β. This effectively means that positive values of β decrease the likelihood of radiation (and/or reception) in group 1. As we want to keep the average radiation (and reception) equal in the population any perturbation in group 1 need to be compensated by changing the radiation (and/or reception) of the second group as well. We spread this second change equally among the members of the second group and therefore their radiation (or reception) will change with a factor $\beta\frac{g_2}{g_1}$ (which is 0.5β in the default scenario). Consequently the perturbation creates two groups in which actors have different local propagation dynamics (based on latent actor characteristics), representing the simplest case of heterogeneity of actors.

5.3.2 Simulation results heterogeneity

The analysis of the effects of heterogeneity actor behavior will consist of three sets of simulations; each focusing on a different type of perturbation. The first set will consider varying perturbation sizes (β) to the radiation sub-process, the second set of simulations will apply the same perturbations to the reception sub-process, and the third set will apply perturbations to both the radiation and reception at the same time.

5.3.2.1 Heterogeneity in radiation

The results of the first set of simulations can be found in figure 5.1, and show the effects of perturbing the radiation with different strengths (β). These results reveal that heterogeneity in the radiation process (regardless of its size) yields no significantly different prevalence for the propagation process. Based on these findings we could state that the prevalence seems unaffected by heterogeneity in the radiation process. Remarkably these results also do indicate that for the extreme perturbations in heterogeneity ($\beta =$ respectively $-0.5, -0.45, 0.5$ or 0.45) the amount time it takes to reach equilibrium (I_e) is larger, and hence the propagation process seems to be slower.



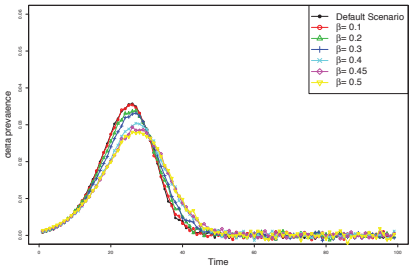
(a) The prevalence under *negative* reductions, increasing the radiation the targeted group(1) by β

(b) The prevalence under *positive* reductions, lowering the radiation of the targeted group(1) by β

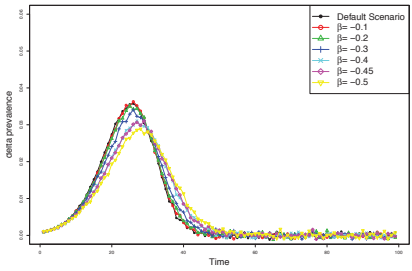
Figure 5.1: The prevalence under different perturbations of the **radiation** sub-process

Especially when looking at figure 5.2, which depicts the rate at which prevalence changes over time, one can see that the peak of the speed of propagation is reduced when heterogeneity in the radiation is introduced. Suggesting that the maximum rate at which the population becomes infected is lower when there is heterogeneity in the radiation of actors. Furthermore one can note that the reduction in height of the peak is offset by extending the width of the peak, suggesting that scenarios with more heterogeneity in

radiation indeed take longer to reach the equilibrium state and hence have a lower overall speed but do reach a similar level of prevalence.



(a) The prevalence under *negative* values of β, increasing the radiation of the target group



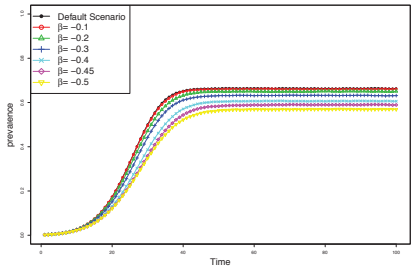
(b) The prevalence under *positive* values of β, reducing the radiation of the target group

Figure 5.2: The changes in prevalence over time ($\frac{\delta I}{\delta t}$) under perturbations of β in radiation ($\alpha_{g_1} = \alpha - \beta$)

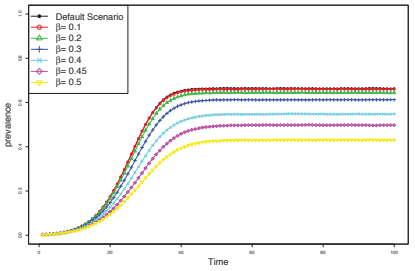
5.3.2.2 Heterogeneity in reception

The results of the second set of simulations can be found in figure 5.3, and show the effect of introducing heterogeneity into the reception process. These results reveal an effect which is very different from that of heterogeneity in radiation, namely that the prevalence becomes significantly smaller when heterogeneity in the reception sub-process is introduced. Even more so the larger the size of the heterogeneity the stronger this effect becomes. Considering the speed of propagation (figure 5.4 once more shows the rate at which the prevalence changes) reveals that the introduction of heterogeneity in the reception process also reduces the speed of propagation significantly, heterogeneity in reception reduces the height of the peak even more than heterogeneity in radiation. Also it shows that the width of the peak is bigger, consequently the time it takes for the propagation processes to reach the stable state (I_e) seems to be bigger when heterogeneity in reception is introduced. When comparing heterogeneity in radiation and reception we find that regardless of where the heterogeneity is introduced the width of the peak is affected equally. As a consequence of the lower height and same width the heterogeneity in reception results in a lower area under the curve and hence a lower overall prevalence in the (semi) steady state (I_e).

One should note that the analysis and consequent results (figures 5.1, 5.2, 5.3 and 5.4)have only considered scenarios with successful widespread propagation. By imposing the constraint $I_e \neq 0$ we have purposely neglected the chance of success (success-rate) of the

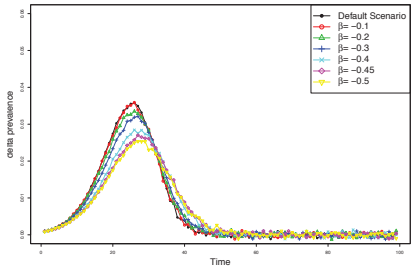


(a) The prevalence under *negative* reductions, increasing the reception the targeted group(1) by β

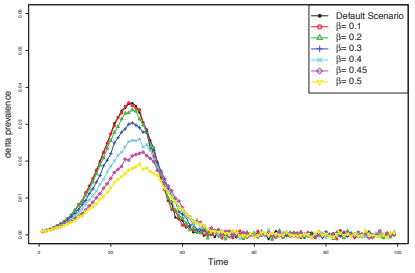


(b) The prevalence under *positive* reductions, lowering the the reception of the targeted group(1) by β

Figure 5.3: The prevalence under different perturbations of the **reception** sub-process



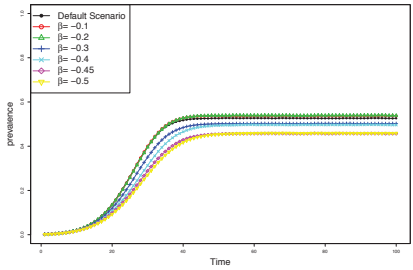
(a) The prevalence under *negative* values of β, increasing the reception of the target group



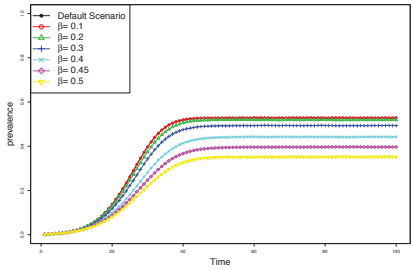
(b) The prevalence under *positive* values of β, reducing the reception of the target group

Figure 5.4: The changes in prevalence over time ($\frac{\delta I}{\delta t}$) under perturbations of β in reception ($\eta_{g_1} = \eta - \beta$)

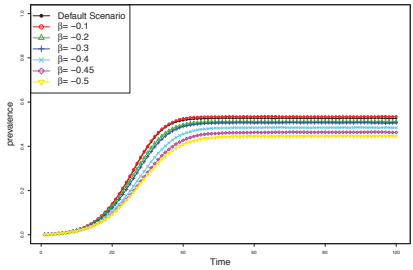
propagation process, as we have anticipated that the overarching propagation likelihood (λ) to be above the epidemic threshold. Removing this constraint allows us to measure also the effect of the success-rate of propagation process as an outcome variable.
At the mean-field this constraint in not present, and such setting argue that both types of heterogeneity would have the same effect on propagation as both have a similar effect on the overarching propagation likelihood ($\lambda = \alpha\phi\eta$). The results of including all simulations (also the unsuccessful ones) can be found in figure 5.5 and indicate that dropping the constraint of successful propagation indeed takes the results closer to the mean-field level of analysis, in the sense that there are no significant differences between heterogeneity on in either part of the propagation mechanism.



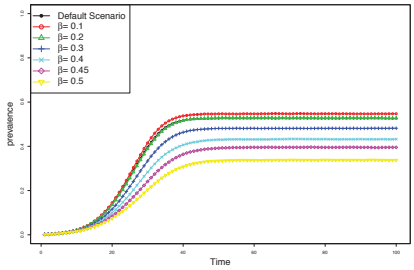
(a) The prevalence under *negative* reductions, increasing the **radiation** the targeted group(1) by β



(b) The prevalence under *positive* reductions, lowering the the **radiation** of the targeted group(1) by β



(c) The prevalence under *negative* reductions, increasing the **reception** the targeted group(1) by β



(d) The prevalence under *positive* reductions, lowering the the **reception** of the targeted group(1) by β

Figure 5.5: These figures describe the prevalence under different perturbations. These values are calculated using the average of ALL simulations both those who converge to a state of successful cascades ($I_e \neq 0$) and those which converge to a state of susceptibility ($I_e = 0$)

Clearly incorporating the success-rate into the results has critical implications on the conclusions one would draw. In fact, the different effects of heterogeneity found in successful propagation processes seem to be completely offset by incorporating the success-rate and averaging over both successful and unsuccessful scenarios. Considering the success-rate associated to both sources of heterogeneity (figure 5.6) reveals that introducing heterogeneity into the radiation sub-process significantly changes the success-rate, while introducing heterogeneity in the reception sub-process does not, a finding which can be better understood when the local dynamics in the early stages of propagation are taken into account.

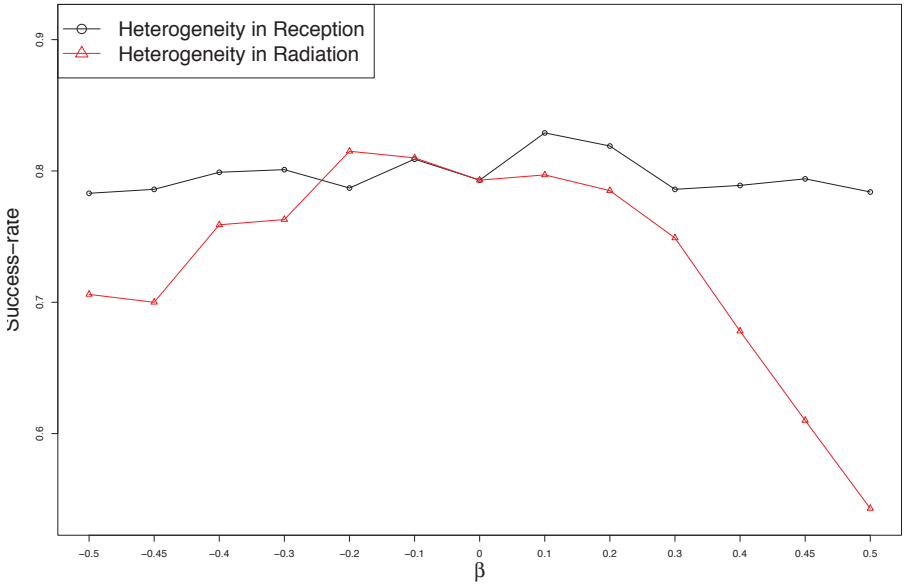


Figure 5.6: This figure describes the chance that the propagation process will converge to a state with widespread propagation ($I_e \neq 0$), the success-rate for processes with varying reductions (β) of the given sub-process

At the earlier stages there is a small seed of infection, for this seed to result in a cascade (a wide-spread propagation process) it will need to expand. By introducing heterogeneity in the radiation of the seed, we place a constraint on propagation potential of part of the population, and consequently of part of the potential seeds. When the initial seed is part of this group the constraint in radiation affect all $\langle k \rangle$ ties around the seed. In contrast

a constraint in reception would only apply to those ties which are directed towards the constrained alters, and hence will only affect the propagation likelihood of part of the ties. Consequently the constraint put into place by heterogeneity in radiation is clearly strongest in the early stages of the process where there is a low number of seeds. Even more so the stochastic nature of recovery in the model implies that in the early stages the propagation process runs the bigger risk of ending up in the stable state without infected actors, simply due to the limited set of seeds. Therefore in our model introducing heterogeneity increases the risk of going extinct in the early stages, and as the constraint for heterogeneity in radiation is stronger we see the biggest effect on the success-rate in those scenarios.

In contrast in the later stages of propagation,where the prevalence is measured, susceptible actors are prone to be subject to a multitude of signals. Here the constraint in radiation would only affect the likelihood of propagation over some of the incoming ties (only those ties coming from low radiation actors) whereas a constraint in reception would affect the likelihood of propagation over all $\langle k \rangle$ ties surrounding the to-be infected actor. Consequently the heterogeneity in reception hampers the overall prevalence more than a similar heterogeneity in radiation, a finding which is indeed supported by figures 5.1 and 5.3.

In conclusion, based on the first two sets of simulations we therefore argue that on average the result of introduction heterogeneity in either radiation or reception seem to be similar, both seem to reduce the prevalence of propagation, while the mechanisms by which they achieve this goal differ radically. Where heterogeneity in radiation reduces mainly the chances of widespread propagation (the success-rate), heterogeneity in reception reduces mainly the prevalence of successful propagation.

5.3.2.3 Combining heterogeneity in radiation and reception

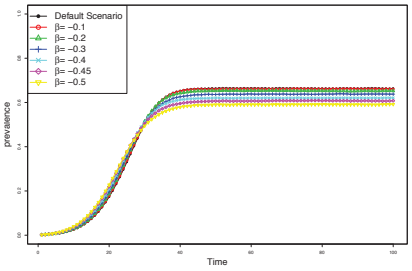
In the previous section we have studied the effects of heterogeneity in either radiation or reception. However, building on the notion that there is a set of latent actor characteristics that drive heterogeneity in the radiation and reception of actors, it seem more likely to assume that both effects occur simultaneously. Therefore, in the next (third) set of simulations, such scenarios are explored.

Starting from the simplest possible notion one could assume that both radiation as well as reception are affected equally by the latent actor characteristics. This effectively means that the group of actors which receives a perturbation in the radiation is also the group receives the same perturbation in the reception ($g_1^{rad} = g_1^{rec}$). As in this scenario the membership to the perturbed group (g_1^{rad} , g_1^{rec}) is linked this scenario is referred to as the

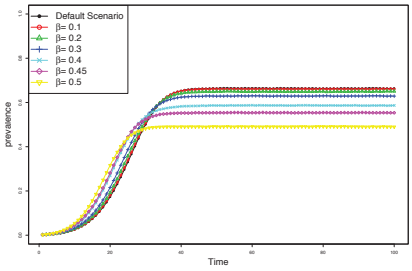
linked assignment. The other extreme of the spectrum would be a scenario in which the membership to the perturbed group for radiation (g_1^{rad}) is independent from the membership to the perturbed group for reception (g_1^{rec}). In this scenario group membership is assigned randomly, and hence this scenario is referred to as random assignment. While many other arbitrary ways could be proposed in which the latent actor characteristics could affect the local propagation mechanism, in this study we focus on two types of effects mentioned above; linked assignment and random assignment, as this is the simplest viable model which captures the interaction between heterogeneity in both parts of the sub-process.

5.3.2.4 Linked assignment

The linked assignment approach (like the previous simulations) yields two groups of actors. For the first group both radiation as well as reception are perturbed by reducing them by β, and for the second group radiation and reception are increased so that the average radiation and reception remain the same and hence the equality constraint is met. The results of the linked assignment scenario (Figure 5.7) show that introducing heterogeneity in radiation and reception at the same time both increases the speed and reduces the prevalence of the infection.



(a) The prevalence under *negative* values of perturbation ($\beta < 0$) in a setting of linked assignment to groups, effectively increasing both the radiation and reception of the targeted group (g_1) by β

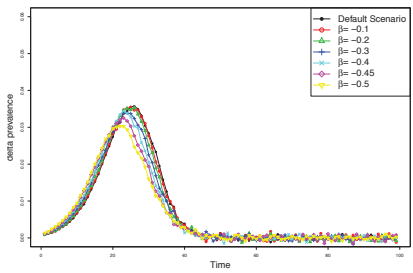


(b) The prevalence under *positive* values of perturbation ($\beta > 0$) in a setting of linked assignment to groups, effectively reducing both the radiation and reception of the targeted group (g_1) by β

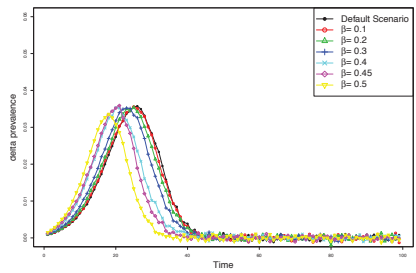
Figure 5.7: The prevalence under different perturbations in the linked assignment scenario

While the reduction in prevalence is in line with what one would expect from introducing heterogeneity, the increased speed seems to be opposite from the previously found

slowdown. Taking a closer look at the speed of propagation (figure 5.8) provides some interesting insights in the dynamics. Whereas negative perturbations (effectively increasing the radiation and reception of the target group) yield speed curves which —compared to the default scenario— seem to decline earlier, effectively having a peak which is lower, narrower, and pushed to the left (be it only slightly). suggesting earlier saturation of the carrying capacity of infections. Positive perturbations (which effectively reduce the radiation and reception of the target group) yield speed curves which —compared to the default scenario— have a peak which is strongly pushed to the left side, slightly narrower of similar in height, suggesting a more burst-like propagation behavior. The shift towards the left side indicates that apparently both perturbations in a linked assignment setting are able to offset the constraints on propagation (be it only for a short while). Even more so the positive perturbations seem to be more effective in doing so but at the same time also seem to be worse off in maintaining these benefits.



(a) The prevalence under *negative* values of β, increasing the reception of the target group



(b) The prevalence under *positive* values of β, reducing the reception of the target group

Figure 5.8: The changes in prevalence over time ($\frac{\delta I}{\delta t}$) under perturbations of β in a linked assignment setting

Considering the local propagation mechanism provides an insight as to why this is happening. In the linked assignment scenarios the actors which are prone to radiate signals are also the ones who are prone to act upon them, and hence to be infected. This combination makes those actors behave like hubs of propagation activity (even though they are not a hub in terms of network structure) and hence critical in driving the propagation process. In the early stages of propagation it is likely that, even though some actors are constrained by heterogeneity, these hub-like actors are driving the propagation process.

We consider two extreme scenarios to clarify these finding. We consider an scenario in which $\beta = 0.5$ *and* $\beta = -0.5$ *and provide a numeric example of what is happening during the early stages of propagation:*

- *Note that, in the default scenario each infected actor infects approximately 0.75 (*$k\alpha\phi\eta = 6 * 0.5 * 0.5 * 0.5 = 0.75$*) alters each time unit.*
- *In the first proposed scenario* $\frac{1}{3}$ *of the population has no radiation and reception (*$\alpha - \beta = 0$ *and* $\eta - \beta = 0$*). Consequently,* $\frac{2}{3}$ *of the population has their radiation and reception increased (to 0.75). In this scenario an infected actor will infect 1.125 (*$6 * 0.75 * 0.5 * 0.5$*) alters each time step if it is in group2 (and 0 if the seed is in group1, in which case the propagation process would die out, and will not be included in our graph and analysis).*
- *In the second proposed scenario* $\frac{1}{3}$ *of the actors always radiates and receives (*$\alpha - \beta = 1$ *and* $\eta - \beta = 1$*). Each infected actor in group1 will thus infect 1.5 alters each time unit (*$6 * 1 * 0.5 * 0.5$*), and each actor in group2 will infect 0.375 alters each time unit (*$6 * 0.25 * 0.5 * 0.5$*). To leverage the the potential speedup it will thus need to spread towards group1. So while this scenario has the potential to out-perform the previous one, it will not do so in each scenario, this clearly limits the potential of reaping the benefits of hub-like actors, and explains why on average it will lag behind the other scenarios.*

What this linked assignment scenario seems to implicate is that the propagation process can capitalize on a backbone of hub-like actors in the early stages of propagation to offset the constraints put into place by introducing heterogeneity. These hub-like actors serve as a 'high-way' of propagation. But as the size of this highway is limited, once this high-way becomes saturated the force offsetting the constraints will dissipate and the effects of heterogeneity start kicking back in.

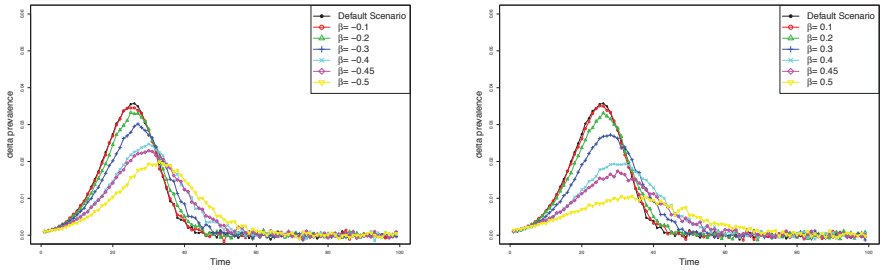
5.3.2.5 Random assignment

The notion that a set of latent actor characteristics drives both radiation and reception seems fairly straightforward, the fact that both are affected in the same manner might be less realistic. Therefore in the following scenario we will relax this assumption by stating that while the size of the perturbation in radiation and reception remains linked the actors which receive these perturbation do not need to be. Effectively this means that the actors which receive a perturbation in radiation are chosen by a process which is independent of the assignment of the perturbation in reception. As in this scenario there

is no link between the radiation and reception of actors the random assignment approach yields four types of actors:

- type1: $g_1^{rad} + g_1^{rec}$ holds $\approx \frac{1}{9}$ of the population
- type2: $g_2^{rad} + g_1^{rec}$ holds $\approx \frac{2}{9}$ of the population
- type3: $g_2^{rad} + g_1^{rec}$ holds $\approx \frac{2}{9}$ of the population
- type4: $g_2^{rad} + g_2^{rec}$ holds $\approx \frac{4}{9}$ of the population

Clearly this assignment mechanism reduces the potential size of the 'highway' by (at least) a factor 3, and hence this approach allows us to verify if the implied logic in the previous setting holds, in which case the found speed-up should be significantly reduced in the random assignment scenarios.
The results of the random assignment approach (Figure 5.9) show that indeed no speeding up of the propagation can be found in these scenarios, suggesting that the size of the 'highway' is insufficient to offset the reduction in speed caused by heterogeneity. In fact, the process has been slowed down to an extent that it is even slower than in the scenarios with only heterogeneity in one sub-process. This suggests that, in terms of speed, the constraints of heterogeneity in both sub-processes are interacting and are amplified when they are introduced simultaneously.



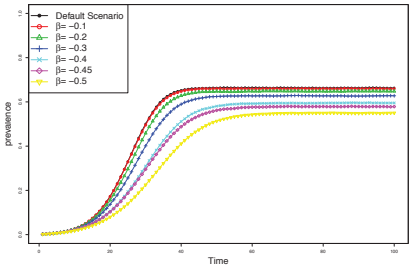
(a) The prevalence under *negative* values of β, increasing the reception of the target group

(b) The prevalence under *positive* values of β, reducing the reception of the target group

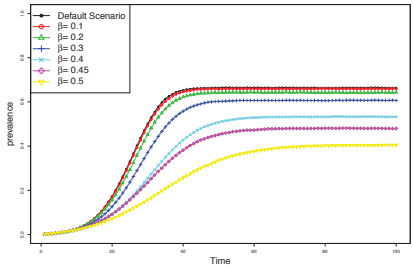
Figure 5.9: The changes in prevalence over time ($\frac{\delta I}{\delta t}$) under perturbations of β in a random assignment setting

When considering the prevalence (Figure 5.10) a similar amplifying effect can be observed. As we have previously observed that heterogeneity in radiation does not (with exception of the extreme cases ($\beta = -0.5, -0.45, 0.45 \text{or} 0.5$)) influence the prevalence, absence of

interaction effects would imply that only in those extreme cases the prevalence effect should add up and vary from the scenario of heterogeneity only in reception. When considering the prevalence (Figure 5.10) we observe the changes in prevalence to be largest in the extreme cases, but present thought out all perturbations. This suggest that, rather than a cumulative effect, an amplifying effect occurs for prevalence. Hence we conclude that also for prevalence the heterogeneity in both sub-processes causes an interaction between the two which causes the constraints to be amplified and further reduces the prevalence.



(a) The prevalence under *negative* values of perturbation ($\beta < 0$) in a setting of random assignment to groups, effectively increasing the radiation and reception of the targeted groups ($g^r ad_1$ and $g^r ec_1$) individually by β



(b) The prevalence under *positive* values of perturbation ($\beta > 0$) in a setting of random assignment to groups, effectively increasing the radiation and reception of the targeted groups ($g^r ad_1$ and $g^r ec_1$) individually by β

Figure 5.10: The prevalence under different perturbations in the random assignment scenario

5.4 Discussion

In this study we have managed to show the impact of heterogeneity in local propagation dynamics caused by actor characteristics on consequent global propagation outcomes. In doing so we have shown that capturing and controlling for the influence of the heterogeneity in actor behavior (even in an homogeneous network structure) requires the adoption of a more nuanced view of propagation incorporating three sub-processes; Radiation, transmission and reception. By means of simulation we have shown that heterogeneity in radiation and reception on average have the same effect on propagation outcomes, but achieve this effect by very different means. Whereas heterogeneity in radiation mainly reduces the speed of propagation in the earlier stages and consequently yields lower success rate for propagation, heterogeneity in reception slows down the propagation process

more in the later stages and consequently reduces the prevalence of the process.
We found that incorporating heterogeneity in both locations in the process (both in radiation and reception) yields an interaction which amplifies the effects of both. This means that the constraints in both are amplified, but also that for some actors an amplified positive effect can occur. These actors will becomes more susceptible to propagation and also will send out more signals. The positive interactions yields a type of actor which serves as a hub for propagation dynamics, even though it is not a hub in terms of network structure. Having a substantial amount of these hub-like actors in the network allows for temporary outperforming the benchmark of a process (which does not include any heterogeneity). Consequently heterogeneity in local dynamics while generally hampering propagation can also enable quick ramp-up of the propagation towards a global cascade.

Our finding have some interesting implications policy makers, especially those who intend to initiate a global cascade (or prevent this). Take for example a marketing setting, and apply our findings. We have shown that in order to 'create' a cascade the radiation process is critical to take under consideration. Targeting the best radiators will increase the success rate of such a propagation process and hence are the most useful targets for seeding a product. While this might be logical from a conceptual standpoint this does imply that no longer the intended users (which are usually those which are most prone to adopt and hence are characterized by high reception) are to be targeted, instead one should rely on the fact that these actors will be reached via the propagation process. Similarly in a setting of contagious disease (where one intents to prevent spreading) assuming a new infection with very little seeds, one should focus their efforts on those who are likely to radiate, rather than those who are likely to receive. Clearly the underlying assumption here is that the cascade can still be averted and pushing the process towards the zero infection stable ($I_e = 0$) state is an option, something which is strongly dependent on the overall contagiousness of the disease.

Furthermore while we find that both locations of heterogeneity (in radiation and reception) yield on average a similar effect on prevalence, the differences in dynamics of reaching those have a strong implication for propagation processes that occur in our environment. As commonly we see cascades occurring only when they have a sufficiently large size, they are likely to have already passed the survival phase and consequently any influence of heterogeneity in radiation seems to have already been accounted for by the time we observe them. Therefore a fruitful direction for future research would be to see what happens to our findings when we assume a small group of seeds (rather then a single one).

Similarly we useful extension of this research would capture the effects of homophily. While the notion of homophily is touched upon in the literature section of this article it is not incorporated into the actual model. Given that we have identified the implications of the highway or backbone of the propagation process, we assume that homophily can strongly influence the effectiveness of such a feature. Using the analogy of a highway, if this highway cannot be used to reach the regions of the population far away from the seed of infection (something which would be in line with the logic of homophily, arguing that similar actors are clustered) would strongly hamper the benefits of having such a highway. Of course a competing argument could also be made, a highway can only be effectively leveraged if they are sufficiently connected, making incorporating the notion of homophily a logical next step in understanding the effects of heterogeneity in local dynamics.

In conclusion, an interesting observation (which is somewhat outside the scope of our study) is that while we found that having hub-like actors will facilitate propagation in the early stages of the propagation process, at some point the inherent constraints of having hub-like actors (namely that others have their radiation and reception reduced) do catch up with them. The benchmark will therefore always yield a higher prevalence in the equilibrium state. The finding that the default scenario (without heterogeneity) is never surpassed in terms of prevalence indicates that not incorporating heterogeneity in the modeling of propagation structurally provides an upper bound to the outcomes of propagation processes, a finding which is in line with the findings of Aral et al. (2013). This indicates that capturing heterogeneity in our models of propagation facilitates more depth and detail in the process, but also introduces additional abortive capacity to the system and reduces the prevalence of infections on them.
In contrast, heterogeneous actor behavior might also be the thing which allows processes to become a cascade in the first place. A propagation process without average behavior which allows them to become a system-wide cascade, might be able to leverage a highway of propagation (facilitated by heterogeneous actor behavior) and consequently cascade throughout the system.
Therefore including heterogeneity of actor behavior into the models of propagation has both a stabilizing and disruptive potential. Which one of the two will be leveraged might be very dependent on the scenario which is considered.

Chapter 6

Study 3: The effects of network structure heterogeneity

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Abstract:
Literature on propagation processes in recent years has focused on identifying the role network structure on the propagation outcomes. It is commonly accepted to assume that the network structure underlying the propagation process determines the outcomes of this process. As these structures are often complex, specifying the exact impact of the network structure on propagation outcomes remains a difficult task. While the network structure has been considered the prime driver impacting the propagation dynamics, in this chapter we show that the mechanism of the propagation process itself plays a crucial role in determining these effects. We show that the mechanism of propagation has a moderating effect on the relationship between the network structure and the propagation outcomes. Hence, our knowledge on the effects of network structure might not be as universally applicable as previously assumed.

[1]This chapter considers a working paper. A preliminary version of this work have been presented at the European Marketing Association Conference 2015 (EMAC2015)

Propagation, the process by which a change in behavior or state of one actor results in the change in one or more of its connected neighbors, is studied in many different fields of research. Examples are studies of contagious disease, spread of knowledge, cascades of failures in connected grids, adoption of innovation/behavior and diffusion of information. There is a vast body of work aimed at understanding and predicting the outcomes of such propagation processes, most of which has focused on studying the structure of interactions among actors. With research identifying the differences between random, scale-free (Barabási et al., 2000; Barabási, 2009) and small-world (Watts and Strogatz, 1998) topologies and others identifying the role of clustering (Girvan and Newman, 2002), it has become the standard to assume that the structure of interaction, the so-called network structure, is the key driver of propagation outcomes.

While this stream of research has lead to important insights on the impact of network topologies, key individuals within networks, and structural characteristics critical for propagation, one caveat should be placed at this body of work; many of it adopts a mechanism of propagation which might be over-simplified. The propagation process is described as the (stochastic) process by which actors adopt a certain state or behavior, effectively describing local propagation dynamics rather than the mechanism by which they appear. Based on ideas from communication literature (Shannon, 1948) this dissertation has posed that the mechanism of propagation consists of three distinct sub-process; Radiation, Transmission and Reception, suggesting that adoption (read reception) is only a part of propagation, and more nuanced view of the propagation mechanism is needed to get a complete picture.

Related work, which similarly has adopted a more nuanced view of propagation mechanism, suggests that the mechanism can play a moderating role in determining propagation outcomes (Centola et al., 2007; Aral et al., 2013). Such findings raise a question regarding the extent to which knowledge on the impact of network structure on propagation dynamics applies across different mechanisms of propagation, and how generalizable the role of network structure is.

In this study we address this question and do so by taking the following steps. In the second section we consider the common literature on the impact of network structure on the propagation outcomes, and further explain the notion of a mechanism incorporating three sub-processes of propagation. The third section will contain a description of the agent-based simulation approach used in this chapter, the results of this approach will then be presented and discussed in the fourth section. The chapter will conclude with a discussion of the implications of our findings.

6.1 Literature Background

The structure of interaction among actors has been the focus of many studies in previous literature. When referring to this structure of interactions the term 'network structure' is commonly used. While this label is accurate, as indeed this structure of interactions can be depicted as a network, the exact meaning of what structure entails remains somewhat unclear. As the network structure can take many different forms, there is no single strict definition of network structure. On the highest level of abstraction the network structure can be considered to be the graph which represents the interaction of actors, in which the actors are the vertices and the interactions are the edges. This graph serves as the infrastructure on which a propagation process can spread and hence underlies the propagation process. In order to have propagation an edge needs to be present between actors, and consequently the structure of the underlying graph constrains the propagation process. However, as the network structure can strongly vary across networks, this high level notion of network structure forgoes capturing the characteristics of that structure. Describing the structure of a network in more detail has proven a difficult task as these structures in practice are often complex. In order to reduce this complexity the structure can be broken down into characteristics on three levels; the micro-level, the meso-level and the macro-level.

Structure on the macro-level

The macro-level view of network structure builds on the notion that the properties of the structure of the network as a whole have a relationship with the propagation dynamics. In recent literature the properties claimed to have such influence are commonly related to the degree distribution, diameter and density of the network (Newman, 2003). This has consequently resulted in a set of network topologies; a regular (ring), random, small-world and scale-free network, which form the cornerstone of the studies of the macro level network effects.

Previous studies have shown that networks having different topologies yield different propagation outcomes. For example it is know that random network are inefficient in facilitating propagation, but are very resilient in their capacity to do so (Albert et al., 2000). Sparse regular networks are generally bad in facilitating propagation, but rewiring a small proportion of the ties randomly (the notion of small-world networks) greatly increases the propagation potential (Watts and Strogatz, 1998), and scale-free structures, by leveraging a skewed degree distribution, allow for even faster and more widespread propagation

(Albert et al., 2000; Pastor-Satorras and Vespignani, 2001a). Clearly, based on these studies one would draw the conclusion that the network topology indeed matters when determining propagation dynamics, and hence should be represented when describing the network structure.

Structure on the micro-level

The potential number of variations in network structures is virtually limitless, raising the question whether the coarse grain classification of network structures using topologies is capable of capturing the details necessary to fully understand the influence of network structure on propagation outcomes. Consequently a different approach of describing the network structure, the micro-level description of network structure, has co-evolved. This approach has focused on the smallest potential network structure; the ego-network. The ego-network describes the structure of one actor and all its connected neighbors and considers the network as a whole to be a sum (or average) of all these ego-networks. The ego-network structure is often described in terms of the density, reciprocity (Westphal and Zajac, 1997) and clustering (Girvan and Newman, 2002; Newman, 2003). This domain of network structural effects has yielded findings which revolve around the novelty of information being propagated. For example it has been suggested that within triads (groups of three actors) open structures are more prone to yield novel information (Burt, 2005; Granovetter, 1973) and closed structures are more efficient in information sharing (Uzzi, 1997), a tension which in social science is referred to as brokerage vs closure (Burt, 2005). These findings on the triadic level could be generalized to the network as a whole, suggesting that higher local density and lower local clustering yield higher levels of propagation.

Structure on the mese-level

Between the two extremes (macro and micro) there is a vast range of different network structural elements which can be considered, these are part of the meso-level analysis of network structure. In principle all properties describing sub-structures with a diameter of more than two (without covering the whole network) are considered part of the meso-level. While the meso-level of network structure is relatively under-studied, this area is where most of current work on network structure research is being done. There are some specific concepts in the meso-level domain which have received quite some attention;

there is work focusing on the communities within networks (Girvan and Newman, 2002), structural equivalence (Burt, 1987; Hanneman and Riddle, 2005) and degree assortativity (Newman, 2002). Other areas, such as the role of network motives (Alon, 2007) have received less attention, leaving large parts of the meso-level domain unexplored. Similar to the micro-level, meso-level measures considers local properties of the network which are then related back to the network (or the individual) level.
The studies on the effects of network structure on the meso-level have also yielded important insights into the role of network structure on propagation dynamics. It has for example been shown that propagation within clusters is usually rapid and propagation between clusters is more difficult (Rahmandad and Sterman, 2008). This once more suggest that also the meso-level characteristics are key in describing the network structure.

What becomes evident when considering these three levels of the network structure is that there is some general idea of which network structural factors impact propagation outcomes, however the question what the exact impact of the network structure is remains open and complex to answer. In literature characteristics relating to the distance/shortest path, the degree distribution (Newman, 2003), the degree correlation (Jackson and Rogers, 2007), the local density/clustering or the global clustering/communities (Girvan and Newman, 2002) are most commonly cited and hence these characteristics seem to be the most critical building blocks for answering this question. While comparing work on the influence of network structure on propagation outcomes one key assumption has however been made; it is assumed that the mechanism of propagation processes studied are similar. This assumption has led us to believe that; first, any variation in outcomes indeed stems from variations in network structure (rather than the process itself), and second, that findings can be translated from one setting to another. However, observing propagation phenomena in practice suggests that propagation mechanisms do differ quite extensively, suggesting that the assumption of similarity, and the subsequent conclusions, are not particularly realistic. Consequently, additional studying of the effects of the network structure under different propagation mechanisms is required to judge the generalizability of the knowledge obtained in this field.

6.1.1 Dynamics of the propagation process

When considering the process of propagation as described in literature, we find that nearly all propagation models are versions of the either the SIS, Bass-like, threshold or cascade models. While these models do differ from one another they share a commonality; they

consider the propagation process as the adoption of a certain behavior/state by an actor. More specifically they claim that the adoption process is determined by a single parameter, be it an infection rate, a proportion of infected neighbors, or a percolation probability, all these models assume propagation to be monolithic. In the information processing view of propagation put forward in this dissertation it is however claimed that propagation process consists of three distinct sub-processes; Radiation, Transmission and Reception (RTR). In this view the mechanism of propagation can therefore be described using three consecutive steps; First (a change in) the state/behavior of an actor results in a signal to this actor's outgoing ties (Radiation), then this signal is transmitted over this tie towards the alter (Transmission) and last the alter receives the incoming signals and changes its state accordingly (Reception). Adopting this way of modeling propagation allows for a more detailed and realistic way of describing the propagation process dynamics (see Chapter 2 and 4).
Decomposing the propagation process into three sequential steps adds additional parameters to the modeling of propagation. These parameters allow for describing the mechanism of propagation rather than only the outcomes (dynamics) of it. It has been shown that doing so increases the accuracy of the modeling even when correcting for such an increase in parameters (Chapter 4). Furthermore as it is observed that propagation mechanism are in reality complex, in contrast to the simple notion of propagation as a monolithic process which is adopted in most current literature, an increase in parameters is in fact required to realistically describe the propagation process.

The observation that the mechanism of propagation can be more complex than the monolithic process assumed in most propagation literature is not strictly ours. Centola et. al for an example already in 2007 posed that in some scenarios the adoption of an alternate state (reception) requires signals from multiple sources. In propagation scenarios where the behavior being propagated is either costly, risky, or controversial the willingness to participate and change behavior/state may require independent affirmation or reinforcement from multiple sources (Centola and Macy, 2007). The authors refer to this mechanism of propagation as 'complex contagion' which links the network structure not only to the propagation dynamics, but also to propagation mechanism. This suggest that the influence of structure becomes two-fold, one direct effect by constraining the potential paths of propagation, and a second indirect effect by influencing the propagation mechanism. They show that adopting a mechanism of complex contagion yields effects of network structure which are different from the effects seen in simple propagation. This provides evidence of a moderating effect of the propagation mechanism on the impact of

network structure on propagation dynamics.

The work on complex contagion has, to our knowledge, been the only work directly considering the interaction between the propagation mechanism and network structure that links this to dynamics of propagation. It provides but a single example of a mechanism in which such an interaction effect can be found, whereas many other could be proposed. A second example can be found when considering the mechanism of costly contagion. Costly contagion refer to the notion that sending out signals (radiation) is inherently costly. Actors sending out signals are constrained in their resources, be it time or money, and therefore are likely to send out only limited amount of signals (Bliss et al., 2014). Even though the network structure might allow for a broader range of signals being send out, the inherent costs of doing so will prevent actors from acting upon this opportunity. Consequently also the mechanism of costly contagion implies that locally the radiation sub-process might be constrained by something else than the network structure.

Another example of more a complicated propagation mechanism is captured by the idea of information overload. Information overload applies mostly to social actors, and refers to the notion that actors are bounded in their ability to effectively process information. As a consequence the amount of information being processed by an actor is constrained, and information which reaches an actor which is overloaded will have little to no effect on its behavior. Translating this to the information processing view suggests that information overload causes actors to have a bounded capacity to process incoming signals (Tushman and Nadler, 1978), implying that actors have a cap in the number of signals processed during the reception sub-process. Once more such a cap would suggest that locally the propagation dynamics are constrained by the propagation mechanism rather than the network structure.

What becomes evident from the previous examples is that the mechanism of propagation can play a critical role in determining the propagation dynamics. And while previous work has indicated the relevance of network structure as the key driver of dynamics, the mechanism has often been oversimplified during these studies. This raises the question whether the effects of network structure are similar under different propagation mechanisms, and under which circumstances the findings with regards to the effects of network structure in fact hold. These questions are explored by studying the effect of network structure in a range of propagation scenarios with varying mechanism. By doing so the potential interaction effect between two drivers of propagation dynamics; the mechanism of propagation and the network structure is explored. This provides insight in the extent to which our current knowledge regarding the impact of network structure can generalized across settings.

6.2 Methodology

Studying the moderating effect of propagation mechanisms requires an approach which allows variations in both the network structural dimension as well as propagation mechanism dimension in a controlled manner. As mechanisms can vary strongly, one would ideally validate the mechanism used by based on samples from field data, however the problem with this type of data is that both the network structure and mechanism vary simultaneously, and consequently it becomes impossible to distinguish between the effects of either driver of propagation dynamics. A simulation approach, in contrast to field data method, allows for changing one dimension at a time, and therefore provides the control and flexibility required for this study. The following sections will further elaborate on the method used and will address how both the network structure and the mechanisms of propagation dimensions are manipulated during this study.

6.2.1 Network structure

As the literature section has put forward there is a wide range of network structural characteristics which seem to play a role in determining the propagation outcomes. Most commonly characteristics relating to the distance/shortest path, the degree distribution (Newman, 2003), the degree correlation (Jackson and Rogers, 2007), the local density/clustering or the global clustering/communities Girvan and Newman (2002) are found to play a role. Observed networks in social systems can be described using these characteristics, and reveal to be commonly characterized by short distance (measured by shortest path length) between pairs of vertices, higher clustering coefficients (as compared to random networks), a 'fat tailed' (or even scale free) degree distribution, positive degree correlation between neighbors (so that higher-degree vertices are more likely to be linked to other higher-degree vertices) and a clustering among the neighbors which is inversely related to the degree (Jackson and Rogers, 2007).
Interesting to note is that while there is a vast number of studies considering the network structure by representing one or a few of these characteristics, the work that considers all of them simultaneously is largely missing. This is in part because a simple approach for generating networks which cover all elements mentioned is lacking, and hence synthetically creating such networks becomes a process which is complex in itself. In an attempt to cover this gap Jackson and Rogers (2007) have come up with a hybrid network generation process which generates networks using a meeting process, which is driven by both network and random meetings. The extent to which each driver plays a role has been parametrized and can be varied, and consequently a wide range of networks can be gener-

ated which fit (most) the previously mentioned characteristics. Jackson and Rogers (2007) show that this approach is well suited to represent networks observed in social systems. When comparing the network topology of the networks generated using this approach to those commonly studied in previous literature, we can see that indeed elements of the scale-free, small-world and random networks are captured using this approach, however the traditionally studied regular network topology is not covered. We therefore extend the approach of Jackson and Rogers (2007) by including a temporal driver in the meeting process. Adding this temporal driver results in a network formation process (of which the details are described below) with in total three drivers which, once parametrized, allow for generating networks all over the network characteristic space.

6.2.1.1 The hybrid network formation model

This section provides a brief summary of the hybrid network formation approach, a more detailed description can be found in the original paper by Jackson and Rogers (2007). The hybrid formation approach builds upon the notion that two forces are driving meeting processes of actors new to the network; a random driver and a network driver. It assumes that each time unit t a new vertex i is 'born' which will meet part of the existing network at time t ($G_t(V, E)$ with vertices $i \in V := \{1, \ldots, n\}$ and an edge-set $e \in E := \{1, \ldots, m\}$) using two distinct stages.
In the first stage, the random driver forces the newly born actor i to meet m_r vertices in the network ($G_t(V, E)$) at random (without replacement) and connects to each of them with probability p_r (in more complex scenarios p_r can be considered as a function of utility). The vertices connected to in this first stage are referred to as the parent vertices (J_p). In the second stage the network driver forces the newly born vertex to select all the neighbors of the parent vertices at distance k (default $k = 1$) and randomly select and meet m_n vertices among them and connect to each of these vertices with probability p_n (which can again be considered as a function of utility). After this has occurred the time is progressed one unit and the process is repeated.

In this study this approach is extended by assuming that there is an additional temporal driver at work during network formation. This temporal driver forces the newly born vertex to meet other vertices not in a random order but in a structured fashion and captures the notion of path dependence in network formation. It is assumed that the being born is not an exogenous process, in fact many forces influence the decision if and when to join a network. Most commonly these are local forces indicating that a neighbor has

connected to the network recently. Example are the building of snowflakes or the joining of social networking site (Kumar et al., 2010), both require some form of local interaction which determines 'when' to join the system. Therefore, it can be assumed that an actor is likely to meet and connect to those actors that have most recently joined the network. The temporal driver would take effect before the previously described random driver, and hence will serve as the first stage of the formation process.

To incorporate the temporal force into the formation process we assign to each vertex an age T_i, which describes the time it has spend in the system (how long ago it was born). The temporal driver then forces the newly born vertex to meet the m_a youngest vertices and connect to them with probability p_a. After this stage the hybrid network formation model goes through the traditional 2 stage as described above, in which we assume that the parent vertices are those connected to by means of both the temporal as well as random driving force.

The network formation process is initiated by forming a network of size $m_a + m_r + m_n + 1$ in which each vertex has $m_r + m_n$ edges. The three stages are then used to grow the network to the desired or network size. We incorporate an addition finalizing step at the end of the generation process after the desired network size is obtained. In this additional step the oldest m_a vertices in the network are selected and are linked to C_a youngest vertices with probability p_a, in which $C_a = \{m_a, \ldots, 0\}$. This way the oldest vertices are also linked to the youngest vertices, and all actors in the network have on average the same number of ties.

Adding the additional (temporal) driving force and finalizing step places the network formation into a larger context by assuming the process is partially driven by exogenous forces, and allows to generate network structures which cannot be obtained from the traditional hybrid formation approach. Our method allows parameters to be chosen such that the resulting network has a fully regular ring structure ($p_a = 1, m_a > 0, m_r = 0, m_n = 0$). By doing so it not only allows for a wider range of network topologies to be generated, but also to better able to achieve global clustering in the networks it generates, a notion which is largely neglected in the original approach of Jackson and Rogers (2007).

The method for network formation in this study is slightly more complicated than the traditional hybrid formation model, requiring a set of 7 (as opposed to 5) parameters as input; three describing the amount of meetings (m_r, m_n, m_a), three describing the chances of success per meeting (p_r, p_n, p_a) and one describing the distance (k) to which an actor can look into the network, the so-called network horizon (van Liere et al., 2008). We can reduce this number further by making two assumptions (similar to Jackson and Rogers

(2007)). It is assumed that the utility of a tie is independent of the meeting process which underlies its formation, and because of this we can state that the probabilities of success during each of these meetings types is equal ($p_r = p_n = p_a$). Furthermore, it is assumed that one cannot look beyond the direct alters of their parents ($k = 1$), leaving a parameter space with only four parameters.

During this study the meeting parameters will range from $0, \ldots, 3$, and the probability to connect during such meetings will be fixed to 1 ($p = p_r = p_n = p_a = 1.0$). Consequently 64 ($4^3$) different network structures are created. From the set of network structures created four are dropped (those in which $m_r = m_n = 0$) because no parent nodes are selected during formation, hence these networks do no reach a giant connected component of the desired size. Two additional networks in which only a single tie is formed each birth ($m_t = 1, m_r = 0, m_n = 0$ and $m_t = 0, m_r = 1, m_n = 0$) are dropped as well, because they yield structures which are unrealistic in their simplicity. A summary of the structural properties of the remaining 58 networks (Table 6.1) shows that the resulting networks have a wide range of structural parameters. A more detailed description of the properties of these networks can be found in table 6.8 in the supplementary information (SI) of this chapter. To validate the ranges in the structural parameters in our synthetic networks they are compared to values obtained from a sample of field data describing twitter interactions. The data of this network has been collected using the twitter API (Stonedahl et al., 2010). It represents a sub-graph of the twitter follower network with the same amount of users as the synthetic networks (1000). Starting with a random Twitter UID between 1 and 10 million a breath-first search was used to add the 999 nodes closest to the starting node to the network. Interactions are considered to represent a friendship when A follows B and B follows A, this resulted in a set of 13,343 friendship ties within this network.
The comparison with the Twitter data reveals that the synthetically created networks have a parameter range with nicely fits around the properties found in field data, suggesting that this set of networks approximates network structures which are realistic at least in social systems. At the same time the synthetic networks vary sufficiently across the structural network properties to cover the multidimensional network structure space well.

6.2.2 The RTR-model of propagation

The networks formation approach discussed in the previous section allows for varying the network structural dimension in this study, however in order to show the interaction affect, one also needs to specify the propagation mechanism. In this study a SIS-like propagation

Table 6.1: This table describes the network structural properties of the synthetic networks created using the hybrid network generation approach in which each meeting results in a link ($p = p_r = p_n = p_a = 1.0$).

	synthetic networks:		*Twitter Data*
	Min	Max	
Density_global	0.002	0.016	0.013
Clustering_local_avg	0	0.774	0.498
Clustering_global	0	0.600	0.246
Diameter	2	500	9
communities_WT	20	109	17
degree_assortativity	-0.333	0.417	−0.153
Degree_dist_skewness	0	31.523	7.445
Degree_min	4	20	1
Degree_max	4	1,998	733

process is assumed, in which actors change state between having adopted a certain behavior *(**I**nfected*) to being able to adopt a certain behavior (***S**usceptible*) effectively making this a binary state process. Similar to traditional SIS model it is assumed that transitions between states are driven by a stochastic process, however rather than assuming that propagation to be monolithic, the information processing view of propagation (Chapter 2 and 4) is adopted. This view dictates that the propagation mechanism consist of three sub-process; Radiation, Transmission and Reception (RTR). Each of these sub-processes is considered to be stochastic and hence on the dyadic level the chance of propagation can be considered as the product each of these processes ($\lambda = \alpha\phi\eta$).
A strictly dyadic propagation process would yield a simple model of propagation, however the propagation process in the information processing view is not strictly dyadic. The radiation sub-process is a one-to-many process, in which one actor can send out signals multiple alters, and reception is a many-to-one process, in which signals from multiple alters can be simultaneously received. The fact that propagation is not a strictly dyadic process, suggests one requires more complicated formulation of the propagation mechanism. The RTR-model of propagation provides such a description and allows for capturing propagation as a network process, rather than a dyadic one (A detailed description of the RTR-model and how it relates to other traditional propagation models can be found in chapter 2).

In this study an RTR-model is used which mimics SIS behavior. In this model the state of actors in binary (it is either infected or susceptible) and any actor which is infected has the potential to radiate signals. Consequently, the RTR-model can be described by three functions; one for each sub-process.
Based on the general RTR-model it is assumed that during each of the sub-processes there is an underlying set of characteristics which influence of the success of the sub-process. The characteristics affecting the radiation from actor i to outgoing edge e at time t are captured by $\alpha_{i,e,t}$. The characteristics affecting the transmission over edge e at time t are captured by $\phi_{e,t}$. The characteristics affecting the reception of an actor i from edge e at time t are captured by $\eta_{e,i,t}$, $\Psi_{i,t}$ then is the vector combining the reception information for each incoming tie of actor i at time t.
Using these parameters the radiation sub-process, which describes how the (change in) state ($s_{i,t}$) of an actor i results in a outgoing signal to an edge e on time t ($p^{out}_{i,e,t}$), can be described as:

$$p^{out}_{i,e,t} \sim Bern(s_{i,t} \times \alpha^{*}_{i,e,t}) \tag{6.1}$$

In which $\alpha^*_{i,e,t}$ is some function of $\alpha_{i,e,t}$ resulting in a chance of success during radiation. As the radiation is claimed to take place in all directions, towards all outgoing edges of i, $P^{out}_{i,t}$ is a vector describing the outgoing signal towards all outgoing edges of actor i on time t.

The transmission sub-process considers the process by which a signal is transported over an edge from actor i to actor j, effectively relating the signal going in to the edge to the signal coming out of the edge. It can therefore be described as:

$$p^{in}_{e,j,t} \sim Bern(\phi^*_{e,t} \times p^{out}_{i,e,t}) \tag{6.2}$$

In which $\phi^*_{e,t}$ is a function of ϕ resulting in a chance of success during transmission.

The reception sub-process describes how the signals from the incoming edges result in a state change of an actor, it can therefore be described as:

$$\Delta s_{j,t} = \begin{cases} \sim Bern(1 - \prod(1 - \Psi^*_{j,t})) & \text{if} \quad s_{i,t} = 0 \\ 0 & \text{if} \quad s_{i,t} = 1 \end{cases} \tag{6.3}$$

In which $\Psi^*_{j,t}$ is the vector containing all $\eta^*_{e,i,t}$, which are some function of a vector $\eta_{e,i,t}$ resulting in the chance of success during reception for each of the incoming ties. The product of the chance of failure for all incoming edges e results in the chance of total failure, and taking the inverse of this yields the chance of success from any of the incoming ties.

During simulations homogeneity in actor behavior assumed, this implies that all radiation parameters are the same ($\alpha^*_{i,e,t} = \alpha$), all transmission parameters are the same ($\phi^*_{i,e,t} = \phi$) and reception parameters are the same ($\eta^*_{i,e,t} = \eta$). Consequently any differences in outcomes can be directly linked to the differences in network structure or propagation dynamics. The moderating effects of propagation dynamics can then be isolated by comparing different network dynamics on the same network structure.

In the initial stage an approach similar to chapter 4 is followed. Four propagation processes with different mechanisms are simulated. Each one of them has a different decomposition into sub-processes, but has the same dyadic propagation likelihood ($\lambda = \alpha \times \phi \times \eta = 0.192$). The first scenario assumes that reception is driving the propagation process ($\alpha = 0.4$, $\phi = 0.6$ and $\eta = 0.8$), the second scenario assumes transmission is driving the propagation process ($\alpha = 0.6$, $\phi = 0.8$ and $\eta = 0.4$) and the third scenario assumes that radiation is driving the propagation process ($\alpha = 0.8$, $\phi = 0.4$ and $\eta = 0.6$). The fourth scenario is the baseline scenario which assumes propagation is monolithic, this implies

that all sub-processes are the same and can be captured by a single parameter ($\alpha = \phi = \eta = 0.5769$).
In the second stage a set of more complex mechanisms of propagation is studied. Using a similar simulation approach as before the mechanisms of simple propagation, complex contagion, costly contagion and information overload will be analyzed and compared.

6.2.3 Simulation

This section considers the details and parametrization of the simulations. The default state of all actors in the network is susceptible. The simulation are initiated by randomly selecting an actor in the network and perturbing its state (changing it to 'infected'), select up to 4 of the neighbors of this actor and give them the same perturbation, yielding a total seed size of up to 5 (0.5% of the network). For each of the described network structures and each propagation scenario a set of 100 simulations is run, each with randomly chosen seed. The results are then averaged over this sample.

As the propagation process is considered to mimic SIS propagation mechanism, a fixed recovery rate ($\rho = 0.2$) is included throughout the set of simulations. In populations with a low proportion of infected actors the average reproduction number is larger than 1 ($R_0 = \frac{\lambda k}{\rho} > 1$), and hence every actor is expected to yield more than 1 infection before it is recovered, resulting in a exponential growth in the number of infected actors (until the population becomes saturated). Combining this growing potential with the moderate size of the initial seed means that scenarios in which the propagation process dies out are very unlikely to occur. Therefore this value for the recovery rate facilitates widespread propagation.

In scenarios with widespread propagation, there are two relevant dimensions in which the propagation outcomes, its dynamics, can be measured; the speed by which the process occurs and size of the population that affected by the process. An SIS propagation mechanism dictates that the size of the population affected by the propagation process will grow until it reaches a (dynamic) equilibrium in which the proportion of newly infected actors is equal to the proportion of recovering actors. Resulting in a s-shaped adoption curve which converges into a (semi stable) dynamic equilibrium state. The proportion of infected actors in this equilibrium state is called the prevalence of an infection, and is often used as the key parameter describing the propagation dynamics on the system level (Pastor-Satorras and Vespignani, 2001a; Pastor-Satorras et al., 2014). In many cases it

is however not the equilibrium state itself, but the path towards it which is of interest for researchers. The time it takes for a propagation process to take off, or reach the equilibrium might be just as relevant.

As both speed and size of the propagation process seem useful in measuring propagation dynamics, for the purpose of this study a measure is used which allows capturing both aspect simultaneously in a single variable. Using the Net Present Value (NPV) for an propagation process (Goldenberg et al., 2007; Peres, 2014) allows us to do so
The NPV method build on a century old notion from economics which argues that money (or a resource in general) represents a value which increases over time (due to interest or investment returns) (Fisher, 1907). Hence having a 1 unit of money now is more valuable than having that same unit of money at a later point in time. A similar logic can be applied to the propagation process outcomes, suggesting that a change in behavior early on in the process has a value which is higher than one at a later stage. The NPV of a propagation process captures the momentum by which it impacts the system, a propagation processes with high NPV will reach a large amount of actors quickly, low NPV in contrast indicates either low a slow process, or a limited width of the process.
The notion of NPV can be implemented by devaluation of adoptions as time progresses. In this study a simple devaluation method is used: we assume that the value of an adoption is reduced by a fixed amount each times step. The NPV of a propagation process can thus be described as follows:

$$NPV = \sum_{t=0}^{100} I_t \times N(d-t) \tag{6.4}$$

In which I_t is the proportion of actors in the infected state at time t,N is the total number of actors in the system, and d is the default value of adoption. In this study a time-step describes only a small unit of time, therefore a devaluation of 1% per time-step is assumed effectively stating that $t = 0.01d$ (and thus $d = 100$). This allows for capping the number of time units (t) in our simulation to 100, at which point any new adoption will be worth 0.0.
Apart from capturing both the speed and size of the propagation process in a single measure, adopting NPV method for considering the outcomes of propagation has the advantage of collapsing the traditionally studied propagation curves into a single variable. By doing so it reduces complexity of interpreting outcomes, which in turn allows for relatively easy comparison of propagation outcomes over multiple scenarios. Given the

large number of different scenarios in this study, this reduction in complexity is critical for successful interpretation of the findings.

6.3 Results

In this study we consider 4 scenarios with varying mechanism and study those in a set of 58 different network structures, resulting in a total of 232 propagation scenarios. For each scenario a bootstrapped sample of simulations is run and the average NPV for each of these scenarios is calculated. To start of, the baseline model is considered. The baseline model assumes the mechanism can be described using a single parameter (one that applies to all three sub-processes). The results of analysis of the baseline model (Figure 6.1) show that there is quite some variation in the NPV across the different network structures, suggesting that indeed the network structure seems to play a crucial role in determining propagation dynamics.

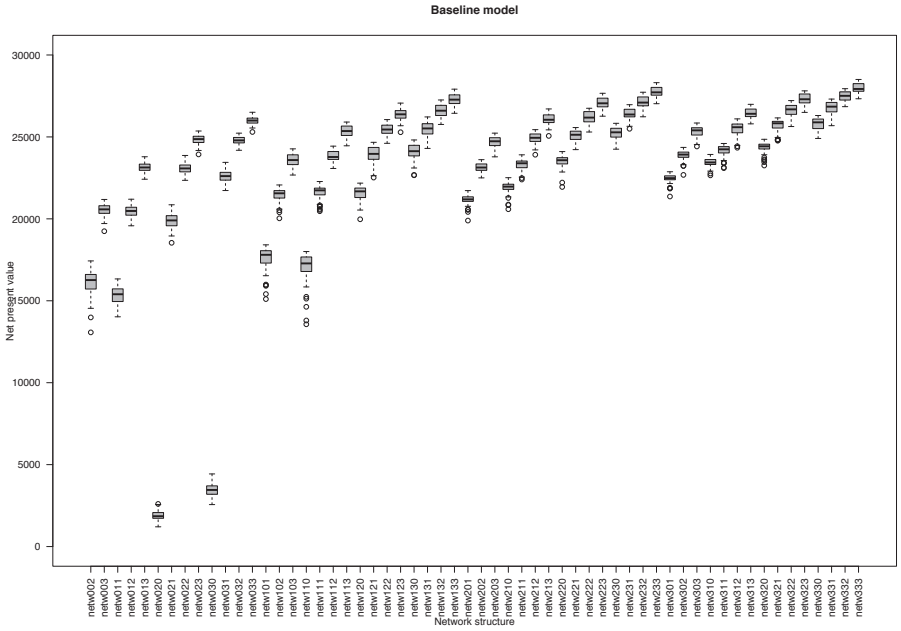

Figure 6.1: This figure shows on the Y-axis the Net Present Value (NPV) in a wide range of network structures (on the x-axis).

While this provides little insight into the effect of specific network structural characteristics, such effect is further explored by running a simple regression analysis. The results

of this analysis can be found in Table 6.2, and show a strong significant positive effects of density, and local clustering, indicating that both these structural characteristics increase the NPV and hence improve the propagation potential. The results also reveals a positive effect of degree distribution skewness supporting notion that a more fat-tailed degree distribution benefits increases propagation outcomes. A negative effect of global clustering is found supporting the notion that propagation between clusters is harder to achieve. All such observations are in line with what is commonly accepted as impact of network structure.

Table 6.2: Regression Results

	Dependent variable:
	NPV
	Simple propagation (baseline)
Global Density	370.534***
Local Clustering	61.512***
Global Clustering	−157.082***
Diameter	1.133.546***
Nr. communities	45.192***
Dgr. Assortativity	91.386**
Dgr. Dis. Skewness	82.609***
Dgr min.	−127.801***
Dgr max.	−48.327***
Constant	5.683.068***
Observations	5,800
Adjusted R^2	0.743
Note:	*p<0.1; **p<0.05; ***p<0.01

Not all results however seem to fit with notions from existing literature. A positive effect of diameter is found, suggesting that distance in the network would facilitate propagation, this is commonly accepted not to be true. Similarly the effects both minimum and maximum degree are negative, which would suggest adding ties has a negative effect. Also a positive effect is found for the number of clusters, while it is suggested that clustering hampers propagation (Rahmandad and Sterman, 2008).

One should note that there is some interdependence among the network structural indicators; often one cannot add ties without increasing the density as well, resulting in a positive net effect even though the effects of minimum and maximum degree are negative.

A similar argument can be made for local clustering and diameter; increasing the local clustering ceterus paribus will decrease the diameter, making these parameters somewhat interrelated. Also for the number of clusters such a claim can be made, when global clustering increases the number of clusters rises, however the net effect on propagation is still negative.
Such correlations might however influence the regression results and therefore a check for multicollinearity has been done. The varince inflation factor (VIF), which measures the extent to which variance of variables correlates, can provide useful insights into whether the correlation among variables poses a problem for analysis. The results of this analysis (table 6.3) show that the VIF values for parameter degree assortativity is (too) high, generally a value below 10 is acceptable.

Table 6.3: Variance inflation factors for regression variables

Variable:	*VIF:*
Dens_gl	6.172
Clust_loc	2.579
Clust_gl	2.232
Diameter	3.199
NR_commun.	1.406
dgr_assort	10.685
Dgr_dist_skew	5.679
Dgr_min	1.531
Dgr_max	3.855

Running the same regression without the degree assortativity (Table 6.4) seems to affect the model in a significant way, the effect of clustering disappears, the effect of degree distribution skewness and maximum degree are inverted. More importantly the overall quality of the model has suffered enormously, the R^2 has dropped by 0.44. suggesting that excluding this variable does not only reduce the multicolinearity, but the overall quality of the model as well. As the VIF of degree assortativity is only borderline problematic, and excluding it significantly harms the models explanatory power, we opt to include the variable in the model. As network structures are recycled across simulations the VIF values of the structural properties of these networks stay the same across scenarios, hence this decision applies throughout the remainder of this study.
One should however be aware of the the fact that some latent structural characteristic of the network might be represented in multiple variables in the regression analysis. There-

Table 6.4: Regression results baseline model excluding degree assortativity

	Dependent variable:
	NPV
Dens_gl	215.966^{***}
Clust_loc	198.060^{***}
Clust_gl	-63.048^{***}
Diameter	517.985^{***}
NR_commun.	-4.230
Dgr_dist_skew	-36.076^{***}
Dgr_min	193.630^{***}
Dgr_max	-198.880^{***}
Constant	$13,263.570^{***}$
Observations	5,800
Adjusted R^2	0.313
Note:	*p<0.1; **p<0.05; ***p<0.01

fore the interpretation of the regression results does require some caution in terms of drawing conclusions on the effect of specific parameters. Regardless, the baseline model as a whole has an adjusted R^2 of 0.743 indicating that the dimensions of structure do provide a good indication of propagation dynamics in this scenario.

The baseline model (Table 6.2) covers only a single mechanism of propagation, and many of such mechanisms exist. As the mechanism propagation is claimed to play a role in the propagation dynamics the baseline model is compared to three additional scenarios. Each of these scenarios covers a propagation process with the same overarching propagation probability, but with different decomposition into the three sub-processes of propagation. Each scenario therefore has the same dyadic propagation dynamics, but a different mechanism of propagation. The results of the comparison (Figure 6.2) reveals no significant differences among these scenarios, indicating that the propagation mechanism seems not to affect the dynamics of propagation.

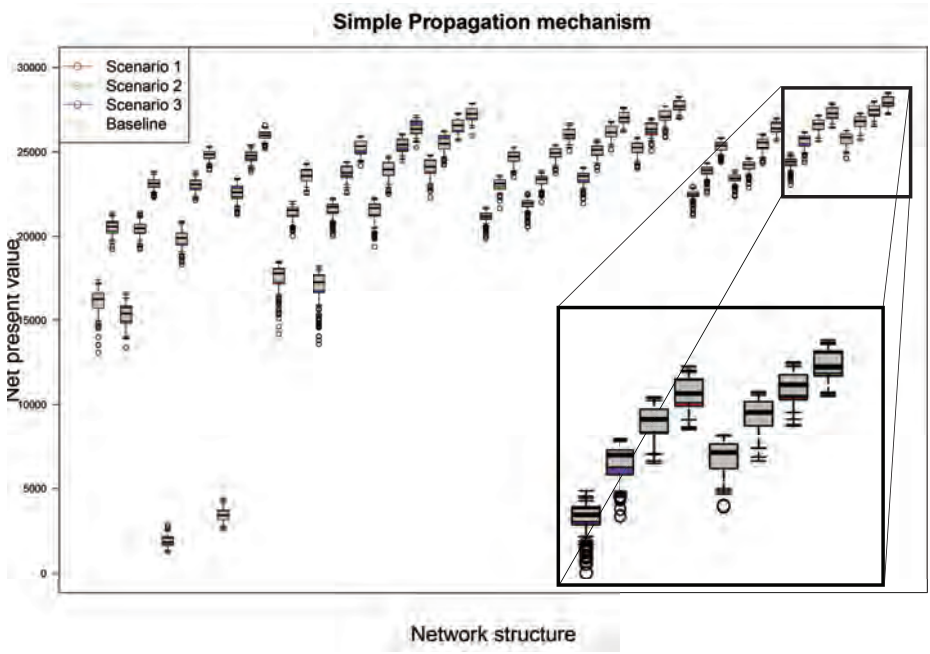

Figure 6.2: This figure shows on the Y-axis the Net Present Value (NPV) of processes with different propagation dynamics (indicated by color) in a wide range of network structures (on the x-axis).

The subsequent regression results presented in Table 6.5 further corroborate the notion that the effect of network structure is the same across all four scenarios.

Table 6.5: Regression results for a simple propagation mechanism

	Dependent variable:			
	NPV			
	(Scenario 1)	(Scenario 2)	(Scenario 3)	(Baseline)
Global Density	368.779***	370.126***	371.547***	370.534***
Local Clustering	61.364***	61.155***	61.586***	61.512***
Global Clustering	−156.348***	−156.612***	−156.934***	−157.082***
Diameter	1,131.935***	1,128.468***	1,138.710***	1,133.546***
Nr. Communities	45.106***	44.743***	43.669***	45.192***
Dgr. assortativity	90.019***	90.904***	92.858***	91.386***
Dgr. Dist. skewness	82.318***	83.575***	84.448***	82.609***
Dgr. min	−125.387***	−127.874***	−124.993***	−127.801***
Dgr. max	−47.215***	−47.376***	−48.446***	−48.327***
Constant	5,726.858***	5,686.745***	5,541.541***	5,683.068***
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.743	0.743	0.744	0.743
Note:				*p<0.1; **p<0.05; ***p<0.01

This finding seems to contradict results indicating that variations in the propagation mechanism, in some structures, yield different system-wide propagation dynamics (Centola and Macy, 2007). It should be noted that all models analyzed up until this point, while varying in their decomposition, have adopted a relatively simple mechanism of propagation. A simple mechanism implies that the propagation mechanism is unaffected by any source of outside influence. In contrast, in more complex propagation mechanisms, such as complex contagion (Centola and Macy, 2007), costly contagion and information overflow, it assumed that the mechanism is influenced by the structure, and hence an interaction effect between structure and the mechanism of propagation is present. The existence of such an interaction effect is explored by comparing our baseline model to models adopting these more complex propagation mechanisms. The full range analysis scenarios can be found in tables 6.9, 6.12, 6.11, 6.10 and figures 6.3, 6.4, 6.5 presented in the SI).

These results show that across the different types of complex propagation mechanisms a strong variance in the propagation dynamics can be observed. When the regression results

of the scenario 1 across the different mechanisms (Table 6.6) are compared it can be observed that the information overload and simple mechanisms are nearly similar, but that they differ radically from the other two mechanisms (which also differ from each other). The results indicate that the effects of local clustering, and the number of communities on NPV have turned negative in the costly contagion mechanism. For the mechanism of complex contagion the diameter, degree assortativity, and degree distribution skewness now have a negative effect on NPV, whereas the effect of minimum degree has turned positive. Also the effect of the maximum degree has disappeared altogether. These results suggest that the effects of some of the network structural characteristics vary significantly across different types of mechanism. Therefore these results refute the notion that the effects of network structure are constant, and give merit to the notions that the propagation dynamics are conditional upon the mechanism used. This supports the claim (Centola and Macy, 2007; Aral et al., 2013) that the mechanism of propagation is a key driver of propagation dynamics.

Table 6.6: Regression results for scenario 1 under different mechanism of propagation

	Dependent variable:			
	NPV			
	(Scenario 1) Complex Contagion	(Scenario 1) Costly Contagion	(Scenario 1) Information Overload	Scenario 1 Simple
Global Density	338.579^{***}	225.797^{***}	369.771^{***}	368.779^{***}
Local Clustering	101.405^{***}	-70.179^{***}	61.233^{***}	61.364^{***}
Global Clustering	-150.677^{***}	-47.473^{***}	-156.592^{***}	-156.348^{***}
Diameter	-398.234^{***}	978.281^{***}	$1.130,909^{***}$	$1,131.935^{***}$
Nr. Communities	19.916^{***}	-9.404^{***}	43.873^{***}	45.106^{***}
Dgr. assortativity	-171.151^{***}	112.655^{***}	93.450^{***}	90.019^{***}
Dgr. Dist. skewness	$-114/289^{***}$	64.000^{***}	83.263^{***}	82.318^{***}
Dgr. min	98.366^{***}	-113.618^{***}	-128.079^{***}	-125.387^{***}
Dgr. max	3.760	-50.000^{***}	-49.712^{***}	-47.215^{***}
Constant	$10,280.770^{***}$	$2,384.362^{***}$	$5,637.771^{***}$	$5,726.858^{***}$
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.898	0.855	0.742	0.743
Note:				*p<0.1; **p<0.05; ***p<0.01

Not only *between* the mechanism types, but also *within* these types variance in the dynamics and the effects of network structure can be observed. This indicates that, in contrast to the simple propagation setting, in complex propagation settings the decomposition of the mechanism plays a critical role in determining the propagation dynamics. This is particularly apparent when considering the costly contagion and complex contagion mechanisms.

In the costly contagion scenario (Figure 6.4) it can for example be observed that the scenario 1 consistently outperforms scenario 2. A closer look at the regression results (Table 6.11) reveals that while the direction of the effects of network structure are constant across the scenarios, the effect-size does differ among them, suggesting that not only the type of mechanism but also the decomposition plays a role in determining the effects of network structure.
Also in the complex contagion setting a difference in the effectiveness of the scenarios can be observed. Figure 6.5 for example shows that in this type of mechanism scenario 2 consistently outperforms scenario 1. Taking a closer look at the regression results in this setting (Table 6.12) shows that also here a strong variance in the effect-size exists across the different scenarios. Even inversion of the direction of minimum degree and degree assortativity can be seen in scenario 2 (although these results are barely significant).
The fact that variance is observed in the effect of the network structural elements indicates that the effects of network structure on propagation dynamics can no longer be considered constant within the context of complex propagation mechanisms. Both the propagation type and the network structure are kept constant in these comparisons, the decomposition of the mechanism is the only source of variance across these scenarios. This suggests that the effect of network structure are mediated not only by the propagation mechanism, but also by the decomposition of the mechanism into the three sub-processes.

While the results in this analysis are not sufficient to make exact claims about the extent or mechanism by which the mediation effect takes place, they can give some indication of how this effect occurs. An interesting observation can be made when considering the costly contagion mechanism. Table 6.11 shows that the effects of all network structural variables on NPV are constant in sign, but differ in strength. This suggests that the effects of the network structure are somehow weakened by the decomposition of the mechanism. Taking a closer look at the effect sizes reveals that there is a clear ordering in the strength of the effects. The effects of network structure are strongest in scenario 1, followed by the baseline scenario and scenario 2 and 3 respectively. Interestingly the radiation likelihood in these scenarios follows a similar (yet inverse trend) and is smallest in scenario 1, and biggest in scenario

Table 6.7: Showing the relationship between radiation probability and structural effect size in scenarios of **costly contagion**

Scenario	Radiation probability	Effect strength
Scenario 1	0.4	++++
Scenario 2	0.6	++
Scenario 3	0.8	+
Baseline	0.5769	+++

3 (Table 6.7). While this in no way provides proof of causality this observation does suggests that, for costly contagion, there is a negative correlation between the effects of network structure, and the probability of radiation. Exploring this relationship would be a viable extension of this research.

6.4 Implications

This study has focused in the role of the propagation mechanism in impacting the relationship between the network structure and propagation dynamics. By adopting an actor level information processing view and taking a closer look at what happens during propagation the mechanism of propagation has been decomposed into three sub-processes: Radiation, Transmission and Reception, and the effects of doing so have been explored. In doing so this study has made three contribution to the literature on propagation specifically and network research in general.

First, by building on the work of Jackson and Rogers (2007) and extending their hybrid model of network formation a new model for generating synthetic network structures has been put forward. This model allows for generating networks with a wide range of network characteristics, covering the space of network structural parameters from regular ring networks towards small-world and scale-free network (and all which lies in between). In doing so it provides a solid basis for generating synthetic network structure which resemble network structures observed in practice.

Second, this study has shown that the mechanism of propagation is a key driver of propagation dynamics on the system level. Significant variance in propagation dynamics across different type of more complex mechanisms has been found proving the importance of the considering mechanism of propagation in determining and understanding propagation dynamics.

Third, by exploring different mechanism across a wide array of networks with varying structural characteristics, it has been shown that the effect of network structure are constant only in scenarios with simple propagation mechanisms. When more complex mechanism of propagation are adopted the a single network structure can yield various propagation dynamics, suggesting that the effect of network structure is conditional on the mechanism of propagation.

This finding indicates a clear boundary to the generalizability of the effects of network structure on propagation dynamics. Whereas existing literature is largely supported in their findings on the impact of network structure, our findings suggest that these findings only hold under a simple propagation mechanism. When more complex mechanisms of propagation are adopted the impact of network structure might significantly change. This conclusion is in line with the conclusion drawn in previous work on complex contagion (Centola and Macy, 2007). In studying more complex mechanisms of propagation it is therefore a prerequisite to know the decomposition of the mechanism and understand the role of each of the sub-process of propagation.

6.5 Supplementary Information

Table 6.8

Network	Nodes	Density_global	Clustering_local_avg	Clustering_global	Diameter	communities_WT	degree_assortativity	Degree_dist_skewness	Degree_min	Degree_max	Degree_mean	Logic
network002	1000	0.00399779979997998	0.00556615737203972	0.00934995547640249	9	109	0.102086594561404	1.88102349373963	4	34	7.988	Bounded
network003	1000	0.00599339399399399	0.00740590477720981	0.01296020073633178	7	42	0.172720593905572	1.77998219486248	6	54	11.976	Bounded
network010	1000	0.002002002002002	0	0	500	34	NaN	NaN	4	4	4	Bounded
network011	1000	0.004	0.00390902430902431	0.00562851782363977	9	68	0.228985850332366	1.91372556750096	6	26	7.992	Bounded
network012	1000	0.00599599599599596	0.00705671419224051	0.0134869345321272	7	100	0.303625276173496	2.04266449989606	8	42	11.98	Bounded
network013	1000	0.00798998998998999	0.01010279217033905	0.01800794681477 1	6	89	0.295758559678829	2.07742327761958	10	58	15.964	Bounded
network020	1000	0.00400600600600601	0.500333333333333	0.500332889480692	250	28	0.197995991983968	22.2935417555851	8	10	8.004	Bounded
network021	1000	0.006002002002002	0.232904756354756	0.1942337852513319	8	65	0.291444478917062	1.91165466861238	10	26	11.992	Bounded
network022	1000	0.007995995995996	0.139532686680365	0.1088615786266494	6	64	0.360981996618155	1.91907949110863	12	42	15.976	Bounded
network023	1000	0.009987987987987799	0.09733033028016554	0.07879209929770537	5	69	0.388781119321166	1.6270100689063	14	50	19.956	Bounded
network030	1000	0.006012012012012012	0.600476190476619	0.600478787072749	167	24	0.330308436545785	17.9170971954461	12	16	12.012	Bounded
network031	1000	0.008006006006006001	0.353899184149184	0.319453076444997	7	37	0.332087321934708	1.81801336022345	14	32	15.996	Bounded
network032	1000	0.009997997997998	0.2386719046265325	0.20077679634154	6	64	0.405131641427346	1.76759066519591	16	42	19.976	Bounded
network033	1000	0.011987987987988	0.176173322272617	0.145732159601416	5	69	0.417162929890286	1.84639530958617	18	60	23.952	Bounded
network101	1000	0.003989989989989999	0.485584550027806	0.014500667842463	7	97	-0.164242828873807	28.6564139731336	4	988	7.972	Bounded
network102	1000	0.005973973973973973	0.175194968054689	0.0122136257793135	5	95	-0.14031591236499	26.1368471354253	4	1130	11.936	Bounded
network103	1000	0.007957957957957796	0.131129177583359	0.0169638682962666	5	59	-0.1060933113981911	26.0166909801898	6	1172	15.9	Bounded
network110	1000	0.003997997997998	0.450835386607571	0.0190283366797143	6	99	-0.13667737190819	31.5225657158367	4	958	7.988	Bounded
network111	1000	0.005891981981981981	0.132414214645176	0.011589458176461	5	63	-0.13328658720813	26.0343813998911	6	1072	11.952	Bounded
network112	1000	0.007973973973973973	0.132902763426898	0.0179091163033782	5	42	-0.104169743351692	26.2409956145827	8	1164	15.932	Bounded
network113	1000	0.009967967967967967	0.106846649489436	0.0228439245450766	4	29	-0.0836091812796215	25.8179525211492	10	1176	19.916	Bounded
network120	1000	0.006	0.490697603314778	0.0315151201057241	5	38	-0.1294565086657 31	27.4808811788225	8	1134	11.988	Bounded
network121	1000	0.007987987987987799	0.277202148743359	0.0305547135495605	5	56	-0.102257867038498	27.2285023134717	10	1210	15.96	Bounded
network122	1000	0.009969969969969697	0.197410012355237	0.0365763935111544	4	52	-0.0777321619785464	26.6859467455266	12	1148	19.92	Bounded
network123	1000	0.011953953953954	0.156639572119698	0.0396250532594802	4	51	-0.0695170521839662	25.6115085903971	14	1178	23.884	Bounded
network130	1000	0.008	0.545651595583291	0.0568294091229871	4	21	-0.100502343619354	27.9048608211018	12	1232	15.984	Bounded
network131	1000	0.009979979979997998	0.352792844962359	0.0609363961379547	4	41	-0.0795631580697 81	26.4320764352368	14	1152	19.94	Bounded
network132	1000	0.011973973973974	0.267159167418328	0.0599716967466116	4	55	-0.0672136490740846	26.9191330066574	16	1216	23.924	Bounded
network133	1000	0.013933933933933939	0.209012410517876	0.0626131547276438	4	42	-0.0581629097924312	25.6788837233844	18	1174	27.84	Bounded
network201	1000	0.005685685685685569	0.653805982458485	0.0144427832087407	4	58	-0.27630731379151 44	23.6269730002109	4	1554	11.36	Bounded
network202	1000	0.007293293293293329	0.338322382639361	0.0157109496626881	4	71	-0.18985755015463	25.5287917655673	6	1706	14.572	Bounded
network203	1000	0.009211211211211121	0.258087430427 36	0.0208881304880647	4	41	-0.145926234458677	25.2395248077155	8	1698	18.404	Bounded
network210	1000	0.005993993993993999	0.592342296248377	0.0149587312378504	3	68	-0.291800195027474	22.3173546332901	6	1402	11.976	Bounded
network211	1000	0.007347347347347735	0.292099409932792	0.0153124993037748	4	57	-0.186739257465144	25.2929952280483	8	1664	14.68	Bounded
network212	1000	0.009221212212212122	0.245014413536969	0.0214122110162581	4	49	-0.143492018691006	25.2433315066265	10	1668	18.424	Bounded
network213	1000	0.011229222922922922	0.196720042135251	0.0265909133618557	4	57	-0.115714373347354	24.9781443694586	10	1662	22.436	Bounded
network220	1000	0.007431431431431143	0.568335003789144	0.0247026354660655	4	35	-0.191661170922266	24.8551204638498	10	1666	14.848	Bounded
network221	1000	0.009223223223223322	0.358950281622038	0.0276799947945473	4	46	-0.14186481687797	25.4878950858259	10	1672	18.428	Bounded
network222	1000	0.011205205205205052	0.272177895951133	0.0335175419612112	4	44	-0.111132683418072	25.6040702434981	12	1660	22.388	Bounded
network223	1000	0.013193193193193192	0.226623340554752	0.0378653963504655	4	63	-0.0966089289194929	25.2343239683795	16	1688	26.36	Bounded
network230	1000	0.009431431431431134	0.564648081330932	0.0432077814088822	4	20	-0.13639013358971	25.5596409149346	14	1642	18.664	Bounded
network231	1000	0.011257257257257573	0.401249415646 03	0.0453463235387072	4	24	-0.111421483874 2	25.7241765571865	14	1674	22.492	Bounded
network232	1000	0.013211211211211212	0.319172527269096	0.0493405262752426	4	51	-0.0943765693454041	25.7201631177943	18	1700	26.396	Bounded
network233	1000	0.015149149149149149	0.263843951135647	0.0536993778217021	4	44	-0.0842170145746533	24.8186952291659	18	1672	30.268	Bounded
network302	1000	0.008160160160160116	0.41795050578373	0.0174990224009832	3	56	-0.216872502064 48	24.1685171466531	6	1932	16.304	Bounded
network303	1000	0.010106106106106061	0.3140746534731	0.0229973441015 94	4	43	-0.163360083292621	24.2676019350047	8	1912	20.192	Bounded
network310	1000	0.007327327327327733	0.774044605065583	0.0164129236704326	2	37	-0.333448623224327	22.5063575767317	8	1998	14.64	Bounded
network311	1000	0.008260260260260926	0.374094749229052	0.0176050121475834	3	52	-0.211770671168261	24.2513784588523	8	1922	16.504	Bounded
network312	1000	0.010112112112121121	0.305568197769509	0.0235722906106686	4	56	-0.160433360172373	24.3320163167178	10	1886	20.204	Bounded
network313	1000	0.012098098098098081	0.237633259889 51	0.0288850224356039	4	26	-0.127687728446052	24.15090816761	12	1838	24.172	Bounded
network320	1000	0.008342342342342234	0.615203838247439	0.0251054388144 99	3	33	-0.213750977209598	23.9226157257965	10	1902	16.668	Bounded
network321	1000	0.010130130130130101	0.397385904146396	0.0280462986517426	3	41	-0.162994167211567	24.1190539705029	12	1876	20.24	Bounded
network322	1000	0.012064064064064041	0.317232501819768	0.0336808527192695	3	62	-0.129773452040332	24.5789149910472	12	1906	24.104	Bounded
network323	1000	0.01404204204204 2	0.255476293421281	0.0384257748274227	4	56	-0.108369484440297	24.4225017185148	14	1880	28.056	Bounded
network330	1000	0.010170170170170702	0.596864996909745	0.0392098369965766	4	21	-0.159588920884279	24.4250591944431	14	1890	20.32	Bounded
network331	1000	0.012146146146146461	0.428943221491271	0.0432633771101 65	4	33	-0.129596664367727	24.3358610768277	14	1874	24.268	Bounded
network332	1000	0.014064064064064041	0.344157586319224	0.0472497594207769	4	32	-0.111455924075933	23.8889035766273	16	1842	28.1	Bounded
network333	1000	0.016084084084084841	0.286359873976681	0.0518105534210148	4	29	-0.0973523667352503	23.6723569070581	20	1846	32.136	Bounded

Table 6.9: Regression Results for **Simple** propagation mechanism

	Dependent variable:			
	NPV			
	(1)	(2)	(3)	(baseline)
Dens_gl	368.779***	370.126***	371.547***	370.534***
Clust_loc	61.364***	61.155***	61.586***	61.512***
Clust_gl	−156.348***	−156.612***	−156.934***	−157.082***
Diameter	1,131.935***	1,128.468***	1,138.710***	1,133.546***
NR_commun.	45.106***	44.743***	43.669***	45.192***
dgr_assort	90.019***	90.904***	92.858***	91.386***
Dgr_dist_skew	82.318***	83.575***	84.448***	82.609***
Dgr_min	−125.387***	−127.874***	−124.993***	−127.801***
Dgr_max	−47.215***	−47.376***	−48.446***	−48.327***
Constant	5,726.858***	5,686.745***	5,541.541***	5,683.068***
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.743	0.743	0.744	0.743
Note:				*p<0.1; **p<0.05; ***p<0.01

Table 6.10: Regression Results for **Information Overload** propagation mechanism

	Dependent variable:			
	NPV			
	(1)	(2)	(3)	(Baseline)
Dens_gl	369.771***	337.894***	359.978***	358.689***
Clust_loc	61.233***	58.189***	59.771***	59.791***
Clust_gl	−156.592***	−152.491***	−155.182***	−155.391***
Diameter	1,130.909***	1,111.535***	1,124.954***	1,122.137***
NR_commun.	43.873***	37.790***	42.190***	42.284***
dgr_assort	93.450***	96.833***	93.927***	94.884***
Dgr_dist_skew	83.263***	83.587***	84.430***	84.884***
Dgr_min	−128.079***	−132.829***	−133.459***	−132.311***
Dgr_max	−49.712***	−48.401***	−47.752***	−47.409***
Constant	5,637.771***	5,422.318***	5,593.661***	5,530.324***
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.742	0.709	0.734	0.733
Note:				*p<0.1; **p<0.05; ***p<0.01

Table 6.11: Regression results for **Costly Contagion** propagation mechanism

	Dependent variable:			
	NPV			
	(1)	(2)	(3)	(4)
Dens_gl	255.797***	184.505***	140.947***	191.757***
Clust_loc	−70.179***	−72.274***	−66.078***	−71.446***
Clust_gl	−47.473***	−33.438***	−24.226***	−35.056***
Diameter	978.281***	714.467***	526.428***	751.296***
NR_commun.	−9.404***	−2.963*	0.840	−2.522
dgr_assort	112.655***	80.617***	56.792***	84.996***
Dgr_dist_skew	64.000***	42.476***	27.183***	43.869***
Dgr_min	−113.618***	−70.001***	−41.620***	−73.809***
Dgr_max	−50.000***	−35.254***	−24.238***	−37.919***
Constant	2,384.362***	2,965.865***	2,959.129***	2,833.484***
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.855	0.859	0.862	0.855
Note:				*p<0.1; **p<0.05; ***p<0.01

Table 6.12: Regression results for **Complex contagion** propagation mechanism

	Dependent variable:			
	NPV			
	(1)	(2)	(3)	(4)
Dens_gl	338.579***	582.286***	457.137***	475.090***
Clust_loc	101.405***	110.658***	108.068***	110.160***
Clust_gl	−150.677***	−251.107***	−200.524***	−204.589***
Diameter	−398.234***	−559.506***	−493.917***	−488.156***
NR_commun.	19.916***	41.856***	27.921***	31.265***
dgr_assort	−171.151***	17.079**	−115.301***	−99.497***
Dgr_dist_skew	−114.289***	0.710	−73.789***	−66.200***
Dgr_min	98.366***	−24.487*	56.170***	55.725***
Dgr_max	3.760	−114.356***	−42.677***	−50.976***
Constant	10,280.770***	8,009.745***	10,291.560***	9,666.867***
Observations	5,800	5,800	5,800	5,800
Adjusted R^2	0.898	0.888	0.915	0.912
Note:				*p<0.1; **p<0.05; ***p<0.01

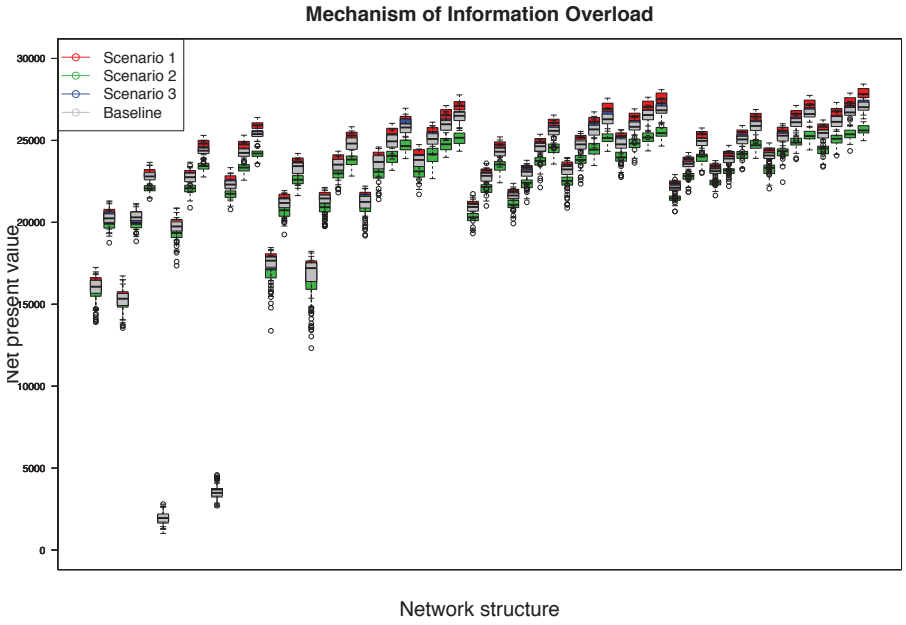

Figure 6.3: This figure shows on the Y-axis the Net Present Value (NPV) under different network structures (x-axis) for three scenarios (indicated by color). Each of these scenarios adopts an **Information Overload** mechanism but has different decomposition of the sub-processes of propagation.

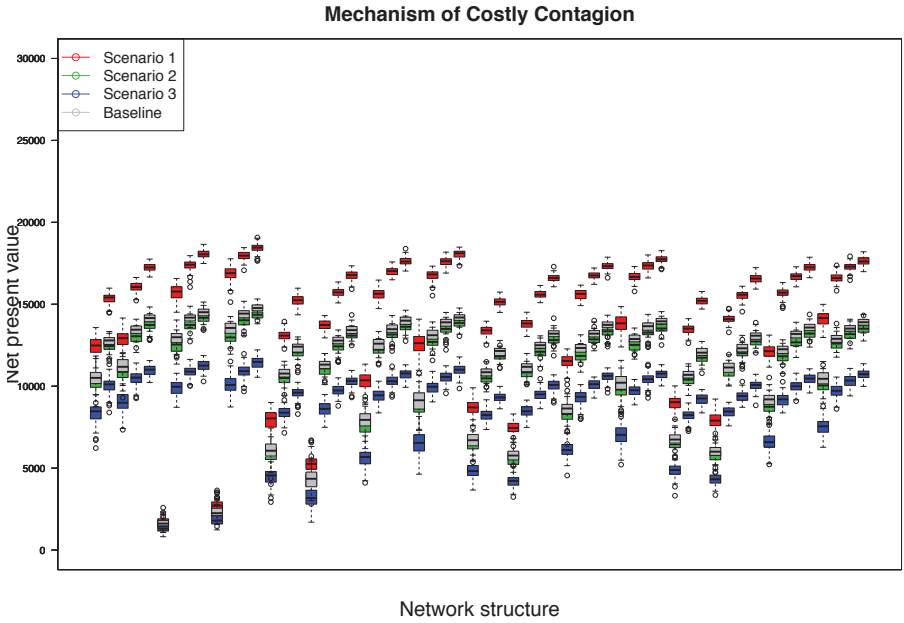

Figure 6.4: This figure shows on the Y-axis the Net Present Value (NPV) under different network structures (x-axis) for three scenarios (indicated by color). Each of these scenarios adopts a **Costly Contagion** mechanism but has different decomposition of the sub-processes of propagation.

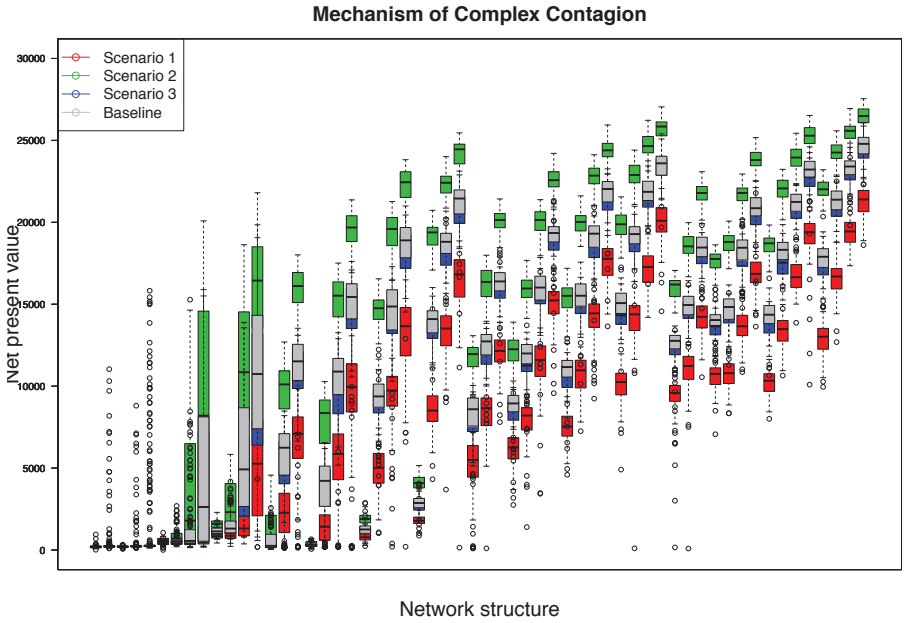

Figure 6.5: This figure shows on the Y-axis the Net Present Value (NPV) under different network structures (x-axis) for three scenarios (indicated by color). Each of these scenarios adopts a **Complex Contagion** mechanism but has different decomposition of the sub-processes of propagation.

Chapter 7

Summary and Conclusion

This dissertation aimed to show the impact of the propagation mechanism on propagation dynamics. It has done so by introducing a framework which considers the direct impact of the mechanism of propagation on propagation dynamics, and the interaction effect of this mechanism with the other drivers of propagation dynamics; the seed of infection and the network structure. This has resulted in three research questions, which subsequently have been the focus of the three studies executed in this dissertation. This chapter will revisit these research questions, and their answers, and will synthesized them into a discussion on the lessons learned with regards to the impact of the mechanism of propagation. Furthermore it will provide a reflection on this discussion focusing on the implications, limitations and future research.

The mechanism of propagation has been studied by adopting the information processing view of propagation. This view, introduced in chapter 2 and 4, considers propagation to be a process which consist of information processing at the actor level. The main research question in this dissertation therefore is:

> *(How) do differences in the information processing at the actor level affect system-wide propagation dynamics?*

The answer provided to this question in this dissertation is that the propagation mechanism indeed is an important driver of propagation dynamics. Adopting the information processing view, which captures such mechanism and describes it by means of three actor level information sub-processes, provides a useful method for studying propagation dynamics on the system level. It has been shown that the decomposition of the mechanism of propagation and identifying the information processing behavior on the actor level is key in determining the effects of interventions, network structure, and heterogeneity in

actor behavior.

It has been found that actor level information processing behavior does not have added value in all scenarios. In scenarios with homogenous or simple propagation mechanisms the propagation dynamics seem to be unaffected by the differences in actor level information processing. Models are by design an abstraction of reality, and the goal is often to keep the models as simple as possible, for this reason modeling propagation by means of a traditional models is a valid choice, in scenarios in which one considers a propagation process with a simple mechanism, and as long as both the effectiveness of interventions and the impact of local heterogeneity are irrelevant.

It should be noted that the propagation mechanisms in practice are likely to be far more complex than assumed in such models. Often the mechanism will vary locally and be dependent on outside factors. This dissertation has shown that under such conditions the decomposition of the propagation mechanism is critical to consider. In such settings the information processing on the actor level will locally affect the propagation mechanism, resulting in heterogeneity in local dynamics. This heterogeneity in turn is essential in order to understand and effectively model propagation dynamics on the system level. It is therefore highly desirable to adopt an information processing view of propagation.

In order to substantiate these conclusions, a recap of the three studies in this dissertation and an discussion on their findings is provided next. The first study in this dissertation (chapter 4) aimed to address part of the primary research question; it focuses on the whether any impact exists of actor level information processing behavior of system-wide propagation dynamics. The question *how* such impact occurs is covered in the second and third study in this dissertation.

7.1 Study 1

To show the effect of differences in actor level information processing, the first study (chapter 4) provides a translation of the information processing view of propagation into a model of propagation. This model considers propagation as a local process which takes place at the actor level and by doing places emphasis especially on the mechanism of propagation. It describes the mechanism of propagation by means of three sub-processes which occur between actors; Radiation, Transmission and Reception, resulting in the

RTR-model of propagation.
Adopting a new model of propagation (such as the RTR-model) first and foremost requires validation. Not only should it be shown that the RTR-model is a valid method for studying propagation in a different way, but also that adopting a different approach indeed makes sense; that it is a difference which makes a difference. Validation of the RTR-model of propagation therefore rests upon two pillars; the model needs to be methodological sound, and it needs to be practically relevant.

The first part of study 1 has tested the ability of the RTR-model to describe propagation phenomena. Field data of the adoption of a mobile phone application (Yahoo! GO 2.0) is used as an example of a propagation phenomenon. This data describes the adoption of Yahoo! GO 2.0 among a network of Yahoo! messenger users, and hence considers propagation in a networked setting. The performance of the RTR-model in describing this adoption process is consequently put forward and compared to more traditional models of propagation.
The results of this analysis (Table 4.5) show that the RTR-model allows for better reproducing actual adoption data, regardless of the measure of fit which is chosen, indicating that it does a better job at describing this propagation phenomenon. In the worst case scenario using the RTR-model yields a reduction in the errors of the number of adopters by a factor 1.5 over traditional single parameter models, which is a vast improvement in model accuracy. Although the exact value of such a reduction is likely to be dependent on the setting studied, it indicates that the RTR-model is superior in terms of describing the propagation process.
To some extent the improvements in fitting results are caused by the model design. The RTR-model adopts more detailed description of the propagation mechanism, it describes the mechanism using 3 rather than 1 parameter. Including more parameters in a model is likely to improve fitting results. However, when penalizing for the inclusion of additional parameters by testing the AIC (Akaike, 1974) lower model scores are observed, indicating that the RTR-model produces results superior over simple single parameter models. This shows that the RTR-model is methodologically sound, and that by providing a more detailed description of the mechanism of propagation this model is able to capture (part of) dynamics of propagation which simpler models do not.

In this dissertation it has been argued that the mechanism of propagation consist of three sub-processes, this has important implication for interventions in propagation processes. If one considers a stochastic, binary state setting, the decomposition of the propagation

mechanism suggests that the local dynamics (the local chance of successful propagation between two actors) are the result of a function of the chances of success in each of the sub-processes $\lambda = \alpha \times \phi \times \eta$. It has shown that interventions can be linked to perturbations in one of the specific sub-processes, which suggests effect of any perturbation (in either α, ϕ or η) will vary depending on the other sub-processes. For this reason the effectiveness of interventions will fluctuate depending on how the mechanism is decomposed into the three sub-process.
The scenario highlighted in chapter 4 (Figure 4.2 & Table 4.3) supports such a claim, and shows that interventions can have very different results depending on the mechanism of propagation. Analysis of the results shows that, in this specific scenario, both an over-estimation by a factor 2 and an under-estimation by a factor 8 of intervention effectiveness are possible. The difference in intervention effectiveness can even vary by a factor 14 when the most extreme mechanisms are compared. While little weight should be attributed to the exact numbers in these results (as they strongly depend on the scenario being simulated), the results clearly indicate that the effect of an intervention can and will significantly vary depending on the decomposition of the propagation mechanism. Consequently, significant mistakes in estimates of intervention effectiveness can occur when the decomposition of the propagation mechanism is foregone. Effectively steering and intervening in the propagation dynamics therefore requires insight into the mechanism of propagation and the decomposition into the three sub-processes, which is a clear indication of the practical relevance of adopting this new RTR-model of propagation.

The results of the first study seem paradoxical with regards to propagation dynamics; on the one hand the mathematical formulation suggest differences in the mechanism play no role in the dynamics, on the other hand field data fitting suggests that the capturing the mechanism allows for capturing part of the dynamics which simpler models do not.
The mathematical formulation of propagation ($\lambda = \alpha \times \phi \times \eta$), suggests that the mechanism does not make a difference for the propagation dynamics. As long as the product of the chances of success in each of the sub-processes ($\alpha \times \phi \times \eta$) remains the same the local dynamics do not differ, which consequently should result in the same system-wide dynamics. The initial simulation results from the first study (Figure 4.22) support this claim, and show that processes with the same local dynamics (the dyadic chance of successful propagation) will yield similar system-wide dynamics, regardless of how their mechanism is decomposed into the three sub-processes. This suggests that the mechanism has no added value for understanding the system-wide propagation dynamics (in scenarios where no interventions occur).

The methodological validation of the RTR-model however seems support an alternative conclusion. It showed that the decomposition of the mechanism into three sub-processes improves the accuracy of the propagation model, suggesting that such decomposition captures part of the dynamics which other models do not.

While these observations seem to contradict, they in fact do not, they are however based on a different set of assumptions. Whereas adopting the information processing view of propagation —and decomposing the mechanism into three sub-process— does not impose any additional constraints on the propagation dynamics, the simple equation based approach does. In the latter make the implicit assumption that the propagation process is purely dyadic, and homogenous across the system. This assumes that the local dynamics of propagation are independent from influences beyond the dyad, and are the same across all ties. As such, the equation based perspective describes a special instance of the more generally applicable information processing view of propagation (which does not (by default) make these assumption).
While these assumptions effectively offsets the impact of the mechanism of propagation on the propagation dynamics, and thus allow for a neat way of formulating the propagation process. One should be aware that in cases where these assumptions do not hold the simple mathematical way of viewing propagation breaks down. As the mechanism of propagation in practice is prone to show local heterogeneity and is not purely independent it is doubtful whether these assumptions are realistic in the first place. Therefore, it is argued that in many real world setting the information processing at the actor level starts playing a significant role in determining the propagation dynamics, and that the mechanism by which propagation occurs becomes critical to consider in such settings.

These insights can be linked to the first research question, and suggest that the differences in actor level information processing have an effect on the system-wide propagation dynamics. However, it does indicate that such effects are conditional on there being local heterogeneity or interdependence in the mechanism. This reveals little about what such an effect might be, but does provide a clear direction for studies aiming to better understanding such effects. Consequently in the following studies the role of respectively the local heterogeneity in mechanism and independence of the mechanism are considered.

7.2 Study 2

The second study in this dissertation (chapter 5) considered the effects of local heterogeneity on propagation dynamics while adopting the RTR-model of propagation. It specifically focused on the actor behavior as a source of heterogeneity and consequently aimed to addressing the third research question:

> *What role does heterogeneity of actor information processing behavior play on propagation dynamics?.*

Answering this question sheds light on the interaction among local heterogeneity and propagation mechanism. It aims to show the effect on the dynamics of propagation of such interaction. By assigning individual parameters for radiation and reception to actors, local heterogeneity can be linked to heterogeneity in actor behavior. Controlling for the effects of the network structure (the third driver of propagation dynamics) and the seed of infection, by respectively keeping the structure regular and constant (the same for all actors at any time) and averaging over all potential seeds, guarantees that all changes in dynamics are the result of heterogeneity in actor behavior.

Initial results of the second study (Figure 5.1) show that heterogeneity in the **radiation** sub-process does not significantly effect the proportion of the population which is affected by a propagation. The 'prevalence', the proportion of the population which is affected at a certain point in time, remains equal after introducing heterogeneity in the radiation sub-process. A closer look reveals that the prevalence for processes that in fact yield wide spread cascades of propagation remain the same, the chance that such widespread cascades occur is however decreased by introducing heterogeneity in the radiation process (Figure 5.6). Introducing heterogeneity in the radiation sub-process also affects time required to reach an (dynamic) equilibrium state in the system. It is found that the larger the extent of heterogeneity introduced in the radiation sub-processes the more the speed of the propagation process seems to be reduced (Figure 5.2).
This suggest that heterogeneity in the radiation sub-process increases the chances of the process dying out in the early stages, it hence reduces the chances of success. But when the propagation process has gained momentum, and has scaled toward a critical mass, such risk are negated and the constraints on propagation dynamics will dissipate, resulting in 'normal' amounts of prevalence. When taking into account also the non-successful instances of propagation, (those where the process dies out) the average prevalence will be reduced, indicating that heterogeneity does have a constraining effect on propagation

dynamics.

Introducing heterogeneity in the **reception** sub-process has a different effect on the propagation dynamics. While the chances of widespread cascades of propagation seem unaffected by introducing heterogeneity in this sub-process (Figure 5.6), the speed of propagation is affected (Figure 5.4). This effectively reduces that rate at which propagation will occur throughout the system. Such a reduction in speed will ceterus paribus translate into lower levels of prevalence at the system level (Figure 5.3), and hence also heterogeneity in the reception process has a constraining effect on propagation dynamics.

The effects of heterogeneity observed in both sub-processes, support the notion that heterogeneity in actor behavior plays a crucial role in determining system-wide propagation dynamics. On average heterogeneity in radiation and reception have a similar effect, both reduce the extent of propagation (Figure 5.5). One should however discriminate among heterogeneity in radiation and reception when one wants to know *how* such an effect occurs. The mechanisms by which either source of heterogeneity effects the propagation dynamics differs radically. Whereas heterogeneity in radiation reduces the chances of successful propagation, which in turn reduces the average prevalence of propagation but not the prevalence of successful propagation, heterogeneity in reception reduces the prevalence of successful propagation.

Heterogeneity in either sub-process, radiation or reception, is likely to occur due to inherent differences in actor characteristics, it is however unlikely that either of them will occur in isolation. A more realistic assumption is that heterogeneity in both sub-processes will occur simultaneously, and that the effects of both sources of heterogeneity will interact. To further answer the question how heterogeneity in actor behavior affects the propagation dynamics the second part of study 2 explored this interaction effect.

Two scenarios have been considered; A scenario of linked assignment, in which for a targeted group of actors both the radiation and reception is perturbed. And a scenario of random assignment, in which heterogeneity is introduced by perturbing of radiation and reception of actors independently and randomly.
When compared to a scenario without heterogeneity, the results of the linked assignment scenario (Figure 5.7 and 5.8) show a speedup of propagation in the early stage of the propagation process, but a strong restriction on the prevalence at the equilibrium stage. This indicates that the propagation process can capitalize on a backbone of highly effective

actors in the early stages. These actors have both high rates of radiation and high rates of reception, making them both easily infected and likely to infect others. Consequently these actors facilitate hub-like behavior and serve as a 'high-way' of propagation. While heterogeneity has shown to have a constraining effect on propagation dynamics, leveraging this high-way of propagation allows for offsetting these constraint, be it temporary. As the size of the high-way is limited, once saturated this effect will dissipate and the constraints of heterogeneity are once more in full effect.
The results of the random assignment scenario (Figure 5.9 and 5.10) show no sign of speedup, indicating that the size of the 'highway' (which in this particular case is three times as small as the previous scenario) is too small to offset the constraints introduced by heterogeneity. Without being able to leverage an initial boost in propagation speed the propagation process is slowed down to such an extent that it is slower than introducing heterogeneity in any one single sub-process. This indicates that effect of introducing heterogeneity in the radiation sub-process is amplified by introducing heterogeneity into the reception process, and vice versa. The interaction of both sources of heterogeneity causes an amplification in the constraints on propagation, further reducing the speed and size of propagation.

In conclusion the second study has shown that heterogeneity in both radiation and reception has a dampening effect on the extent of propagation on the system level. The mechanism by which such effects occur however differs among both sources of heterogeneity, whereas heterogeneity in radiation reduces the chances of successful propagation, heterogeneity in reception reduces the prevalence during successful propagation. Interaction between both effects has a amplifying effect on these constraints, further reducing propagation, but such effects can be initially outweighed by leveraging a potential 'high-way' of connected actors which show hub-like propagation behavior.

7.3 Study 3

The third study in this dissertation (chapter 6) aimed to address the interaction effect between the propagation mechanism and the network structure. It considered the assumption that the propagation mechanism itself, and the consequent effect of differences in this mechanism, are independent from this network structure. In doing so it focuses on answering the second research question in this dissertation, which reads:

> *How does information processing at the actor level affect the effects of network structure on system-wide propagation dynamics?*

The notion that the structure of interaction underlying the propagation process, the network structure, determines the dynamics of the propagation process is widely accepted in literature. Therefore in recent years literature on propagation has focused on identifying the impact of different network structural characteristics on propagation dynamics (e.g. (Barabási et al., 2000; Barabási, 2009; Rahmandad and Sterman, 2008; Watts and Strogatz, 1998)). This study shows that it is crucial to consider the mechanism of the process itself while doing so, as the mechanism can have a moderating effect on the relationship between the network structure and the propagation dynamics.

Using an agent-based simulation approach the effects of network structure under different scenarios of propagation are explored. Propagation scenarios with varying mechanisms (the same dyadic chance of propagation but different decomposition of their mechanism into the three sub-processes) are put on a set of networks with various network characteristics. The effect of the structural characteristics of the network are regressed against the outcomes of the propagation simulation, which in this study is measured by means of the Net Present Value (NPV) (Stonedahl et al., 2010; Goldenberg et al., 2007). The results of this regression (Table 6.9) show that regardless of the decomposition into sub-processes the network structure has a similar effect on the propagation dynamics. The found effects for different elements of network structure seem to be in line with findings from previous literature. They suggest that for example skewness of the degree distribution (Albert et al., 2000; Pastor-Satorras and Vespignani, 2001a) enables propagation, and that clustering facilitate local propagation but reduced propagation between clusters (Rahmandad and Sterman, 2008).

These initial simulation results indicate that the effects of network structure are constant over different mechanisms of propagation. Which would be an argument for stating that the actor level information processing does not have a mediating effects on the impact of network structure. However these findings are based on, and only hold in, scenarios with a simple propagation mechanism. In such scenarios the mechanism is assumed to be independent from outside influences, and homogenous across the system. In reality however the mechanism of propagation is likely to be more complex. To capture the effect of such complexity three additional complex propagation mechanisms have been introduced in this study;

- Complex Contagion (Centola and Macy, 2007): Assuming that for reception signals from multiple sources are required

- Costly Contagion: Assuming that actors are constrained in the extent to which they can radiate signals
- Information Overload: Assuming that actors are constrained in the amount of signals they can process (receive)

For each of these mechanisms the same analysis has been performed and outcomes are compared to the normal 'simple' scenario. The results (Tables 6.5, 6.10, 6.11 and 6.12) show that the effect of network structure is different under different mechanism of propagation. For example local clustering while facilitating propagation under simple propagation scenarios hampers propagation when costly mechanism is adopted. Similarly skewness of the degree distribution, directly linked to the extent and presence of hubs, is positively correlated to propagation dynamics under normal, bounded and costly mechanisms, but this correlation is negative under mechanisms of complex contagion. These findings suggest that the effects of network structure are mediated by the mechanism of propagation.

The results of especially the costly contagion (Table 6.11) mechanism indicates that the decomposition into sub-processes is a critical factor when considering the effect of network structure. More specifically, the effect size of all structural characteristics seems to be negatively correlated with the radiation likelihood (Table 6.7).
Also under mechanisms of complex contagion (Table 6.12, Figure 6.5) big difference in effect size of the structural elements are observed. While the cause of these fluctuations are far less obvious, both mechanisms support the notion that the decomposition into subprocess is critical to understand the effect of network structure on propagation dynamics, only when more complex propagation mechanisms are adopted.
In conclusion, the role of actor level information processing in affecting the effect of network structure depend on the scenario which is studied. Similar to the first study (chapter 4) it is found that in scenarios in which a simple independent propagation mechanism is adopted, the information processing on the actor level has no impact on the effects of network structure. However, in scenarios with more complex propagation mechanisms the actor level information processing will play a critical role. In such scenarios the network structure no longer has a monolithic and constant effect. Instead, the effects of network structure will vary across different mechanisms of propagation. And the effect of network structure will strongly depend on the decomposition of the mechanism in three sub-processes of propagation, making the information processing behavior on the actor level a prerequisite for studying the effect of network structure in such scenarios.

7.4 Reflection

This dissertation proposes a different view on the propagation process; the information processing view of propagation. By considering the process, rather than the outcomes of this process this view emphasizes the mechanism by which propagation takes place. The argument for doing so is that differences in this mechanism are likely to affect the propagation dynamics locally and result in heterogeneity which can yield significant fluctuation of the propagation dynamics on the system level. Understanding the system-wide propagation dynamics therefore requires knowledge on the actor level mechanism.

The information processing view of propagation considers propagation as a process by which information regarding changes in the state/behavior are communicated among actors. Building on the traditional model of communication (Shannon, 1948), which has identified three prerequisites for communication: A sender, a channel and a receiver, the information processing view considers the mechanism of propagation to consist of three distinct information processing sub-processes: Radiation, Transmission and Reception (RTR).

In this dissertation it has been shown that the decomposition of the propagation mechanism into these sub-processes does not always have an significant impact on the propagation dynamics on the system level. In simple scenarios in which the mechanism is assumed to be independent from outside influences, and is the same across all ties, adding a detailed description on the mechanism of propagation has no additional value for understanding the propagation dynamics. Traditional models for propagation, be it threshold models (Kleinberg, 2007), Bass-like models (Bass, 1969) or SIS/SIR mean field models (Pastor-Satorras et al., 2014) have leveraged this homogeneity and consequently provided key insights in the dynamics of propagation in under these assumptions.

It is however questionable to which extent the underlying assumptions in these models are realistic. Especially in social systems actors are a representation of social entities with characteristics that are seldom homogenous. Resulting in (propagation) behavior that is likely to vary locally. Traditional mean-field models have difficulty incorporating such local heterogeneity, as their methodology builds on leveraging homogeneity assumptions within compartments (Rahmandad and Sterman, 2008; Pastor-Satorras et al., 2014). Agent-based Models, in contrast, are particularly well suited to capture such local variations, and therefore an agent-based model based on the information processing view, the RTR-model, has been developed in this dissertation. This model has consequently been used to explore the dynamics of propagation in various heterogeneous settings.

The numerous simulations with this RTR-model of propagation suggest that when hetero-

geneity in the actor level information processing is introduced, the decomposition into the three sub-processes of propagation becomes a prerequisite for understanding the system-wide dynamics. This holds in scenarios in which this heterogeneity stems from a more complex mechanism of propagation (as studied in chapter 6) or from variations in actor characteristics (as studied in chapter 5).

Based on the research in this dissertation it remains very difficult to explicitly specify what the effect of actor level information processing behavior is. Also the specifying the impact of a specific decomposition of the mechanism of propagation remains hard. As such effects are conditional on the mechanism, the network structure and the extent of heterogeneity in the scenario, making meta level claims in this area difficult by design. Often the findings are highly scenario specific, making generalization difficult.
However, what consistently has been shown is that the mechanism of propagation is a critical from understanding the propagation dynamics on the system level in scenarios in which some form of heterogeneity is present. The information processing behavior on the actor level can have a moderating effect on the effect of network structure and heterogeneity in actor behavior, and therefore the impact of these drivers of propagation dynamics should not be considered without taking into account the mechanism of propagation.

7.4.1 The role of network structure

The network structure has been widely accepted to be a driver of propagation dynamics. As the network structure provides the infrastructure on which propagation takes place it constrains the propagation dynamics both on the local as well as on the system level. As most network structures observed in practice are heterogeneous, this has pushed scholars in many fields to study the effects of network structure on propagation dynamics. The result; a rich body of work considering the impact of various structural characteristics on propagation dynamics.
While this body of work has greatly increased our understanding of the role of heterogeneity in network structure in the propagation process, it has been based on a fairly simple notion of propagation. This notion describes the local dynamics of propagation using a single parameter, rather that the mechanism which has lead to such dynamics. When focusing on the description of the propagation process, it is shown that often the process describes what is in fact only a single sub-process of propagation, propagation is adoption (the reception sub-process) or sending signals (the radiation sub-process). This assumes

that this single sub-process is representative of the remaining sub-processes, effectively assuming that all sub-process are exactly the same. This covers only a very constrained special case of the propagation mechanism.

Adopting a version of the RTR-model which matches this special case has yielded support for the structural effect of scale-free (Barabási et al., 1999) and small world (Watts and Strogatz, 1998) network topologies on propagation. Also notions regarding clustering from (Rahmandad and Sterman, 2008; Girvan and Newman, 2002) found in existing literature are corroborated by the findings. Therefore the results seem in line with existing theory on the impact of network structure.
Decomposing the mechanism of propagation into three sub-processes however allows for a broader range of mechanisms to be studied. By extending the scope of propagation mechanisms, it has been shown that the finding in this special case do not generalize to all scenarios. It has been found that in scenarios with simple mechanisms the network structure has a constant and uniform effect. When moving beyond this subset and considering more complex mechanisms, the effect of network structure becomes very different. In some scenarios the presence of hubs seems to have a constraining effect on propagation dynamics rather than to facilitate it, and in others the positive effect of long jumps becomes a constraint rather than a virtue (Centola and Macy, 2007). This indicates that the effect of network structure is not unique, as has been assumed in previous literature, and that a clear set of boundary conditions applies to our knowledge on the effects of network structure.
This dissertation indicates that the mechanism of propagation, and the consequent decomposition of the propagation process into actor level information processing behavior, plays a important role in moderating the effects of network structure. As such this actor level behavior should be considered as one of the boundary conditions on the impact of network structure.

7.4.2 The role of heterogeneous actor behavior

It is widely recognized that actors are likely to be heterogeneous. Especially in social system the assumption of homogeneity makes little sense, as social entities never have exactly the same characteristics. Such variance in characteristics is likely to result in differences in behavior. This not only yields differences in the way people interact, resulting in heterogeneous network structures, but also in differences in the way they cope with

signals, which translates into heterogeneous information processing.

This heterogeneity has been recognized before, resulting in a limited body of work studying the impact of heterogeneity in actor behavior on the propagation dynamics (e.g. (Goldenberg et al., 2001; Rahmandad and Sterman, 2008; Young, 2009; Jackson and Lopez-Pintado, 2013)). This body of work has however presented fluctuating results; for example Rahmandad and Sterman (2008) find that there is little impact of heterogeneity in actor behavior, whereas Jackson and Lopez-Pintado (2013) does find a significant effect of heterogeneity. The decomposing the mechanism of propagation and specifying the location of heterogeneity —the sub-process in the mechanism which is affected by it— can explain these seemingly conflicting results.
Both studies exemplified above have adopted a relatively simple notion of propagation. Consequently it is not directly obvious which type of heterogeneity is observed in either study. A closer look at these papers reveals that the work of Rahmandad and Sterman (2008) considers *'the rate at which an infected individual generates new cases'*, which can be best described as a radiation sub-process. Whereas the work of Jackson and Lopez-Pintado (2013) considers a process which describes *'the proclivity to adopt'*, which is can be interpreted as a reception sub-process.
In this dissertation it has been shown that heterogeneity in different sub-process has a very different mechanism by which it has an effect on propagation dynamics. It shows that heterogeneity in radiation primarily affects the chance of success and not prevalence, which explains why Rahmandad and Sterman (2008) find little effect of propagation while considering the eventual infection size. In contrast it has been observed that heterogeneity in reception primarily affects prevalence, which explains why Jackson and Lopez-Pintado (2013) do find a strong effect of heterogeneity. Distinguishing between the type of heterogeneity which is considered, which is facilitated by adopting a nuanced look at the propagation mechanism, allows to combine these seemingly conflicting results into a common framework.

7.4.3 Practical implications

Apart from the methodological and conceptual contributions discussed above, this dissertation also provides critical insights for policy makers and practitioners. These policy implications can be linked primarily to intervention strategies. Not only has it been shown (chapter 4) that the mechanism of propagation is a prerequisite of understanding the in-

tervention effectiveness. Also the location or targets of such interventions can be better identified by adopting the actor level information processing view of propagation.
There is a wide body of work on seeding strategies, especially in the fields of marketing and immunology, which primarily focus on the structure of the network to find the optimal seeds (Kempe et al., 2003; Stonedahl et al., 2010). While this provides a good initial step, two things should be realized when adopting such strategies; first, the impact of network structure is not unique, and second, heterogeneity actor behavior can similarly cause variations in local dynamics.

As has been put forward in this dissertation the impact of the network structure can be conditional on the propagation mechanism. In scenarios with complex propagation mechanisms the impact of the network structure cannot be considered without knowing the actor level information processing characteristics, and being able to decompose the mechanism of propagation. While the mechanism of propagation in practice is difficult to measure, especially upfront, decision makers should be aware of the impact the assumptions with regards to the mechanism can have. Incorrect specification of the mechanism can result in adopting seeding strategies which attempt to leverage structural elements which in fact hamper propagation. Therefore more emphasis should be placed on either making realistically assumption with regards to mechanism propagation, or deducing the impact of each sub-process in an early stage in the process.

While most papers that capture heterogeneity in actor behavior point out that heterogeneity results both in actors with high propensity for propagation, and actors with a low propensity for propagation. Especially when clustered, these actors can facilitate a speed-up (or slow-down) of propagation Jackson and Lopez-Pintado (2013). This observation support the existence of what in this dissertation has been dubbed 'the high-way of propagation', a set of connected actors with high propensity to propagate, that serve as hubs for propagation.
A parallel can be drawn with the work of Albert et al. (2000), which has shown that presence of hubs in the structure, makes (scale-free) networks more robust to random attacks but weak against targeted attacks. While Albert et al. (2000) emphasize the effect of hubs in terms of network structure, the same logic can be applied to hubs for propagation. As hub are rare they are unlikely to be randomly seeded, resulting in a negative mean effect of heterogeneity, but once selected they can yield a radical positive effect. By targeted seeding this positive effect could be leveraged.
This suggest that, in networks with heterogeneity in actor behavior, on average (random)

seeding strategies are less effective, while targeted seeding strategies are more effective. If actor behavior is known to policy makers the highway of propagation can be used in such seeding strategies; one can either target the actors presented with hub-like behavior (when propagation is desired) or try to avoid/protect them (if minimizing propagation is the objective).

7.5 Limitations

This research has yielded insights which have increased our understanding of both the mechanism of propagation and the dynamics of propagation processes on the system level. One should however be clear on the limitations of the research in this dissertation. Two limitations are critical to mention for those seeking to build on this research; the results are scenario specific, and the simulation method is computationally intensive.

The simulation method used in this dissertation is both a blessing and a burden. A relatively complex model of propagation has been put forward with a relatively large parameters space. A simulation methodology allows from modeling propagation in a rich variety of scenarios with strict conditions, which allows for such a parameter space to be explored relatively systematically. Using such a multidimensional parameter space does however also imply that the results of any single scenario strongly depend on the the settings used in the scenario. The use of this amount detail makes it difficult to compare finding across scenarios and extrapolate finding towards general knowledge. The strict set of constraints used in a sense hampers the ability for making generalized statements which apply to a wide range of settings.

The second limitation can again be attributed to the methodology, more specifically to the way in which propagation is simulated. Traditional models use a single parameter to describe local dynamics, and assume that this parameter is independent of outside factors. Such models can leverage edge based simulation approaches, which especially in sparse networks yield significant reductions in computation resources and time. The emphasis on the mechanism of propagation in this dissertation however requires an actor based simulation approach which is more computationally intensive. This burden is further increased by the assumption that the mechanism is not an independent dyadic process, but can be affected by other signals in the system. Consequently, interaction among signals is allowed in this method; Effectively meaning that rather than working on a edge

list the simulation approach requires the full network graph, which makes the simulation approach even more computationally intensive. As the network graph scales quadratically with the number of actors in the network, the computational requirements especially in large networks are high, this presents a challenge in the scalability of this method.

7.6 Future research

This dissertation has given merit to the notion of incorporating the mechanism of actor level information processing into our models of propagation. Large parts of the impact of such mechanism have been explored, yet one aspect has been left somewhat unfinished; the seed of infection.

While identified as a driver of propagation dynamics the seed of infection remains only briefly touched upon in the studies in this dissertation. The importance of the seed is distilled from the notion that there will be local differences in the propagation dynamics, which in turn can yield differences in system-wide dynamics. Such local differences can stem from heterogeneity in either actor behavior or network structure. And while the impact of both sources of heterogeneity are explored in this dissertation, it is assumed that seeding takes place randomly, ignoring targeted seeding and the potential impact of seeding strategies. As the impact on seeding strategies might be particularly interesting to policy makers a follow-up study on the effect of the propagation mechanism on the effectiveness of seeding strategies is already being prepared.

Also the methodological side provides opportunity for further exploration. Especially the scalability of the methodology with regards to network size can be improved. Currently the full graph is being used during simulation while propagation is an process which within a single time-step will take place locally. By taking advantages of clustering techniques it should be possible to only use part of the graph for simulation and by doing so reduce the computational resources required. The lessons learned from such clustering, and community detection techniques, would not only improve the speed and scalability of the method, but could also be used for the studying of propagation among coupled networks.

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Nederlandse Samenvatting (Summary in Dutch)

Systemen vertonen vaak gedrag wat moeilijk te voorspellen en sturen is. Interacties op het microniveau (tussen actoren binnen een systeem) leiden tot gedrag dat propageert en dus de dynamiek op systeemniveau benvloedt. Het begrijpen van de effecten van propagatiehet process waarmee actoren elkaar benvloedenis dan ook cruciaal voor het begrijpen van hoe de staat en het gedrag van een systeem zal veranderen.
In de literatuur wordt veelal gekeken naar hoe propagatiedynamiek van een lokaal niveau naar systeemniveau schaalt; hierbij wordt de netwerk structuur aangewezen als voornaamste drijfveer van systeembrede dynamiek. Echter, door de nadruk op structuur te leggen is het het belang mechanisme waarmee propagatie plaatsvindt naar de achtergrond geschoven. In dit proefschrift wordt gesteld dat juist dit mechanisme een belangrijke rol speelt in hoe de dynamiek van propagatie opschaalt van micro- naar het systeemniveau.
Om het mechanisme van propagatie in kaart te brengen wordt in dit proefschrift een raamwerk van propagatie als een informatieverwerkingsproces gentroduceerd. In dit raamwerk bestaat het propagatie mechanisme uit drie seperate sub-processen; het uitzenden van informatie (radiation), het transporteren van informatie (transmission) en het verwerken van informatie (reception).
In dit proefschrift wordt aangetoond dat een dergelijk raamwerk niet alleen leidt tot een meer gedetaileerd en methodologisch sterker model van propagatie, maar ook dat het onderscheiden van de sub-processen noodzakelijk is om effectief te kunnen interveniren in propagatie. Ook wordt aangetoond dat heterogeniteit in de verschillende delen van het propagatie mechanisme een substantieel ander effect heeft op dynamiek op systeemniveau. Dit impliceert dat het specificeren van het mechanisme cruciaal is voor het begrijpen van systeem breed gedrag wanneer actoren heterogeen gedrag vertonen. Bovendien wordt aangetoond dat het effect van de netwerkstructuur op systeembrede dynamiek afhankelijk is van het mechanisme van propagatie. Wanneer meer complexe propagatiemechanismen worden vergeleken, kan een zelfde netwerk structuur in zeer verschillende dynamiek op

systeemniveau resulteren.
Aldoende identificeert dit proefschrift het mechanisme van propagatie als een kritiek element in het begrijpen hoe interactie op microniveau kan resulteren in gedrag op systeemniveau.

Curriculum Vitae

Wilhelmus Hubertus (Wouter) Vermeer was born on August 29 1984 in Rotterdam, the Netherlands. He has studied at the Rotterdam School of Management Erasmus University, where he first has obtained a B.Sc. in Business Administration. Following he has obtained a M.Sc in Business administration focusing on Business Information Management (BIM), which he completed in 2009. After graduating he worked for one year as a research assistant associated with the New Worlds of Work (NWoW) research project, which that year received the 'ERIM impact award'.

In 2010, Wouter started his Ph.D. at the department Decision and Information Sciences at the Rotterdam School of Management, Erasmus University. In 2014, he spend four months as a visiting scholar at the Robert H. Smith School of Business in the University of Maryland. His research interests include network science, diffusion of information, agent-based Modeling and complexity. His research has been linked to the multi-disciplinary complexity group set up by the Netherlands Organisation for Scientific Research (NWO). He has presented his work at various international conferences including the Workshop on Information in Networks, Workshop on Complex Networks, Conference for complexity in Business, International conference on Network Science and the international conference of the network of social network analysis.

Apart from his research activities Wouter has served as a PhD representative at the daily board in his department, and has been heavily involved in teaching. For 6 years he has been involved in the master elective course 'Smart Business Networks' of which the last two years he has been the sole teacher.

Currently, Wouter holds a position as post-doctoral research fellow associated with the Center for Connected Learning (CCL), the Center for Prevention and Implementation and Methodology (Ce-PIM) and the Northwestern Institute for Complexity (NICO) at Northwestern University.

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PROPAGATION IN NETWORKS
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This thesis addresses the analysis of system-wide dynamics of propagation in networks. It argues that an emphasis should be placed on the mechanism by which propagation takes place between actors on the micro level. This mechanism is critical in order to understand system-wide propagation dynamics. This thesis puts forward the information processing view of propagation, a framework in which describes this mechanism using three distinct sub-processes of propagation; Radiation, Transmission and Reception. Decomposing the propagation mechanism into three sub-processes yields a more detailed and methodologically stronger model of propagation which is better suited for capturing the complexity of the propagation processes in practice; the RTR-model of propagation. Agent-based simulations adopting this model show that distinguishing the three sub-process of propagation is critical in order to: 1) understand the effects of interventions in the propagation process, 2) incorporate the heterogeneous behavior of actors, and 3) understand the role of the network structure in propagation.

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