

ABSTRACT

WICKER, ANDREW W. Leveraging Multiple Mechanisms for Information Propagation. (Under the direction of Jon Doyle.)

Extant models of how social influences affect the spread of information across a population of individuals have employed simple conceptions of influence that utilize a single influence mechanism for inducing changes in a population of behaviorally-uniform individuals. We present here a new model of social influence that recognizes and leverages multiple influence mechanisms involving multiple types of relations among individuals and multiple types of individual response to influence. These characteristics of our model provide increased expressivity and extensibility over that of related models and facilitate analysis of influence effects in a variety of social contexts. We support the claimed improvements offered by our model with results pertaining to optimal targeted populations for the influence maximization problem.

Leveraging Multiple Mechanisms for Information Propagation

by
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DEDICATION

To my parents

BIOGRAPHY

Andrew White Wicker was born and raised in Greensboro, North Carolina. Upon receiving a Bachelor of Science degree in computer science with a minor in mathematics from North Carolina State University, he decided to continue in the graduate program to pursue his interests in artificial intelligence.

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TABLE OF CONTENTS

List of Figures	vii
Chapter 1 Introduction	1
1.1 Motivating Example	2
1.2 Plan of Dissertation	6
Chapter 2 Related Work	8
2.1 Voter Models	8
2.2 Random Interacting Networks	10
2.3 Cascade Models	12
2.4 Mathematical Epidemiology	14
2.5 Assessment of the Models	16
Chapter 3 Formalizing Influence Mechanisms	17
3.1 Populations and Mental States	17
3.2 Influence Mechanisms	19
3.3 Mechanism States	20
3.4 Mechanism Relations	22
3.5 Influence Neighborhoods	24
3.6 Markov Transition Probabilities	24
3.7 Examples of Influence Mechanisms	28
3.7.1 Preference Similarity	28
3.7.2 Group Conformity	29
3.7.3 Voter Model Mechanism	30
3.7.4 Authoritarian	32
3.7.5 Contagion	33
Chapter 4 Combining Multiple Influence Mechanisms	35
4.1 Combination Methods	36
4.2 Examples of Combination Methods	38
4.2.1 Random Combination	38
4.2.2 Dictatorial Combination	39
4.2.3 Convex Combination	40
4.3 Behavioral Types	42
Chapter 5 Maximizing Influence	45
5.1 Expected Influence	45
5.2 The Influence Maximization Problem	48

Chapter 6	Population Topology and Targeted Optimality	49
6.1	Connected Populations	50
6.2	Bridges Between Populations	52
6.3	Optimal Targeted Populations	54
6.4	Population Monotonicity	56
6.5	Component Restricted Optimal Targeted Populations	58
6.6	Targeted Population Size	60
6.7	Decomposition of Targeted Populations	63
6.8	Independence of Influence on Selection of Targeted Population	67
Chapter 7	Analysis of Influence Mechanisms	69
7.1	Mechanism Interactions	70
7.1.1	Mechanism Interference	70
7.1.2	Mechanism Monotonicity	71
7.2	Examples of Analysis	73
7.2.1	Authoritarian Mechanism	73
7.2.2	Authoritarian and Contagion Mechanisms	76
7.2.3	Group Conformity and Voter Model Mechanisms	79
7.3	Example Computation	81
Chapter 8	Diminishing Influence	88
8.1	Mechanism Submodularity	90
8.2	Mechanism Orderings	91
8.3	Component Restricted Mechanism Orderings	95
8.4	Local Bias Magnitude	98
8.5	Principal Component Analysis	100
8.6	Violated Squares Approach	102
Chapter 9	Concluding Remarks	103
9.1	Future Work	105
References		108
Appendix		113
Appendix A	Example Matrices	114
A.1	Individual Transition Matrices for Section 7.2.1	114
A.2	Counter-example for Proof of Theorem 22	117

LIST OF FIGURES

Figure 1.1	Graphs of two symmetric binary relations <i>is-coworker-of</i> and <i>is-friend-of</i> over the same set of individuals. Using both relations with one or more influence mechanisms may produce very different results than using only a single relation.	4
Figure 1.2	Depiction of the spread of information across three time steps $t = 1, 2, 3, 4$ through multiple influence relations. The bold directed edges correspond to the direction of the exerted influence at the specified time.	5
Figure 2.1	Comparison of model features, where $1 = [23]$, $2 = [2]$, and $3 = [27]$	16
Figure 7.1	Depiction of a relation over which a group conformity mechanism <i>Conf</i> can exert influence.	79
Figure 7.2	Graphs of r_{Conf} and r_{Voter} representing two different relations over the same set of individuals.	81
Figure 8.1	Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value.	92
Figure 8.2	Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering over a set of positively interfering mechanisms. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity, but not diminishing returns.	94
Figure 8.3	Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering over a set of negatively interfering mechanisms. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity, but not diminishing returns.	94

Figure 8.4 Illustration of one possible shape for the expected influence corresponding to a diminishing mechanism ordering. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity and diminishing returns. . . 95

Chapter 1

Introduction

We address the problem of how social influence affects information propagation in a population. The study of population dynamics and how information flows throughout a population has been an increasingly active area of research. Work in these areas has benefited greatly from the proliferation of digital communication methods, which facilitates construction of social networks built upon different types of relations.

Not surprisingly, the vast amount of work on population dynamics has resulted in a variety of models being used to study information propagation problems. An overarching goal of mathematical modeling, in general, is the creation of a model that is as simple as possible, and many researchers are quick to point out the mathematical convenience of using extant models of information propagation. The formulation of many of these models, however, is not supported by evidence from the social psychology literature. The resulting models inadequately reflect the phenomena being modeled and may lead to inaccurate results.

It is well-established within the field of social psychology that social influence is dependent on the relations that exist between individuals [34]. For example, the mechanism(s) by which a mother influences her child may be very different from the mechanism(s) by which a Twitter

user influences a follower because being someone’s mother is a very different relation than being someone’s Twitter follower. To understand the influence mechanisms, therefore, one must also consider the different relations among individuals.

As with the study of multiple social relations, the effects of influence on individuals in a population when using multiple influence mechanisms has received attention in the social psychology literature [35]. Despite an increasing interest in formal models of information propagation, there is a lack of work that seeks a formal understanding of how multiple mechanisms and multiple relations impact results to well-known problems of social influence. This forms the basis that motivates our work in this dissertation.

We have developed a new model of social influence for studying information propagation in a population. Our model leverages multiple influence mechanisms for transmitting information between individuals and multiple types of relations connecting individuals over which influence mechanisms may exert influence. In this way, our model improves upon existing related models by facilitating analysis of multiple types of influence that is exerted over multiple types of relations. The following example helps highlight such improvements offered by our model.

1.1 Motivating Example

Consider a set $\{x_1, x_2, \dots, x_8\}$ of eight individuals, which we will refer to as a population. Individuals are assumed to occupy one of two states at any given instant. Each of these individuals may be related to another through one of two binary relations: *is-coworker-of* and *is-friend-of*. For this example, assume that coworkers tend to be influenced by an authoritarian influence mechanism (e.g., “accept this because I said so”), whereas friends tend to be influenced by a group conformity influence mechanism since they desire acceptance by their friends (e.g., “all of my friends play musical instruments, so I will too”). We denote influence mechanisms of

these types by *Auth* and *Conf*, respectively.

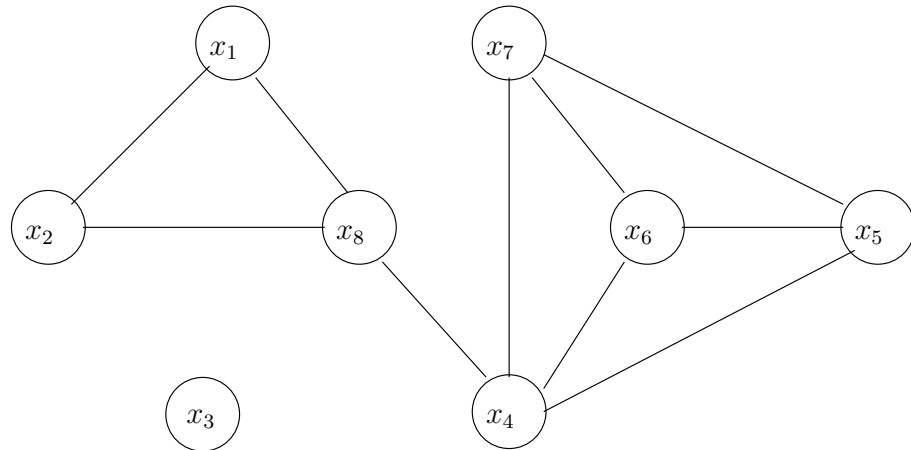
Figure 1.1 graphs the relations *is-coworker-of* and *is-friend-of*. We assume these relations are symmetric, but this may not be the case for other relations. Note that many individuals share both relations, whereas others are related only through a single relation, and the graphs might be disconnected, as is the case with *is-coworker-of*.

If we use only a single influence mechanism, then we are limited to influence only within the single relation corresponding to the mechanism. For example, assume that individuals in $\{x_5, x_6, x_7\}$ are each in the same state and that state is different from the state of the rest of the population. Assume also that those individuals exert influence on x_4 using an authoritarian influence mechanism *Auth*. If these individuals are unable to influence x_4 (e.g., none command sufficient authority), then the remaining subpopulation has no chance of being influenced to adopt the state shared by those in $\{x_5, x_6, x_7\}$.

The use of *Auth* and *Conf* mechanisms together might increase the expectation for influencing the remaining population over what would be obtained using either mechanism alone. For example, individuals x_5 and x_6 may be influenced by x_7 using *Auth* over *is-coworker-of*. Individual x_4 may subsequently be influenced by x_5 , using *Conf* over *is-friend-of*. Next, individuals x_4 and x_7 may influence x_1 , x_8 , and x_3 using *Conf* over *is-friend-of*. Finally, x_8 may influence x_2 using *Auth* over *is-coworker-of* (see depiction in Figure 1.2).

Restriction to a single type of influence mechanism and relation could produce misleading results. Using only the relation *is-coworker-of*, we may conclude that individuals on the cut-edge (x_8, x_4) must necessarily be influenced in order to influence more than half of the population. Moreover, we may conclude that x_1 is unable to exert influence directly on x_7 , or that x_3 is unable to exert influence on any other individual (or be influenced itself) since it is disconnected in *is-coworker-of*. However, the population structure and potential for exerting influence changes with the inclusion of relation *is-friend-of* corresponding to the mechanism

is-coworker-of:



is-friend-of:

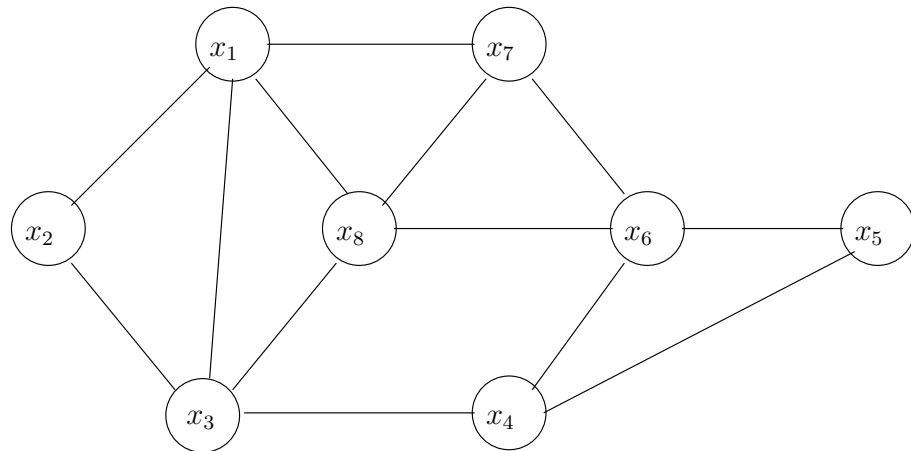
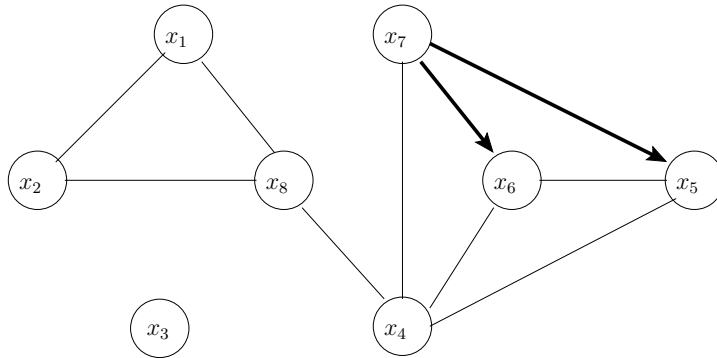
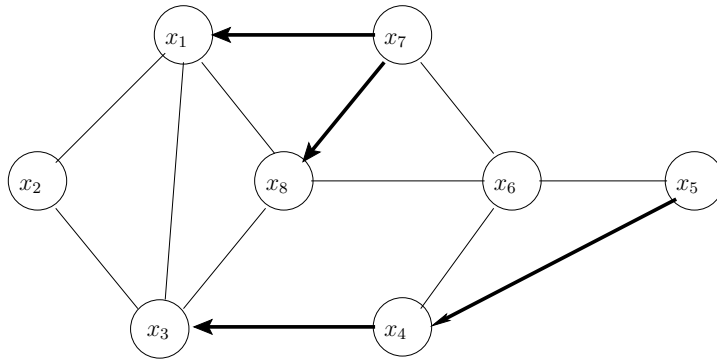


Figure 1.1: Graphs of two symmetric binary relations *is-coworker-of* and *is-friend-of* over the same set of individuals. Using both relations with one or more influence mechanisms may produce very different results than using only a single relation.

$t = 1$:



$t = 2, t = 3$:



$t = 4$:

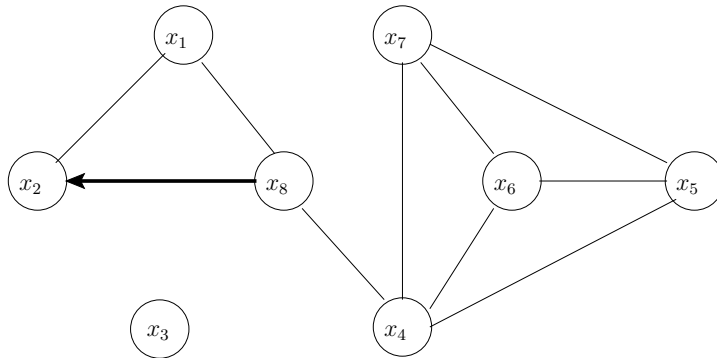


Figure 1.2: Depiction of the spread of information across three time steps $t = 1, 2, 3, 4$ through multiple influence relations. The bold directed edges correspond to the direction of the exerted influence at the specified time.

Conf. With both types of mechanisms and relations, there are numerous potential pathways for propagating information.

This informal example helps demonstrate the impact that multiple types of influence mechanisms and relations could have on the potential for influencing individuals in a population. Marketing campaigns, for example, are increasingly using social media to influence individuals to form positive opinions about new products. Such campaigns could benefit from the dissemination of product information via multiple types of influence mechanisms and social relations. These benefits could be in the form of reduced overhead associated with marketing campaigns by minimizing the number of people they are required to target initially in order to achieve a desired level of influence in a population.

1.2 Plan of Dissertation

We begin in Chapter 2 with an overview of related works. There are many different models that have been used to study information propagation, but we focus on only a few of those that our model builds upon. We show how each of the related models is incapable of capturing a variety of different influences and relations.

We introduce and define influence mechanisms in Chapter 3. These are the objects of primary interest for our work. We give an exposition of the various components that constitute an influence mechanism. We conclude with several examples of types of mechanisms expressed within our model that have been studied in the social influence literature.

Leveraging multiple influence mechanisms for information propagation requires us to make clear assumptions about the ways in which individuals combine the effects of influence from different mechanisms. In Chapter 4, we introduce combination methods, which define how each individual responds to different influences. We show how these combination methods can

be used to characterize behavioral types of individuals.

In Chapter 5, we specify the influence problem to which we apply our model: the influence maximization problem. This problem seeks a given number of individuals in a population that maximize the expected number of individuals that will be influenced in the long-term. Our aim in this dissertation is to study the effects that multiple mechanisms and relations have on potential solutions to the influence maximization problem.

We show in Chapter 6 how the relational structure of a population impacts optimal solutions to the influence maximization problem. We state conditions under which selection of individuals for the influence maximization problem can be decomposed within components of the population. We also state a lower bound on the optimal number of individuals for the influence maximization problem such that the selection of any fewer individuals gives a sub-optimal solution.

In Chapter 7, we give some examples of analysis, along with properties for describing the types of interactions between multiple influence mechanisms. We state properties of some of our example types of influence mechanisms and use the types of interactions between mechanisms to address the question of whether or not inclusion of an additional mechanism will lead to a greater expectation for influence in a population than under only a single mechanism.

We conclude in Chapter 8 with a presentation of some results obtained in our preliminary investigations on diminishing returns when using multiple mechanisms. We show that our measure of information propagation exhibits submodularity with respect to some sets of influence mechanisms. The lack of more general conditions for such submodularity leads us to seek alternative notions of diminishing returns, each of which we show presents difficulties.

Chapter 2

Related Work

Research on influence models is a highly active area of research and an increasingly multi-disciplinary pursuit. The fields of sociology, economics, physics, mathematics, biology, and computer science have each contributed to modeling the process and effects of influence. Despite being conceived out of tangential fields, these various influence models have much in common.

2.1 Voter Models

The general voter model (see [12, 23, 43]) is one of the earliest formal models of influence and is often cited in later developments of influence models [2, 3]. Voter models are closely related to contact processes [33]. The primary appeal of the voter model is the simplicity of its specification and its amenability to analysis.

The voter model was originally introduced as the invasion process in [12]. The invasion process is a model of spatial conflict among competing species. In particular, the goal was to model territory acquisitions over time. The voter model as we present it here was first intro-

duced in [23], independently of [12].

The specification of the general voter model is very simple. A relation between individuals in the population is assumed to induce a regular lattice structure. Each individual (i.e., voter) is assumed to have a binary state (-1 or $+1$), however this can be generalized to non-binary states. At each time step, an individual is chosen at random from the population of individuals. That individual adopts the state of an individual in its neighborhood selected uniformly at random. This process is repeated *ad infinitum* or until a consensus is reached. It has been shown that the voter model with a finite population will converge to a consensus opinion as time approaches infinity [9].

There are many simplifying assumptions that are made in the voter model. The voter model requires that a single random individual adopts the opinion of a random neighbor. The complete adoption of the opinion (or “state”) of a neighbor is only a single specific type of influence mechanism. Although some situations do conform to this all-or-nothing adoption, it does not translate well into other models of influence, such as on preferences or beliefs. For example, an individual may revise its beliefs to become more similar to its neighbor without adopting all of its neighbor’s beliefs.

Another simplifying assumption of the voter model is that individuals are assumed to be related through a single type of relation. There is no acknowledgement of the fact that individuals may share different relations with each other and that each type of relation may result in a different probability of adopting the state of another.

There are variations of the voter model that each attempt to remedy some of the aforementioned shortcomings of the general voter model. Notable among these variations are the *vacillating voter model* [31] and the extension to heterogeneous graphs [43].

The vacillating voter model [31] assumes that individuals have some doubt about their current state. This is captured by the following process. If a randomly selected individual selects

at random one of its neighbors that has the same state, then the individual will change state to that of another randomly selected neighbor. The global effect is that consensus is inhibited, unlike in the finite population general voter model.

The motivation behind this vacillating voter model is that the general voter model implicitly assumes that all individuals have no confidence in their current state. By requiring state transitions to different states, the vacillating voter model explicitly models the lack of confidence by each individual in their current state. No evidence, however, from the social psychology literature is presented in [31] that supports the formulation of this vacillating voter model.

Most work on voter models assumes regular, lattice graphs that represent the relations among individuals in the population. This simplified view is relaxed in [43] by looking at voter model behavior in non-regular, or heterogeneous graphs. The update rule is identical to that used in the general voter model (i.e., a randomly selected voter adopts the state of a randomly selected neighbor). It is shown in [43] that consensus is still reached with the voter model applied to heterogeneous graphs and the time it takes to achieve consensus is decreased by high-degree nodes. The approach of [43] still offers no support from the social psychology literature for the update rule that is used. The decision to use non-regular graphs is, however, supported by the lack of regularity in most social networks.

2.2 Random Interacting Networks

There has been a tremendous amount of work on models that can be categorized as random interacting networks. Such works are prevalent in the field of physics, in which the interactions are between particles. We here focus on one particular model of random interacting networks that our work closely resembles. The model we focus on is Asavathiratham's Influence Model [2, 3].

Asavathiratham’s Influence Model was developed to model the influences among the various entities in a power grid, although it can be applied to other similar domains as well. This model of influence is quite similar to our model in that it uses Markov chains as the underlying probabilistic state transition model, but contains some distinct differences.

Each entity in the Influence Model has a Markov chain that captures the probabilistic transitions over its states (e.g., a power grid transitioning between high/medium/low loads). Each entity may be in one of m states, where the state of each entity i at time k is given by a status vector $s'_i[k] = [0 \dots 010 \dots 0]$ that contains a single 1 representing the current state and 0s everywhere else. Entities are connected in a larger network in which each directed edge is assigned a weight. The directed edge weights correspond to the amount of influence that one entity may exert on another.

The evolution of the population of entities proceeds as follows (see [3]):

1. Entity i selects a neighbor j at random with probability d_{ij} .
2. The present status of j determines the probability $p'_i[k + 1]$ of the next status of i .
3. Entity i transitions to its next status $s'_i[k + 1]$ with probability $p'_i[k + 1]$.

The probabilities d_{ij} given above define the network of connections among entities. This network is denoted by $\Gamma(D')$, where D' is the matrix in which each i, j entry is given by d_{ij} .

The status vector $s'_i[k + 1]$ and next-state probability vectors $p'_i[k + 1]$ for each entity i are combined to form a multi-status vector $s'[k + 1]$ and multi-probability vector $p'[k + 1]$. Thus, $s'[k + 1]$ and $p'[k + 1]$ form matrices in which each i th row corresponds to $s'_i[k + 1]$ and $p'_i[k + 1]$, respectively. Most of the analysis of the Influence Model focuses on the properties and evolution of these matrices.

Analysis of the Influence Model proceeds by first considering the homogeneous case in which all entities have the same set of states. These sets of states are assumed to be binary

for simplicity. Analysis then proceeds by looking at the heterogeneous case in which entities may have different sets of states. These are referred to as the (binary) homogeneous Influence Model and the heterogeneous Influence Model. It is shown for the binary homogeneous model that a consensus is reached whenever $\Gamma(D')$ is ergodic.

The network of connections among the entities captures only a single, semantically vague notion of relation. That is, entities are only viewed as being related or not. There is no notion of different types of relations, except from what is expressed in the edge weights. It is entirely plausible that two relations may form very different network structures, but, in terms of the Influence Model, exert the same amount of influence (i.e., same edge weights). This makes edge weights insufficient for expressing different types of relations.

Only a single influence mechanism is used in the Influence Model. This influence mechanism gives a probability of adopting the state of one's neighbors as a weighted sum of the entities that are in a particular state along with the edge weight connecting the two entities. Such an influence mechanism is very similar to a group conformity type of mechanism that we present in Section 3.7.2 and is similar to the mechanism used in the general voter model.

Although the status-dependent Influence Model described in [2] allows entities to have state transition probabilities (i.e., influence probabilities) that are dependent on their own current state, it does not represent a different influence mechanism. In any case, the status-dependent Influence Model is only briefly developed and not elaborated upon.

2.3 Cascade Models

Cascade models [27, 28, 30, 8, 36, 19, 41] have received a lot of attention recently from those studying information propagation. The simplicity of cascade models makes them amenable to formal analysis. Cascade models come in a variety of forms, but we here focus on what is

referred to as the *independent cascade model*.

The independent cascade model situates homogeneous individuals in a single social network and focuses on the influence maximization problem of selecting a specified number of individuals to target for initial influence such that those targeted individuals influence the largest number of individuals in the long-term. The individuals are in either an active or inactive state, and only active individuals may influence neighboring individuals. An individual is active only for the next time step after they are first influence; after which they are inactive and incapable of exerting influence.

The mechanism by which individuals influence each other is a weighted sum of the number of individuals in an individual's neighborhood that are active. If this weighted sum exceeds some predetermined activation threshold, then that individual is influenced and, thus, active in the next time step.

There are some obvious shortcomings of the independent cascade model as a model of influence and information propagation. To begin with, they do not use multiple influence mechanisms. By using only a weighted sum of active neighbors, these cascade models are limited to a single type of influence mechanism.

Another shortcoming of independent cascade model is their use of simplistic state descriptions, which leads to an all-or-nothing state change similar to voter models. This seems to ignore that people are often not influenced by the entirety of some information. For example, a person may be influenced by another's preference for rock music over classical, but not by their preference for chocolate over vanilla.

2.4 Mathematical Epidemiology

Mathematical models of epidemiology are increasingly being used to study a variety of phenomena that involve diffusion or propagation. The original development of a mathematical epidemiological model [29] has continually evolved in order to better fit the observed data for which the previous models were inaccurate. The refinements made to these models have benefited research on models of influence (see, for example, [52, 13, 6, 53, 47]).

In the following, we give an introduction to the most widely studied formalisms of mathematical epidemiology. We do not explore all of the myriad variations or alternatives of the standard epidemiological models. Instead, we give an introduction to a basic development of these models that is sufficient for understanding their application to models of influence.

Epidemiological models typically characterize a population by compartmentalizing individuals into one of a variety of types, each of which identifies an individual's status with respect to the epidemic being modeled. The most common of these compartmentalizations are S , I , and R . An individual is in compartment S if they are susceptible to infection by the epidemic being modeled. The compartment I contains individuals that are currently infected by the epidemic. Individuals that are recovering (or, sometimes, removed) from the epidemic are in compartment R . A model using these three compartments is referred to as an SIR model, and was first introduced in [29].

The primary concern in analysis of an SIR model are the numbers of individuals in each of the three compartments given as a function of time t . The numbers of individuals that are susceptible, infected, or recovering at time t are given by $S(t)$, $I(t)$, and $R(t)$, respectively. In the simplest of cases, the population size N is assumed to be constant, so that $S(t) + I(t) + R(t) = N$ for each time t . The evolution of the population is described by a set of differential

equations:

$$\frac{dS}{dt} = -\beta IS \quad (2.1)$$

$$\frac{dI}{dt} = \beta IS - \gamma I \quad (2.2)$$

$$\frac{dR}{dt} = \gamma I \quad (2.3)$$

where β is the contact rate constant and γ is the recovery rate constant. The last equation can be written as $R(t) = N - S(t) - I(t)$ since the population size is assumed to be constant.

It is outside the scope of this dissertation to describe in detail all of the variations of the basic SIR model. Parameters such as noise, birth rates, time-dependent birth rates, and time-dependent contact rates result in models that better fit historical data on epidemics (e.g., plague, measles, influenza). We can also reformulate the basic SIR model by considering different sequences of status evolution, such as SIS and SIRS models.

Other works on modeling influence in a population have adopted epidemiological models as approximations for the spread of information guided by influence effects.

The work presented in [53] applies epidemiological models to a computer security problem by studying information propagation in a network. The main idea put forth is that standard epidemiological models do not support virus countermeasures to take place before transitions occur between compartments (i.e., S, I, R). A new compartment E is introduced that contains individuals that have been *exposed* to a virus. This new model called e-SEIR is applied to information propagation problems in a network, where the information is a virus. The shortcomings of using only a single relation and using population level functions of compartment transitions prevent this approach from being expressive enough at the individual level for the purposes of our work.

Instead of using the standard epidemic models, [47] uses cellular automata to model epi-

<i>Feature</i>	1	2	3	Our model
multiple influence mechanisms	no	no	no	yes
combined influence mechanisms	no	no	no	yes
multiple relations	no	no	no	yes
behavioral types	no	no	no	yes

Figure 2.1: Comparison of model features, where 1 = [23], 2 = [2], and 3 = [27].

demics. Although not explicitly a model of influence, it does represent something analogous to an influence model. A significant limitation of [47] is the simplification of only considering homogeneous populations with a regular lattice relational structure. Moreover, they use only a single local transition measure, which restricts the general applicability of their model.

2.5 Assessment of the Models

Through our study and critique of the aforementioned related models, we have identified several features that have motivated the development of our influence model. Notable among these features are: 1) multiple influence mechanisms, 2) combined influence mechanisms, 3) multiple relations, and 4) behavioral types. We develop in this dissertation an influence model that supports each of these desirable features. In contrast, the models described in [23], [2], and [27] support none of these features (see Figure 2.1).

Chapter 3

Formalizing Influence Mechanisms

The primary objects of analysis in this dissertation are what we refer to as influence mechanisms. Each influence mechanism expresses a particular type of influence that can be exerted among individuals connected through a corresponding relation. In this chapter, we begin the presentation of our influence model with a formalization of influence mechanisms. We define populations on which influence mechanisms exert their influence and the aspects of an individual's mental attitude that are relevant to the forces exerted by each different influence mechanism. We conclude this chapter with examples of several well-known types of influence that we have formalized within the framework of our model.

3.1 Populations and Mental States

We assume a finite set \mathcal{X} of *individuals*, and call each $X \subseteq \mathcal{X}$ a *population*, with \mathcal{X} constituting the complete population and \emptyset constituting the empty population. We sometimes refer to any $X \subset \mathcal{X}$ as a *subpopulation* in order to distinguish it from the complete population. We assume an enumeration of individuals in the population given by $\langle \mathcal{X} \rangle = \langle x_1, x_2, \dots, x_{|\mathcal{X}|} \rangle$.

In the present treatment, we require that the set of individuals \mathcal{X} does not change and leave consideration of changing populations to future studies.

Each individual $x \in \mathcal{X}$ has associated with it a set Ψ_x of possible *individual mental states*, such that at each instant x can inhabit exactly one mental state in Ψ_x . We assume that the sets of possible mental states for all individuals are the same, that is, $\Psi_x = \Psi_{x'}$ for all $x, x' \in \mathcal{X}$.

We define mental states of populations to be products of the mental states of the individuals they contain. That is, for each $X \subseteq \mathcal{X}$, we define Ψ_X , the set of *population mental states* of X , by the ordered product $\Psi_X = \prod_{x \in \langle X \rangle} \Psi_x$, where we order the factors according to the enumeration of \mathcal{X} . We refer to Ψ_X as *complete* or *global* population mental states. We have $\Psi_{\{x\}} = \Psi_x$ in the case of singleton subpopulations, and for the degenerate population \emptyset , we define $\Psi_\emptyset = \{\emptyset\}$.

We can view each population mental state $\psi \in \Psi_X$ as a function that takes each $x \in \mathcal{X}$ to a mental state $\psi(x) \in \Psi_x$, and takes each population $X \subseteq \mathcal{X}$ to a population mental state $\psi(X) \in \Psi_X$. More generally, for each population $X \subseteq \mathcal{X}$ we can regard each $\psi \in \Psi_X$ as a function that takes each $x \in X$ to a mental state $\psi(x) \in \Psi_x$, and takes each subpopulation $X' \subseteq X$ to a population mental state $\psi(X') \in \Psi_{X'}$. Under this interpretation, if $\psi \in \Psi_X$, then we have $\psi(X) = \psi$ and $\psi(\emptyset) = \emptyset$.

Our treatment of influence mechanisms is based on the probabilistic notions of Markov chains. Use of measures over mental state spaces Ψ_x requires the identification of sets \mathfrak{S}_x of measurable events over each Ψ_x . For finite mental state spaces, every subset of mental states is measurable, that is, $\mathfrak{S}_x = Pwr(\Psi_x)$.

We put these elements of populations with measurable mental state spaces together in the following definition.

Definition 1. A measurable population $(\mathcal{X}, \Psi, \mathfrak{S})$ consists of

- A set of individuals \mathcal{X} ;
- A mapping Ψ associating a set Ψ_x of individual mental states with each $x \in \mathcal{X}$; and
- A mapping \mathfrak{S} associating a measurable space (Ψ_x, \mathfrak{S}_x) with each $x \in \mathcal{X}$.

3.2 Influence Mechanisms

Informally, an influence mechanism is a function that expresses the probabilistic effects of a certain type of influence exerted on a particular individual by others related to that individual in a way characteristic of the mechanism. A similarity metric over some aspects of individual mental states or a measure of group conformity are both types of influence mechanisms that can be used to define the transition probabilities of an individual. We also have that the authoritarian type of influence over the relation *is-coworker-of* from the example in Section 1.1 is an influence mechanism.

Definition 2. An influence mechanism $m = (r_m, \hat{\Psi}_m, \mathfrak{S}_m, \phi_m, \mu_m)$ over a measurable population $(\mathcal{X}, \Psi, \mathfrak{S})$ is characterized by

- A binary mechanism relation r_m over \mathcal{X} . Each mechanism relation r_m induces an influence neighborhood function $\delta_m : \mathcal{X} \rightarrow \text{Pwr}(\mathcal{X})$;
- A set $\hat{\Psi}_m$ of mechanism states and associated set of events \mathfrak{S}_m ;
- A mental state projection mapping ϕ_m that associates a surjective individual mental state projection function $\phi_m^x : \Psi_x \rightarrow \hat{\Psi}_m$ with each $x \in \mathcal{X}$ that takes mental states of x to a unique corresponding mechanism state; and
- A mechanism measure mapping μ_m that associates to each individual x an individual measure assignment function μ_m^x that takes mechanism states $\hat{\psi} \in \phi_m(\Psi_{\delta_m(x)})$ of the

influence neighborhood of x to a transition probability measure $\mu_m^x[\hat{\psi}] : \mathfrak{S}_m \times \mathfrak{S}_m \rightarrow [0, 1]$.

We write \mathcal{M} to denote the finite set of all influence mechanisms under consideration and write $M \subseteq \mathcal{M}$ to denote a subset of mechanisms. We assume an enumeration of influence mechanisms given by $\langle \mathcal{M} \rangle = \langle m_1, m_2, \dots, m_{|\mathcal{M}|} \rangle$.

The preceding definition of a mechanism applies only to the case in which the probabilities of state transitions do not change over time, that is, to time-homogeneous Markov chains. For time-dependent probabilities, the model would need to make mechanism measure mappings functions of time.

A significant benefit of our abstraction of influence mechanisms from transition probabilities is that influence mechanisms, unlike individual transition probabilities, are not restricted to a specific individual or mental state. Different individuals may have different transition probabilities that are defined in terms of the same influence mechanism.

In the discussion that follows, we describe each of the elements that constitute a mechanism. This is followed by several examples of types of influence mechanisms.

3.3 Mechanism States

Many related models of influence regard individuals as inhabiting the same set of states, which can be thought of as simple binary values such as *active* and *inactive* (see [27]). People, however, are known to maintain much more complex mental states. A more natural model of individual states distinguishes beliefs, preferences, and other mental attitudes, and acknowledges that different influence mechanisms might affect change in different aspects of mental states.

We address these concerns by separating the notion of mental states of individuals from those aspects of mental states relevant to a particular influence mechanism. The set $\hat{\Psi}_m$ repre-

sents those properties of individual mental states relevant to mechanism m , and the individual mental state projection functions $\phi_m^x : \Psi_x \rightarrow \hat{\Psi}_m$ identify which individual mental states correspond to which individual mechanism states.

For example, if we want to understand how ice cream preferences change in a population under the forces of social influence, then we could specify the relevant individual mechanism states as being the individual's preferences over ice cream flavors. In this case, if $\psi \in \Psi_x$, we might have $\phi_m^x(\psi) = \textit{chocolate} \succ \textit{vanilla}$, meaning that the individual mental state information in ψ relevant to m is *chocolate* \succ *vanilla*.

In this way, we say that two mental states of individuals are equivalent with respect to a mechanism if they map to the same mechanism state. Formally, we say that $\psi \in \Psi_x$ and $\psi' \in \Psi_{x'}$ are *equivalent with respect to m* , written $\psi \sim_m \psi'$, and call \sim_m the *mechanism state equivalence relation* for m , just in case $\phi_m^x(\psi) = \phi_m^{x'}(\psi')$. Clearly, \sim_m is an equivalence relation on Ψ_x for each $x \in \mathcal{X}$. For each $\psi \in \Psi_x$, we write $[\psi]_m$ to denote the equivalence class in Ψ_x of ψ with respect to m , that is, the set of all individual mental states that map to the mechanism state $\phi_m^x(\psi)$.

For our purposes in this dissertation, we assume that $\phi_m^x = \phi_m^{x'}$ for all $x, x' \in \mathcal{X}$ and $m \in \mathcal{M}$, which follows from our assumption that all individuals maintain the same set of individual mental states. Moreover, we assume that $\phi_m^x = \phi_m^{x'}$ for all $x, x' \in \mathcal{X}$ and $m \in \mathcal{M}$ whenever $\hat{\Psi}_m = \hat{\Psi}_{m'}$. As a special case, we assume $\hat{\Psi}_\emptyset = \{\emptyset\}$. We also assume that $\hat{\Psi}_m$ is finite for each $m \in \mathcal{M}$. Consideration of infinite mechanism state spaces requires additional assumptions to be made with regard to the underlying Markov chains that we use to formulate state transitions (see Section 3.6).

The mental state projection mappings for any mechanism $m \in \mathcal{M}$ are defined for any set of individuals $X \subseteq \mathcal{X}$ by a *population mental state projection* function $\phi_m^X = \prod_{x \in X} \phi_m^x$, where we order the factors according to the enumeration ordering $\langle \mathcal{X} \rangle$. For any mechanism $m \in \mathcal{M}$

and population mental state $\psi \in \Psi_X$, we have the population mechanism state corresponding to ψ given by $\phi_m^X(\psi)$ such that $\phi_m^X(\psi)(x) = \phi_m^x(\psi(x))$ for each $x \in X$. We often write simply $\phi_m(\Psi_X)$ to mean the population mental state projection function ϕ_m^X applied to Ψ_X , where the population X of interest is clear from the argument.

In terms of equivalence classes, we have for any $X \subseteq \mathcal{X}$ and $\psi, \psi' \in \Psi_X$ that $\psi \sim_m \psi'$ if and only if $\psi(x) \sim_m \psi'(x)$ for each $x \in X$. This gives an equivalence class $[\psi]_m = \{\psi' \in \Psi_X \mid \psi \sim_m \psi'\}$.

For most of the remaining discussion, we are concerned only with mechanism states, as opposed to mental states. As such, we will often refer simply to a *state* where the distinction of mental state or mechanism state is either irrelevant to or clear from the context of the discussion.

3.4 Mechanism Relations

Individuals in a population are connected through different social relations, each of which may have associated a different type of influence [34]. Our informal example in Section 1.1 used two different types of influence exerted over different relations. We now consider the relationships among individuals through which individuals exert social influences.

We note that multiple mechanisms may make different uses of the same relation. For example, a hierarchical relation such as the superior/subordinate relation among members of some organization might support one mechanism by which superiors influence subordinates and a different mechanism by which subordinates influence superiors.

Let \mathcal{R} be a finite set of binary relations over \mathcal{X} . We write $r \in \mathcal{R}$ and $R \subseteq \mathcal{R}$ to denote particular relations in and subsets of \mathcal{R} . Although social influences presumably might involve relations of arbitrary arity, we treat only the case of binary relations. This restriction need not

limit the applicability of the theory if one allows expansion of the set of individuals to include new individuals representing tuples of the original individuals. In the present treatment, we assume that relations among individuals do not change, and leave consideration of changing relations to future studies.

We will not distinguish between relations and their graphs or set representations as tuples. In this manner, we say that a relation r is *reflexive* if $(x, x) \in r$ for all $x \in \mathcal{X}$; *irreflexive* if $(x, x) \notin r$ for all $x \in \mathcal{X}$; *symmetric* if $(x', x) \in r$ for all $(x, x') \in r$; *antisymmetric* if $x = x'$ whenever $(x, x') \in r$ and $(x', x) \in r$; *asymmetric* if $(x, x') \in r$ implies $(x', x) \notin r$; and *transitive* if $(x, x'') \in r$ whenever $(x, x') \in r$ and $(x', x'') \in r$.

The *inverse* r^{-1} of a relation r is the relation given by $r^{-1} = \{(x', x) \in \mathcal{X}^2 \mid (x, x') \in r\}$. The *symmetric closure* r° of r is the relation given by $r^\circ = r \cup r^{-1}$. The *transitive closure* r^+ of r is the least relation that contains r and contains (x, x'') whenever it also contains (x, x') and (x', x'') for some $x' \in \mathcal{X}$. We write $r^\oplus = (r^\circ)^+$ for the relation obtained by first symmetrizing r and then taking the transitive closure of the result, which is necessarily both symmetric and transitive. The relation r^\oplus is also conditionally reflexive in the sense that $(x, x) \in r^\oplus$ must hold if $(x, x') \in r^\oplus$ for any individual x' . We write r^* for the full reflexive, symmetric, transitive closure of r .

Unions of relations are again relations. We say that a set $R \subseteq \mathcal{R}$ is reflexive, irreflexive, etc., just in case the relation $\bigcup R$ formed as the union of all the relations in the set is reflexive, etc., respectively.

We write $r[x]$ to denote the set of individuals that the individual x is related to by r , that is, $r[x] = \{x' \in \mathcal{X} \mid (x, x') \in r\}$.

3.5 Influence Neighborhoods

Each mechanism relation r_m determines, for each individual x , a set $\delta_m(x)$ of individuals called the *influence neighborhood* of x . This set consists of those individuals that could potentially exert influence on x using influence mechanism m . We define the influence neighborhood of x under r_m by $\delta_m(x) \stackrel{\text{def}}{=} r_m^{-1}[x]$, that is,

$$\delta_m(x) = \{x' \in \mathcal{X} \mid (x', x) \in r_m\}. \quad (3.1)$$

We extend the notation δ_m to populations X by defining $\delta_m(X) = \bigcup_{x \in X} \delta_m(x)$. Looking back to Figure 1.1 where we assumed $r_{Auth} = \textit{is-coworker-of}$, we have $\delta_{Auth}(x_2) = \{x_1, x_8\}$, $\delta_{Auth}(x_8) = \{x_1, x_2, x_4\}$, and $\delta_{Auth}(\{x_2, x_8\}) = \{x_1, x_2, x_4, x_8\}$.

3.6 Markov Transition Probabilities

An individual can be influenced only by its neighbors, as captured in the mechanism relation and its influence neighborhood function. For each $m \in \mathcal{M}$, $x \in \mathcal{X}$, and $\hat{\psi} \in \phi_m(\Psi_{\delta_m(x)})$, we interpret $\mu_m^x(\hat{\psi})$ as the individual mechanism state transition probabilities resulting from influence exerted through m by the individuals in the influence neighborhood of x in neighborhood mechanism state $\hat{\psi}$.

Our focus on influences exerted on individuals by their neighbors means we assume that if an individual has no neighbors, it does not change individual mechanism state. Formally, we write $\mu_m^x(\emptyset)$ whenever $\delta_m(x) = \emptyset$, and require that

$$\mu_m^x(\emptyset)(\hat{\psi}, \hat{\psi}) = 1 \quad (3.2)$$

for all $m \in \mathcal{M}$, $x \in \mathcal{X}$, and $\hat{\psi} \in \hat{\Psi}_m$. A more general treatment would permit nonzero probabilities of spontaneous change.

Each $m \in \mathcal{M}$ and $x \in \mathcal{X}$ has an associated *individual mechanism state transition probability space* $(\hat{\Psi}_m, \mathfrak{S}_m, \mu_m^x)$ such that $\mu_m^x(\hat{\psi})(\hat{\psi}', \hat{\psi}'')$ gives the probability of x transitioning from individual mechanism state $\hat{\psi}' \in \hat{\Psi}_m$ to $\hat{\psi}'' \in \hat{\Psi}_m$ when its neighbors are in neighborhood mechanism state $\hat{\psi} \in \phi_m(\Psi_{\delta_m(x)})$. For each $m \in \mathcal{M}$, $x \in \mathcal{X}$, $\hat{\psi} \in \phi_m(\Psi_{\delta_m(x)})$, and $\hat{\psi}' \in \hat{\Psi}_m$, we require $\sum_{\hat{\psi}'' \in \hat{\Psi}_m} \mu_m^x(\hat{\psi})(\hat{\psi}', \hat{\psi}'') = 1$. We typically write $\mu_m^x(\hat{\psi})(\hat{\psi}'' | \hat{\psi}') = \mu_m^x(\hat{\psi})(\hat{\psi}', \hat{\psi}'')$ for the probability of x transitioning from $\hat{\psi}'$ to $\hat{\psi}''$.

For each $m \in \mathcal{M}$ and $X' \subseteq \mathcal{X}$ with non-empty $X = \bigcup_{x \in X'} r_m^\oplus[x]$, we obtain a population measure assignment function μ_m^X for the population X that takes each neighborhood mechanism state $\hat{\psi} \in \phi_m(\Psi_{\delta_m(X)})$ to a probability measure $\mu_m^X[\hat{\psi}] : \mathfrak{S}_m^X \times \mathfrak{S}_m^X \rightarrow [0, 1]$ giving state transition probabilities over $\phi_m(\Psi_X)$ as the product probability measure given by

$$\mu_m^X \stackrel{\text{def}}{=} \prod_{x \in X} \mu_m^x, \quad (3.3)$$

where \mathfrak{S}_m^X is taken to be the set of all events over $\phi_m(\Psi_X)$. For the complete population \mathcal{X} , we write μ_m to mean $\mu_m^{\mathcal{X}}$.

When $\delta_m(X) \subseteq X$, we typically omit reference to a neighborhood mechanism state and write simply $\mu_m^X(\hat{\psi}'' | \hat{\psi}')$ for the probability of transitioning from $\hat{\psi}'$ to $\hat{\psi}''$. We introduce this simplified notation since each $\hat{\psi}' \in \phi_m(\Psi_X)$ contains also a neighborhood mechanism state for each $x \in X$ whenever $\delta_m(X) \subseteq X$.

We formulate the mechanism state transitions in terms of Markov chains. Markov chains provide a simple expression of probabilistic state transitions that is adequate for our purposes but that can be extended to express more complex behavior than is considered in our present treatment (e.g., time-dependent transitions, higher-order Markov assumption). Moreover, sev-

eral related works on influence modeling and the diffusion of information have used Markov chains with good results [17, 3, 1].

In the following discussion, we give a description of Markov chains and some of their basic properties as required by our work. We avoid rigorous formal analysis of the Markov chains themselves, as this is readily available in textbooks [25, 37] and would require a substantial departure from the primary focus of this dissertation.

For any $m \in \mathcal{M}$ and $X \subseteq \mathcal{X}$, we write $(\hat{\psi}_m^t(X))_{t \geq 0}$ for the Markov chain induced by the mechanism state transition measure μ_m^X over $\phi_m(\Psi_X)$. We assume each such Markov chain $(\hat{\psi}_m^t(X))_{t \geq 0}$ is of the first-order, that is, each state transition depends only on the current state and not on any previous states. Our focus on mechanisms that express transition probabilities that do not change over time means that we consider only time-homogeneous Markov chains. Our assumption of finite mechanism state spaces places our interest exclusively on finite-state Markov chains.

The state distribution at any time $t \geq 0$ is given by $\pi_m^t(X)$, where $\pi_m^t(X)(\hat{\psi})$ gives the probability of being in state $\hat{\psi} \in \phi_m(\Psi_X)$ at time t . An initial state distribution over $\phi_m(\Psi_X)$ is denoted by $\pi_m^0(X)$. We typically write $(\hat{\psi}_m^t)_{t \geq 0}$ to mean $(\hat{\psi}_m^t(\mathcal{X}))_{t \geq 0}$ and π_m^t to mean $\pi_m^t(\mathcal{X})$.

A state $\hat{\psi}'$ is *reachable* from state $\hat{\psi}$ (written $\hat{\psi} \rightarrow \hat{\psi}'$) in a Markov chain $(\hat{\psi}_m^t)_{t \geq 0}$ if $\mu_m(\hat{\psi}^{t+n} \mid \hat{\psi}^{t-1}) > 0$, for some time $t \geq 0$ and $n \geq 0$. Two states $\hat{\psi}$ and $\hat{\psi}'$ are said to *communicate* (written $\hat{\psi} \leftrightarrow \hat{\psi}'$) if $\hat{\psi} \rightarrow \hat{\psi}'$ and $\hat{\psi}' \rightarrow \hat{\psi}$. That is, two states communicate if they are reachable from each other. A set $Com \subseteq \phi_m(\Psi_X)$ is a *communicating class* if all states $\hat{\psi} \in Com$ communicate with each other and they do not communicate with any state $\hat{\psi}' \notin Com$. A Markov chain is called *irreducible* if all states are in the same communicating class.

A state $\hat{\psi}$ is *periodic with period k* if k is the greatest common divisor of the recurrence periods $\{n > 0 \mid \mu_m(\hat{\psi}^{t+n} \mid \hat{\psi}^t) > 0\}$. That is, returning to state $\hat{\psi}$ must occur in time multiples

of k . If $k = 1$ for state $\hat{\psi}$, then $\hat{\psi}$ is said to be *aperiodic*. A Markov chain is called aperiodic if all states are aperiodic.

A *transient* state is one for which there is a non-zero probability that it will never be revisited. A *recurrent* state is one that is not transient. The states in a communicating class Com are either all transient or all recurrent. A finite-state Markov chain has at least one recurrent state, so all states in a finite-state, irreducible Markov chain are recurrent.

Central to our subsequent discussions is the notion of a stationary distribution of a Markov chain. We give the following explanation about the usage of stationary distributions for our purposes and refer the reader to [32] for a more comprehensive exposition.

For any finite-state, time-homogeneous, irreducible Markov chain, there exists a unique stationary distribution. Moreover, the long-term behavior of such a Markov chain is independent of the initial state distribution. In general, we can not assume irreducibility of the Markov chain induced by an influence mechanism transition probability measure and, therefore, must consider the case of reducible Markov chains. For any finite-state, time-homogeneous, reducible Markov chain, there may exist more than one stationary distribution.

To see this, assume any $m \in \mathcal{M}$ and let $(\hat{\psi}_m^t)_{t \geq 0}$ denote the Markov chain induced by m over $\phi_m(\Psi_{\mathcal{X}})$ and let Com be the set of all communicating classes of $(\hat{\psi}_m^t)_{t \geq 0}$. The states in each communicating class are either all recurrent or all transient, which we denote by $Com^r \subseteq Com$ and $Com^t \subseteq Com$, respectively. Every state in each $Com \in Com^t$ will eventually transition to a state in some $Com' \in Com^r$ and remain in that recurrent communicating class. The long-term behavior of a Markov chain starting in a transient state, thus, conforms to that of states in recurrent communicating classes.

For the remainder of our discussions, we do not require specification of the sets of communicating classes. As such, we write simply $\pi_m(X)$ for a stationary distribution of the Markov chain induced by $m \in \mathcal{M}$ over $\phi_m(\Psi_X)$. We write π_m to mean $\pi_m(\mathcal{X})$.

3.7 Examples of Influence Mechanisms

We now present examples of how one might use our formalization of an influence mechanisms to express types of influences that have been studied in various forms in the social psychology literature. For each of the following example mechanisms, we define only the transition probability measure. The transition probability measures of some mechanisms characterize the influence exerted across a specific relation, but the transition probability measures of other commonly studied mechanisms can be applied to a variety of relations.

For example, the transition probability measure for a group conformity influence mechanism that we define in one of the following examples might be applied to the *is-friend-of* or the *is-coworker-of* relation, as well as others. The choice of relation for a group conformity mechanism is, therefore, dependent on the context of the problem being analyzed. This is in contrast to a mother's influence that may be defined, for example, such that it applies only to the relation *is-family-member-of*.

3.7.1 Preference Similarity

Existing work has shown that people have a tendency to adopt preferences of others when the preferences held by the others are similar to those of the person being influenced [45]. For example, if an individual x is deciding where to vacation and individual x' has similar preferences on vacation spots, then x is likely to adopt a new vacation spot for which x' has formed a preference but x has not (see [20] for a related treatment that uses belief similarity).

One can capture an influence mechanism with this character by regarding preference orders over alternatives as the states of the mechanism, with the mental state projection mapping identifying the preferences of each individual in the common terms. One then identifies a similarity measure over preference orders, and defines a transition measure in which the probability that

one individual will adopt the preferences of another is in terms of the similarity between the preferences of the individual and the preferences of the other.

We write s to denote a metric on the set of preferences. See, for example, [48, 49, 50, 51] for expositions of both traditional and improved similarity metrics over preferences. We write $Sim(s)$ to denote a *preference similarity influence mechanism* based on s , for population mechanism states $\hat{\psi}, \hat{\psi}' \in \phi_{Sim(s)}(\Psi_{\mathcal{X}})$ representing the preference information relevant to $Sim(s)$ as:

$$\mu_{Sim(s)}^x(\hat{\psi})(\hat{\psi}'(x) | \hat{\psi}(x)) \stackrel{\text{def}}{=} \begin{cases} \alpha^x \cdot s(\hat{\psi}(x), \hat{\psi}'(x)) & \text{if } \hat{\psi}'(x) = \hat{\psi}(x') \text{ for some } x' \in \delta_{Sim(s)}(x), \\ 0 & \text{otherwise,} \end{cases} \quad (3.4)$$

where α^x ensures that $\sum_{\hat{\psi}'(x) \in \hat{\Psi}_{Sim(s)}} \mu_{Sim(s)}^x(\hat{\psi})(\hat{\psi}'(x) | \hat{\psi}(x)) = 1$ for any $\hat{\psi} \in \phi_{Sim(s)}(\Psi_{\mathcal{X}})$.

3.7.2 Group Conformity

Individuals have a tendency to become similar to their peers when a sufficiently large number of their peers have equivalent states with respect to some relevant notion of state equivalence [16, 4, 5]. Moreover, conformity with a group preference increases as the group increases in size. For example, if an individual's peer group all have equivalent preferences for a certain type of music, then that individual is more likely to adopt that music preference than if the group consisted of only one individual.

The group conformity effect of influence has been shown to plateau at around five group members [46]. We now define one form of a group conformity influence mechanism motivated by such work, which discounts the magnitude of influence by the group size such that we achieve a plateau roughly at group sizes of 5. We also define a non-discounted group conformity mechanism under which the probability of influence is a simple fraction of the group size of

individuals that maintain a common state.

We write *Conf* to denote the *non-discounted group conformity influence mechanism*. For all $x \in \mathcal{X}$ and $\hat{\psi}, \hat{\psi}' \in \phi_{\text{Conf}}(\Psi_{\mathcal{X}})$, we define μ_{Conf}^x as:

$$\mu_{\text{Conf}}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \frac{|\{x' \in \delta_{\text{Conf}}(x) \mid \hat{\psi}(x') = \hat{\psi}'(x)\}|}{|\delta_{\text{Conf}}(x)|}. \quad (3.5)$$

Let *DConf* denote the *discounted group conformity influence mechanism*. Assume we have a set of n individuals that might exert influence on x and each have *DConf* mechanism states equivalent to each other. Such a set is given for $\hat{\psi}, \hat{\psi}' \in \phi_{\text{DConf}}(\Psi_{\mathcal{X}})$ by $\iota(x, \hat{\psi}, \hat{\psi}') = \{x' \in \delta_{\text{DConf}}(x) \mid \hat{\psi}(x') = \hat{\psi}'(x)\}$. For all $x \in \mathcal{X}$ and $\hat{\psi}, \hat{\psi}' \in \phi_{\text{DConf}}(\Psi_{\mathcal{X}})$, we define μ_{DConf}^x as:

$$\mu_{\text{DConf}}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \beta^x \frac{1}{1 + 10 \exp^{-|\iota(x, \hat{\psi}, \hat{\psi}')|}}, \quad (3.6)$$

where β^x is a normalizing constant so that $\sum_{\hat{\psi}'(x) \in \hat{\Psi}_{\text{DConf}}} \mu_{\text{DConf}}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) = 1$ for any $\hat{\psi} \in \phi_{\text{DConf}}(\Psi_{\mathcal{X}})$. The factor of 10 in the denominator is used to achieve a plateau at the $n = 5$ value reported in the social psychology literature [46].

In our subsequent discussions, we typically refer to *Conf* as a *group conformity mechanism* and maintain the discounted distinction when referring to *DConf*.

3.7.3 Voter Model Mechanism

We present here a formalization of a mechanism motivated by the voter model. Our formalization does not exhibit the exact same behavior as the voter model, but does still provide an interesting mechanism motivated by the work on voter models. The primary difference between the mechanism we present here and the voter model is that the voter model uses asynchronous transitions (i.e., only one individual changes state at each instant), whereas we require every

individual to assess a state change at each instant.

We write *Voter* to denote a *voter model mechanism*. In order to simplify comparisons with other types of mechanisms, we assume a binary mechanism state space given by $\hat{\Psi}_{\text{Voter}} = \{0, 1\}$. Recall that with the voter model, at each instant an individual is selected to transition to the state of a randomly selected neighbor. We capture a related synchronous process by assuming that each individual $x \in \mathcal{X}$ maintains a *neighborhood weight distribution* given by $p_x : \delta_{\text{Voter}}(x) \rightarrow [0, 1]$, where $\sum_{x' \in \delta_{\text{Voter}}(x)} p_x(x') = 1$. At each instant, $p_x(x')$ gives the probability that x will adopt the individual mechanism state of $x' \in \delta_{\text{Voter}}(x)$.

For all $x \in \mathcal{X}$ and $\hat{\psi}, \hat{\psi}' \in \phi_{\text{Voter}}(\Psi_{\mathcal{X}})$, we define μ_{Voter}^x as:

$$\mu_{\text{Voter}}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \sum_{x' \in S} p_x(x'), \quad (3.7)$$

where $S = \{x' \in \delta_{\text{Voter}}(x) \mid \hat{\psi}(x') = \hat{\psi}'(x)\}$ is the set of all neighboring individuals that are in the individual mechanism state $\hat{\psi}'(x)$.

Although group conformity influence has received a tremendous amount of attention (however, informally) in the social psychology literature, it is the voter model and other types of contact processes that have been of more interest in the fields of computer science, mathematics, and physics. We can show that the influence exerted by a group conformity mechanism *Conf* is equal to that of a voter model mechanism *Voter* under uniform neighborhood distributions p_x .

Theorem 1. *If p_x is uniform for all $x \in \mathcal{X}$, $r_{\text{Voter}} = r_{\text{Conf}}$, and $\hat{\Psi}_{\text{Voter}} = \hat{\Psi}_{\text{Conf}}$, then $\mu_{\text{Voter}} = \mu_{\text{Conf}}$.*

Proof. Assume $r_{\text{Voter}} = r_{\text{Conf}}$, $\hat{\Psi}_{\text{Voter}} = \hat{\Psi}_{\text{Conf}}$, and $p_x(\cdot) = \frac{1}{|\delta_{\text{Voter}}(x)|}$ for all $x \in \mathcal{X}$. For all $\hat{\psi}, \hat{\psi}' \in \phi_{\text{Voter}}(\Psi_{\mathcal{X}})$ and $x \in \mathcal{X}$, we have $\mu_{\text{Voter}}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) = \sum_{x' \in S} \frac{1}{|\delta_{\text{Voter}}(x)|} = \frac{|S|}{|\delta_{\text{Voter}}(x)|}$,

where the set S as defined as for Equation 3.7. This is precisely the definition for μ_{Conf}^x given in Equation 3.5. It follows from Equation 3.3 that $\mu_{Voter} = \mu_{Conf}$. \square

3.7.4 Authoritarian

It is not uncommon for individuals to be susceptible to influence simply due to the authoritative status of some other individual. The authoritarian influence mechanism used for the example in Section 1.1 is one particular form of such a mechanism, where we have a mechanism relation *is-coworker-of*, binary mechanism states, and a transition measure as defined in the following Equation 3.8. We now give a formal definition of the influence exerted by an authoritarian influence mechanism.

An authoritarian type of influence requires assignment of a level of authority to each individual. We assume a total strict order \succ^a over \mathcal{X} defined such that $x \succ^a x'$ if and only if x is more authoritative than x' .

An *authoritarian influence mechanism*, denoted by $Auth$, is defined such that an individual x is influenced directly by another individual x' if $x' \in \delta_{Auth}(x)$ and $x' \succ^a x$. We define μ_{Auth}^x for any $x \in \mathcal{X}$ and $\hat{\psi}, \hat{\psi}' \in \phi_{Auth}(\Psi_{\mathcal{X}})$:

$$\mu_{Auth}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \begin{cases} 1 & \text{if } \hat{\psi}'(x) = \hat{\psi}(x'), x' \succ^a x, \text{ and} \\ & x' = \max_{\succ^a}(x'' \in \delta_{Auth}(x)), \\ 0 & \text{otherwise.} \end{cases} \quad (3.8)$$

Our definition for an authoritarian mechanism follows from our motivating example in Section 1.1. We assume that the authority ordering \succ^a is consistent with the enumeration $\langle \mathcal{X} \rangle$, that is, $x_i \succ^a x_j$ if and only if $i > j$. This suits our purposes in this dissertation, but we recognize the usefulness of an authority ordering that is a total preorder. In that case, an authoritarian

mechanism would permit individuals with the same level of authority, as might exist in a hierarchical organization such as a corporation.

3.7.5 Contagion

The study of information propagation is analogous to that of disease transmission within mathematical epidemiology. We do not approach information propagation in the same manner as compartmentalized models (e.g., SIRS models), but our view of the study is closely related to the use of contact processes in mathematical epidemiology. In this way, we define the susceptibilities to infection in terms of individual transition measures.

We give a definition for the very simplistic *contagion influence mechanism*, denoted by $Cont$, that captures the spread of infections of a highly infectious disease. For simplicity, we assume that individuals are infected at first contact and we avoid the numerous parameters incorporated into many mathematical epidemiology models (e.g., recovery rates).

We define μ_{Cont}^x for each $x \in \mathcal{X}$, $\hat{\psi}, \hat{\psi}' \in \phi_{Cont}(\Psi_{\mathcal{X}})$, and $\hat{\psi}'' \in \hat{\Psi}_{Cont}$ as:

$$\mu_{Cont}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \begin{cases} 1 & \text{if there exists } x' \in \delta_{Cont}(x) \text{ such that } \hat{\psi}(x') = \hat{\psi}'(x) = \hat{\psi}'', \\ 1 & \text{if } \hat{\psi}(x) = \hat{\psi}'(x) \text{ and there does not exist } x' \in \delta_{Cont}(x) \\ & \text{such that } \hat{\psi}(x') = \hat{\psi}'', \\ 0 & \text{otherwise.} \end{cases} \quad (3.9)$$

where $\hat{\Psi}_{Cont} = \{0, 1\}$ and $\hat{\psi}''$ is taken to be the state representing infection by the contagious disease, or what we will later refer to as the influence information.

Our definition of the contagion mechanism assumes that infection happens with certainty when at least one neighboring individual is itself infected. Of course, this could be relaxed by parameterizing the definition with a value that gives the susceptibility to infection. For example,

we could have that μ_{Cont} infects individuals with certainty under some relations, but has a low probability of infection under other relations.

Chapter 4

Combining Multiple Influence

Mechanisms

One of the advantages of extant influence models that use a single influence mechanism comes from the simplicity of specifying how each individual is influenced. Such models assume that all individuals are susceptible to influence through the same influence mechanism. This assumption, however, is inconsistent with an abundance of empirical evidence in social psychology and related fields suggesting that different individuals respond to influence in different manners (see [10, 7]). Although one may argue that a single influence mechanism is sufficient for simplified problem domains, it is insufficient for modeling influence effects among individuals that may have different responses to the same influence mechanism.

Just as different physical materials (e.g., wood, steel) can have different levels of susceptibility to different forces acting upon them (e.g., magnetic forces), so too can different individuals have different levels of susceptibility to different influence mechanisms (see [17, 7]). Consideration of multiple influence mechanisms that may simultaneously exert influence on an individual requires a method for combining the aggregate effects into a single measure of

influence acting on the aspects of that individual's mental attitude that are relevant to each of the mechanisms.

In this chapter, we give a formal definition of a combination method, which specifies how influence effects of multiple influence mechanisms are combined. Different individuals are permitted to use different combination methods, and we interpret each combination method as characterizing the behavioral type of individuals. We give a few examples of combination methods.

4.1 Combination Methods

Consider the influence mechanisms $M = \{m_1, m_2, m_3\}$ and assume that influence is exerted on individuals in a population using some combination of influence mechanisms in M . Each individual may combine the influence exerted through these different influence mechanisms in different ways to produce different behavior in the presence of influence. For example, one individual may be influenced only by m_2 , whereas another individual may be influenced by a non-zero positive weighted average of each influence mechanism in M . We refer to these different methods for combining influence mechanisms as a combination method.

Definition 3. A mechanism combination method is a function c that takes a set of mechanisms $M \subseteq \mathcal{M}$ over a measurable population $(\mathcal{X}, \Psi, \mathfrak{S})$ to a combined mechanism $c(M) = (r_{c(M)}, \hat{\Psi}_{c(M)}, \mathfrak{S}_{c(M)}, \phi_{c(M)}, \mu_{c(M)})$ over $(\mathcal{X}, \Psi, \mathfrak{S})$ and is characterized by

- The combined mechanism relation $r_{c(M)} = \bigcup_{m \in M} r_m$, which induces a combined influence neighborhood function $\delta_{c(M)} : \mathcal{X} \rightarrow \text{Pwr}(\mathcal{X})$;
- The set $\hat{\Psi}_{c(M)} = \prod_{\hat{\psi} \in \{\hat{\psi}_m | m \in M\}} \hat{\Psi}$ of combined mechanism states, where the order of the product is consistent with the enumeration $\langle \mathcal{M} \rangle$, and associated set of events $\mathfrak{S}_{c(M)}$;

- A combined mental state projection mapping $\phi_{c(M)}$ that takes each $x \in \mathcal{X}$ to a surjective function $\phi_{c(M)}^x : \Psi_x \rightarrow \hat{\Psi}_{c(M)}$; and
- A combined mechanism measure mapping $\mu_{c(M)}$ that associates to each individual x an individual measure assignment function $\mu_{c(M)}^x$ that takes combined mechanism states $\hat{\psi} \in \phi_{c(M)}(\Psi_{\delta_{c(M)}(x)})$ of the combined influence neighborhood of x to a transition probability measure $\mu_{c(M)}^x[\hat{\psi}] : \mathfrak{S}_{c(M)} \times \mathfrak{S}_{c(M)} \rightarrow [0, 1]$.

We denote the set of all combination methods by \mathcal{C} .

By defining the combined mechanism states as we have, we eliminate duplication and inconsistencies when combining mechanisms that have the same mechanism state space. For example, assume $M = \{m_1, m_2\}$ and $\hat{\Psi}_{m_1} = \hat{\Psi}_{m_2} = \{0, 1\}$. We have $\bigcup_{m \in M} \hat{\Psi}_m = \{0, 1\}$. Taking the product gives $\hat{\Psi}_{c(M)} = \{0, 1\}$, which is precisely the state space of each individual mechanism. If we used the product space over each set of mechanism states to get a combined mechanism state space of $\{00, 01, 10, 11\}$, then we would have inconsistent states (e.g., 01 means an individual is simultaneously in mechanism state 0 and 1) and duplication of state information (e.g., 11). Of course, the inclusion of mechanisms that have different mechanism state spaces will result in combined mechanism states that include each different mechanism's states.

It follows from $r_{c(M)}$ that $\delta_{c(M)}(x) = \bigcup_{m \in M} \delta_m(x)$ for all $x \in \mathcal{X}$ and $M \subseteq \mathcal{M}$. For the combined influence neighborhoods from Figure 1.1 with $M = \{Auth, Conf\}$ and $X = \{x_3, x_5\}$, we have $\delta_{c(M)}(x_3) = \delta_{Auth}(x_3) \cup \delta_{Conf}(x_3) = \{x_1, x_2, x_4, x_8\}$ and $\delta_{c(M)}(X) = \delta_{c(M)}(x_3) \cup \delta_{c(M)}(x_5) = \{x_1, x_2, x_4, x_6, x_7, x_8\}$.

For any combined mechanism state $\hat{\psi} \in \hat{\Psi}_{c(M)}$, we sometimes need to identify the aspects of $\hat{\psi}$ relevant to $M' \subseteq M$. To this end, for any $\hat{\psi} \in \hat{\Psi}_{c(M)}$, we write $\hat{\psi}[M'] \in \hat{\Psi}_{c(M')}$ for the combined mechanism state capturing only those aspects of $\hat{\psi}$ relevant to $M' \subseteq M$. For any

$\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, if the set of mechanisms $M \subseteq \mathcal{M}$ under consideration is clear from the context, then we sometimes write simply $\hat{\psi}$ when it is understood to mean $\hat{\psi}[M]$.

4.2 Examples of Combination Methods

Different individuals are not required to use the same method for combining the effects of different influences. We present three different combination methods. We begin with a brief discussion of two of the simplest combination methods: random and dictatorial combinations. These are followed by a convex combination method, which is the combination method that we will assume for all of our results in the following chapters.

4.2.1 Random Combination

One of the simplest method for combining multiple influence mechanisms is the *random combination method*. The introduction of this random combination method is not due to empirical support such a method, however, it could still serve a useful purpose for comparisons with other combination methods.

The specification of this random combination method is very straight-forward. Given a set of influence mechanisms $M \subseteq \mathcal{M}$, each individual in a population selects an influence mechanism $m \in M$ according to some probability distribution over \mathcal{M} and all subsequent influence on that individual occurs through the mechanism m . The choice of probability distribution may itself be arbitrary or may be chosen to reflect any knowledge about individual behavior in the population. The simplicity of the random combination method does not warrant elaboration at this point. Although more could be said about this combination method, we continue on to some other combination methods.

4.2.2 Dictatorial Combination

Another simple method for combining multiple influence mechanisms is to order all influence mechanisms and select the first applicable mechanism from that ordering for exerting influence.

We call this the *dictatorial combination method*.

For each $x \in \mathcal{X}$, let $\succ^{\mathcal{D}_x}$ denote a total strict ordering over \mathcal{M} . The ordering $\succ^{\mathcal{D}_x}$ captures the level of susceptibility of x to each of the influence mechanisms. If $m \succ^{\mathcal{D}_x} m'$, then we say that influence by m overrules influence by m' on x .

Given a set of influence mechanisms $M \subseteq \mathcal{M}$, each $x \in \mathcal{X}$ has influence exerted on it by the mechanism $m \in M$ that appears first in the ordering \mathcal{D}_x (i.e., the mechanism to which it is most susceptible). We assume that the ordering \mathcal{D}_x does not vary over time for each individual $x \in \mathcal{X}$.

Let DICT denote the dictatorial combination method. We define $\mu_{\text{DICT}(M)}^x$ for any $M \subseteq \mathcal{M}$, $x \in \mathcal{X}$, and $\hat{\psi}, \hat{\psi}' \in \phi_{\text{DICT}(M)}(\Psi_{\mathcal{X}})$ as:

$$\mu_{\text{DICT}(M)}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \mu_m^x(\hat{\psi}[m])(\hat{\psi}'[m](x) \mid \hat{\psi}[m](x)) \quad (4.1)$$

where $m \in M$ and $m \succ^{\mathcal{D}_x} m'$ for all $m' \in M \setminus \{m\}$.

If we assume the mechanism ordering is static for each individual, then the spread of information will be restricted to certain population structure pathways. That is, given a set of mechanisms M , each individual $x \in \mathcal{X}$ will only be susceptible to influence across a single relation corresponding to the mechanism $m \in M$ that occurs first in the mechanism ordering $\succ^{\mathcal{D}_x}$. We may seek to elaborate on this observation as part of our future work.

4.2.3 Convex Combination

A common method for combining multiple measures into a single measure is to take the average of the values given by each measure [44]. When applied to influence mechanisms, one may view each influence mechanism as expressing an expert’s assessment of how an individual is influenced. The combination of these influence mechanisms can be interpreted as the combination of each expert’s assessment of the influence probabilities. This type of combination has been widely studied in the area of group decision making using what is called the linear opinion pool method (see, for example, [44, 38, 26, 11]). A related method for defining transition probabilities of individuals is used in [17].

The linear opinion pool method has been shown in other areas to offer improvements over probabilities derived from any single measure. For example, weather forecasting typically makes use of multiple models and aggregates the respective probability measures into a single forecast [38, 40]. It is concluded in [40] that the linear opinion pool method provides better forecasts than each separate model.

The linear opinion pool method is simply a type of convex combination of values, or “opinions”. We now present what we refer to as the *convex combination method*, which is motivated by the work on the linear opinion pool method and is flexible enough to capture more specific combination methods by adjusting the convex weighting distribution. We denote the convex combination method by c .

For each $M \subseteq \mathcal{M}$, let $w_c : M \rightarrow [0, 1]$ be a function that assigns a weight to each $m \in M$ such that we have $\sum_{m \in M} w_c(m) = 1$. We define the *convex combination method measure*

function $\mu_{\mathbf{c}(M)}^x$ for all $x \in \mathcal{X}$, $M \subseteq \mathcal{M}$, and $\hat{\psi}, \hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ as:

$$\mu_{\mathbf{c}(M)}^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) \stackrel{\text{def}}{=} \begin{cases} 1 & \text{if } \delta_{\mathbf{c}(M)}(x) = \emptyset \text{ and} \\ & \hat{\psi}(x) = \hat{\psi}'(x), \\ 0 & \text{if } \delta_{\mathbf{c}(M)}(x) = \emptyset \text{ and} \\ & \hat{\psi}(x) \neq \hat{\psi}'(x), \\ \gamma \cdot \sum_{m \in M'} w_{\mathbf{c}}(m) \cdot & \\ \mu_m^x(\hat{\psi}[m])(\hat{\psi}'[m](x) \mid \hat{\psi}[m](x)) & \text{otherwise,} \end{cases} \quad (4.2)$$

where $M' = \{m \in M \mid \delta_m(x) \neq \emptyset\}$ is the set of mechanisms in M under which x may be susceptible to influence and $\gamma = 1 / \sum_{\hat{\psi}'' \in \hat{\Psi}_{\mathbf{c}(M)}} \sum_{m \in M'} w_{\mathbf{c}}(m) \cdot \mu_m^x(\hat{\psi}[m])(\hat{\psi}''[m] \mid \hat{\psi}[m](x))$ is a normalizing constant that ensures $\sum_{\hat{\psi}'' \in \hat{\Psi}_{\mathbf{c}(M)}} \mu_{\mathbf{c}(M)}^x(\hat{\psi})(\hat{\psi}'' \mid \hat{\psi}(x)) = 1$.

Our definition of the individual measure assignment function for the convex combination method ignores the transition probabilities for those mechanisms under which an individual has an empty neighborhood since such individuals can not be influenced to transition by those mechanisms. Moreover, if we did include the transition probabilities of individuals with empty neighborhoods, then we can see from Equation 3.2 that the convex combination would lead to those individuals tending to stay in their same state. The resulting combined mechanism measure would give misleading transition probabilities.

Although the convex combination method permits arbitrary weight distributions, we assume for each $M \subseteq \mathcal{M}$ and $m \in M$ that $w_{\mathbf{c}}(m) = \frac{1}{|M|}$. That is, we assume the weights assigned to each mechanism form a uniform distribution for the set of mechanisms that are being combined with the convex combination method. This assumption prevents us from evaluating mechanisms that have a weight of zero and, thus, have no effect on any individual.

For the remainder of this dissertation, we assume that all sets of mechanisms are combined

using the convex combination method with uniform weight distributions.

4.3 Behavioral Types

One natural interpretation we give to combination methods is that they characterize various individual behavioral types. That is, two individuals are of different behavioral types if and only if they combine the influence exerted upon them using different combination methods. We give a brief development of this interpretation of combination methods.

The composition of a population, in terms of behavioral types, may have a significant impact on the expected spread of information. It is shown in [13] that behavioral type heterogeneity in a population leads to a faster diffusion of information than under homogeneous populations, where an individual's behavioral type is defined in terms of its susceptibility to influence by its neighbors. The impact of population heterogeneity on information propagation has also been explored in work on stochastic interacting systems [14]. These works highlight the importance of making clear any assumptions about the individual behavioral types in a population.

We define an individual's behavioral type by the combination method that they use to combine the effects of multiple types of influence. The behavioral type of each individual is given by a function $\tau : \mathcal{X} \rightarrow \mathcal{C}$ that takes each $x \in \mathcal{X}$ to the combination method $\tau(x) \in \mathcal{C}$ employed by x .

Individuals of the same behavioral type may be further differentiated by their parameterization of the corresponding combination method. With respect to the dictatorial combination method, individuals with the same ordering of influence mechanisms are the same behavioral subtype.

Individuals $x, x' \in \mathcal{X}$ with $\tau(x) = \tau(x') = \text{DICT}$ are the same behavioral subtype if and

only if $\succsim^{D_x} = \succsim^{D_{x'}}$. In this case, there does not exist a set of influence mechanisms $M \subseteq \mathcal{M}$ for which individuals x and x' will be influenced by different influence mechanisms based on their respective orderings. Thus, exertion of influence on x and x' is the same for any set of influence mechanisms and they are therefore the same behavioral subtype. For the DICT combination method, each behavioral subtype forms an equivalence class with respect to influence mechanism orderings.

We define a simple distribution over the behavioral types in a population that characterizes the individual behaviors. Let $F : \text{Pwr}(\mathcal{X}) \rightarrow (\mathcal{C} \rightarrow [0, 1])$ be the *behavioral type distribution function* defined for any $X \subseteq \mathcal{X}$ and $c \in \mathcal{C}$ by

$$F(X)(c) \stackrel{\text{def}}{=} \frac{|\{x \in X \mid \tau(x) = c\}|}{|X|}, \quad (4.3)$$

with $\sum_{c \in \mathcal{C}} F(X)(c) = 1$. The function $F(X)$ gives the distribution of behavioral types in the population X . The probability of a randomly selected individual $x \in X$ having behavioral type $c \in \mathcal{C}$ is given by $F(X)(c)$.

A population $X \subseteq \mathcal{X}$ in which there exists more than one behavioral type is called a *heterogeneous population*. Otherwise, a population X is called a *homogeneous population*. A homogeneous population X is thus one in which $F(X)(c) = 1$ for some $c \in \mathcal{C}$ and, necessarily, $F(X)(c') = 0$ for all $c' \neq c$. A population consisting of only a singleton $\{x\}$ is always a homogeneous population. A homogeneous population in which all individuals are the same behavioral subtype is called a *subtype homogeneous population*.

There are many questions that one may ask about the influence and information propagation in a population consisting of many different behavioral types (e.g., rate of influence propagation in homogeneous vs. heterogeneous populations). We restrict our work in this dissertation to behavioral subtype homogeneous populations (i.e., populations in which all individuals com-

bine multiple mechanisms in the same manner). As we have already stated, we consider only a uniform weighted convex combination method throughout the remainder of this dissertation. We defer a more comprehensive treatment of behavioral types to future work.

Chapter 5

Maximizing Influence

The influence maximization problem [27] has received considerable attention in recent years. We now give a statement of this problem using our formal framework.

5.1 Expected Influence

Analysis of information propagation in a population requires a method for determining the expected number of individuals that have been influenced in some way. The notion of expected influence that we make use of assumes that we have a set of individuals that are each initially in a specified individual combined mechanism state and that a set of mechanisms is used to influence others to adopt that same individual combined mechanism state. The expected influence is given by the long-term behavior of the influence propagation. In the following, we give specification to the notion of expected influence that we use throughout this dissertation.

Let $\mathcal{I} : Pwr(\mathcal{X}) \times Pwr(\mathcal{M}) \times \hat{\Psi}_{c(\mathcal{M})} \rightarrow [0, |\mathcal{X}|]$ be the *expected influence function*. We refer to any argument $X \subseteq \mathcal{X}$ of \mathcal{I} as a *targeted population* and any argument $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ of \mathcal{I} as *influence information*.

We define the expected influence function \mathcal{I} for all targeted populations $X \subseteq \mathcal{X}$, influence mechanisms $M \subseteq \mathcal{M}$, and influence information $\hat{\psi} \in \hat{\Psi}_{c(M)}$ as:

$$\mathcal{I}(X, M, \hat{\psi}) \stackrel{\text{def}}{=} \sum_{\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})} o(\hat{\psi}') \cdot \pi_{c(M)}(\hat{\psi}'), \quad (5.1)$$

where $o : \phi_{c(M)}(\Psi_{\mathcal{X}}) \rightarrow \mathbb{R}^+$ is the objective function that assigns a value to each population mechanism state and $\pi_{c(M)} : \phi_{c(M)}(\Psi_{\mathcal{X}}) \rightarrow [0, 1]$ is a stationary distribution of the Markov chain induced by M .

For our purposes, we define the *objective function* o for all mechanisms $M \subseteq \mathcal{M}$, influence information $\hat{\psi} \in \hat{\Psi}_{c(M)}$, and population states $\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})$ as:

$$o(\hat{\psi}') \stackrel{\text{def}}{=} |\{x \in \mathcal{X} \mid \hat{\psi}'(x) = \hat{\psi}[M]\}|, \quad (5.2)$$

which gives a count of the individuals that have been influenced to adopt the specified influence information $\hat{\psi}$ relevant to the mechanisms M . Our subsequent results assume an objective function defined as in Equation 5.2, however, we may choose to investigate alternate interpretations of the values assigned to states, such as importance, utility, or cost.

Although there may be more than one stationary distribution for the Markov chain induced by M , we can use an initial state distribution to impose a particular stationary distribution. We use the targeted population and influence information to place constraints on initial state distributions that are used for the Markov chain induced by the mechanism measures. Given any population $X \subseteq \mathcal{X}$, population combined mechanism state $\hat{\psi} \in \hat{\Psi}_{c(M)}$, and influence mechanisms $M \subseteq \mathcal{M}$, we require an initial state distribution $\pi_{c(M)}^0$ to satisfy the following constraints:

1. $\pi_{c(M)}^0(\hat{\psi}') = 0$ for all $\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})$ such that $\hat{\psi}'(x) \neq \hat{\psi}[M]$ for all $x \in X$;

2. $\pi_{\mathbf{c}(M)}^0(\hat{\psi}') = 0$ for all $\hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ such that $\hat{\psi}'(x) = \hat{\psi}[M]$ for some $x \notin X$;
3. $\pi_{\mathbf{c}(M)}^0(\hat{\psi}') > 0$ for all $\hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ such that $\hat{\psi}'(x) = \hat{\psi}[M]$ for all $x \in X$.

That is, we require that all individuals in the targeted population have states equal to the aspects of $\hat{\psi}$ that are relevant to M and all individuals not in the targeted population have states that are not equal to the influence information relevant to M . These constraints are similar to those made in related work [27].

For any $M \subseteq \mathcal{M}$, if $|\hat{\Psi}_{\mathbf{c}(M)}| = 2$, then an initial state distribution will be deterministic (i.e., $\pi_{\mathbf{c}(M)}^0(\hat{\psi}') = 1$ for some $\hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ and $\pi_{\mathbf{c}(M)}^0(\hat{\psi}'') = 1$ for $\hat{\psi}'' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ such that $\hat{\psi}' \neq \hat{\psi}''$). Otherwise, we assume a uniform distribution over the states $\hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ that satisfy our constraints on the initial state distribution. Of course, specification of an initial state distribution is irrelevant for sets of mechanisms that induce an irreducible Markov chain over the complete population since it will have a unique stationary distribution that is independent of an initial state distribution.

For any $X \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(\mathcal{M})}$, we define $\mathcal{I}(X, \emptyset, \hat{\psi}) = 0$ since state transitions are defined in terms of the influence mechanisms used. The lack of any influence mechanisms and specification of $\hat{\Psi}_{\emptyset} = \emptyset$ result in no influence information with which to influence a population. Similarly, we define $\mathcal{I}(\emptyset, M, \hat{\psi}) = 0$ since no individuals are given the influence information in the first place.

The difficulty of computing the expected influence function given by Equation 5.1 is not due to the objective function or the summation. Instead, the difficulty is in finding a stationary distribution for the Markov chain induced by a set of mechanisms.

Consider any $M \subseteq \mathcal{M}$ and let $n = |\phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})|$. Finding a stationary distribution for a Markov chain involves computing an eigenvector with eigenvalue 1. A common algorithm for doing this is to use the inverse power method. Established complexity results state that

the inverse power method takes time on the order of $O(n^3)$. Clearly, the majority of the time required to compute expected influence lies in the eigenvector (i.e., stationary distribution) solution. There are other algorithms that might solve this problem more efficiently under specific assumptions about the problem (e.g., size of state space, sparsity of matrix), but they still take up the majority of the time in computing expected influence.

5.2 The Influence Maximization Problem

The influence problem to which we apply our model is called the influence maximization problem. This problem was identified in [15] and has been studied extensively in more recent work [27, 28, 30, 8]. We give the following statement of the influence maximization problem.

Given an integer $k > 0$, find k individuals that maximize the expected number of individuals that have adopted some specific influence information (i.e., individuals that have been influenced). A formal statement of this problem follows:

The Influence Maximization Problem : Given $k > 0$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(\mathcal{M})}$, what targeted population $X \subseteq \mathcal{X}$ of size k maximizes $\mathcal{I}(X, M, \hat{\psi})$?

We use the influence maximization problem to demonstrate some of the properties and claimed improvements of our model over related influence models. By taking this approach, we view the influence maximization problem as a general problem that can be studied under any influence model, each having different properties with respect to the problem.

Chapter 6

Population Topology and Targeted Optimality

The primary contribution of our approach to modeling influence comes from our usage of multiple influence mechanisms for spreading information throughout a population. In this chapter, we show how the topology of a population impacts the expected influence and how knowledge of the population structure under the various mechanisms can improve our ability to recognize optimal targeted population sizes for the influence maximization problem.

We begin by identifying the population structure that arises from mechanism relations under one or more influence mechanisms. This is followed by a definition of optimal targeted populations and a discussion on how the population structure affects the determination of an optimal targeted population.

6.1 Connected Populations

We apply graph-theoretic terms to binary relations by regarding pairs in the representation as edges of a graph. In this way, we can rephrase the notion of transitive closure to say that r^+ contains (x, x') whenever there is some finite path from x to x' in r . Most importantly, the graph-theoretic view focuses attention on connectivity properties of the relations. As seen in Figure 1.1, relations may leave a population disconnected, with some individuals occurring in connected components, and others standing isolated from all other individuals.

We write $[x]_r$ to denote the set of individuals that are connected to x by a path of any length in the transitive closure of the symmetrization of r , which we identify as the *component* of the graph of r containing x . Stated formally, $[x]_r = r^\oplus[x]$.

We say that a set $X \subseteq \mathcal{X}$ is a *connected set* under r just in case each individual in X is reachable from each other individual in X under the transitive closure of the symmetrization of r , that is, X is connected under r just in case $x \in [x']_r$ for each $x, x' \in X$ with $x \neq x'$. By symmetry, if $x \in [x']_r$, then $[x]_r = [x']_r$.

A component $[x]_r$ need not contain x , for if x is not related either to itself or to any other individual by r , then $[x]_r = \emptyset$. In fact, $[x]_r$ is the maximal connected set of x under r , and we call it the *maximal connected component* of x under r .

We say that x is *isolated* in r if it is not connected to any other individual by r^\oplus . This can happen in two ways: either r contains no pair containing x , in which case $[x]_r = \emptyset$, or the only pair containing x is the reflexive pair (x, x) , in which case $[x]_r = \{x\}$. If x is not isolated, then it is connected to at least one other individual $x' \neq x$, and hence $[x]_r$ must contain both x and x' by symmetrization so that $|[x]_r| > 1$.

We write $Isolates(r) = \{x \in \mathcal{X} \mid [x]_r \subseteq \{x\}\}$ to denote the set of all individuals isolated under r , and write $Components(r)$ to denote the set of all maximal connected components

under r apart from the isolated individuals, that is, $Components(r) = \{[x]_r \mid |[x]_r| > 1\}$. The set of all maximal connected components including individuals isolated under r is denoted by $Components^+(r) = Components(r) \cup \{\{x\} \mid x \in Isolates(r)\}$. For example, in Figure 1.1, *is-coworker-of* has one maximal connected component and one isolate; *is-friend-of* has only one maximal connected component.

We extend the notion to sets of relations by defining $[x]_R$ by $[x]_R = [x]_{\bigcup R}$, representing the set of individuals that are connected to x under $(\bigcup R)^\oplus$. From this, the definitions for sets of maximal connected components extend naturally to sets of relations and we write $Isolates(R)$, $Components(R)$, and $Components^+(R)$ to denote all such sets. We often refer only to a single relation since $\bigcup R$ itself constitutes a single relation.

For any $r \in \mathcal{R}$, we write $\eta(r) = |Components(r)|$ for the number of distinct non-isolated maximal connected components under r . We include the number of isolated individuals under r by writing $\eta^+(r) = \eta(r) + |Isolates(r)|$.

The following lemmas help to clarify the effects on the number of maximal connected components when using multiple relations and, hence, multiple influence mechanisms.

Lemma 1. *For all $R \subseteq \mathcal{R}$, $Isolates(R) \subseteq \bigcup_{r \in R} Isolates(r)$.*

Proof. Consider any $R \subseteq \mathcal{R}$. Assume $Isolates(R) \not\subseteq \bigcup_{r \in R} Isolates(r)$. There exists $x \in Isolates(R)$ such that $x \notin \bigcup_{r \in R} Isolates(r)$. By definition, we have $\bigcup R = \bigcup_{r \in R} r$. Therefore, we must have $x \in Isolates(r)$ for some $r \in R$. This gives a contradiction and concludes proof of the claim. \square

Lemma 2. *For all $R' \subseteq R \subseteq \mathcal{R}$, $\eta^+(R) \leq \eta^+(R')$.*

Proof. By definition of $\bigcup R$, all ordered pairs of $\bigcup R'$ are included in $\bigcup R$ for each $R' \subseteq R \subseteq \mathcal{R}$. Therefore, the number of maximal connected components under $\bigcup R$ cannot be greater than under $\bigcup R'$. \square

Lemma 3. For all $R \subseteq \mathcal{R}$, $\eta(R) \leq \min_{r \in R} \eta^+(r)$.

Proof. The claim follows directly from Lemma 2. □

Lemma 4. For all $R \subseteq \mathcal{R}$, $\eta^+(R) \leq \sum_{r \in R} \eta^+(r)$.

Proof. The claim follows directly from definition of $\bigcup R = \bigcup_{r \in R} r$ for any $R \subseteq \mathcal{R}$. □

Lemma 5. For all $R \subseteq \mathcal{R}$, $\eta(R) \leq \sum_{r \in R} \eta(r)$.

Proof. The claim follows directly from definition of $\bigcup R = \bigcup_{r \in R} r$ for any $R \subseteq \mathcal{R}$. □

6.2 Bridges Between Populations

Under the relation *is-coworker-of* that is used in Section 1.1, the population exists within two maximal connected components. However, the population exists within a single maximal connected component under the relation *is-friend-of*. Two individuals that exist in different maximal connected components in one relation may exist in the same maximal connected component in a different relation. The presence or absence of any such pairs of individuals provides information about the potential expected influence in a population subject to multiple mechanisms and their corresponding relations.

Definition 4. A pair (x, x') is called a bridge between $r, r' \in \mathcal{R}$ if and only if $[x]_r \neq [x']_r$ and $(x, x') \in (r')^+$.

In Figure 1.1, we can see that x_8 is unable to exert influence on x_3 since x_3 is isolated under relation *is-coworker-of*. Under relation *is-friend-of*, x_3 is not isolated and is connected to each individual in $\{x_1, x_2, x_4, x_8\}$. By definition of a bridge, we have, for example, that the pair (x_3, x_8) is a bridge between *is-coworker-of* and *is-friend-of*.

Theorem 2. For all $R \subseteq \mathcal{R}$, $\eta(R) = \sum_{r \in R} \eta(r)$ if and only if there does not exist a bridge between any $r, r' \in R$.

Proof. We prove both directions of the claim by contradiction. Consider any $R \subseteq \mathcal{R}$. Assume $\eta(R) = \sum_{r \in R} \eta(r)$ and there exists a bridge between $r, r' \in R$. Since there exists a bridge, there must exist $[x]_r, [x']_r \in \text{Components}(r)$ such that $[x]_r \neq [x']_r$ and $(x, x') \in (r')^+$. This results in $[x]_{\{r, r'\}} = [x']_{\{r, r'\}}$. It follows that $\eta(R) \neq \sum_{r \in R} \eta(r)$, which contradicts one of our assumptions.

For the other direction, assume now that there does not exist a bridge between any $r, r' \in R$ and $\eta(R) \neq \sum_{r \in R} \eta(r)$. From Lemma 5, we have $\eta(R) < \sum_{r \in R} \eta(r)$. We rewrite this inequality as $|\text{Components}(R)| < |\text{Components}(r_1)| + |\text{Components}(r_2)| + \dots + |\text{Components}(r_{|R|})|$. It follows by the pigeonhole principle that there must exist $x, x' \in \mathcal{X}$ and $r_i, r_j \in R$ such that $[x]_{r_i} \neq [x']_{r_i}$ and $[x]_{r_j} = [x']_{r_j}$. By definition, we can choose x, x' such that (x, x') is a bridge. This leads to a contradiction of our assumption that there does not exist a bridge. \square

Corollary 1. For all $R \subseteq \mathcal{R}$, $\text{Components}(R) = \bigcup_{r \in R} \text{Components}(r)$ if and only if there does not exist a bridge between any $r, r' \in R$.

Proof. We prove both directions of the claim by contradiction. Consider any $R \subseteq \mathcal{R}$. Assume $\text{Components}(R) = \bigcup_{r \in R} \text{Components}(r)$ and there exists a bridge between $r, r' \in R$. Since there exists a bridge, there must exist $r, r' \in R$ and $[x]_r, [x']_r \in \text{Components}(r)$ such that $[x]_r \neq [x']_r$ and $(x, x') \in (r')^+$. This results in $[x]_{\{r, r'\}} = [x']_{\{r, r'\}}$. It follows that $\text{Components}(R) \neq \bigcup_{r \in R} \text{Components}(r)$, which contradicts one of our assumptions.

For the other direction, assume now that there does not exist a bridge between any $r, r' \in R$ and $\text{Components}(R) \neq \bigcup_{r \in R} \text{Components}(r)$. We rewrite this inequality as $\text{Components}(R) \neq \text{Components}(r_1) \cup \text{Components}(r_2) \cup \dots \cup \text{Components}(r_{|R|})$. It follows that there must exist $x, x' \in \mathcal{X}$, distinct $r_i, r_j \in R$, $X \in \text{Components}(r_i)$, and $X' \in \text{Components}(r_j)$ such

that $x \in X$, $x \in X'$, $x' \notin X$, and $x' \in X'$; otherwise, we would have $\text{Components}(R) = \bigcup_{r \in R} \text{Components}(r)$. By definition, we can choose x, x' such that (x, x') is a bridge. This leads to a contradiction of our assumption that there does not exist a bridge. \square

6.3 Optimal Targeted Populations

Given influence mechanisms $M \subseteq \mathcal{M}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, the expected influence function \mathcal{I} induces a total preorder $\succsim_M^{\hat{\psi}}$ over $\text{Pwr}(\mathcal{X})$ such that $X \succsim_M^{\hat{\psi}} X'$ if and only if $\mathcal{I}(X, M, \hat{\psi}) \geq \mathcal{I}(X', M, \hat{\psi})$. The induced orderings over sets of individuals give rise to notions of maximizing and optimal targeted populations.

Definition 5. *Given influence mechanisms $M \subseteq \mathcal{M}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, X is maximizing with respect to M and $\hat{\psi}$ if and only if $X \succsim_M^{\hat{\psi}} X'$ for all $X' \subseteq \mathcal{X}$.*

We define the set of *maximizing targeted populations* for $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ as $O_{M, \hat{\psi}}^* \stackrel{\text{def}}{=} \{X \subseteq \mathcal{X} \mid X \succsim_M^{\hat{\psi}} X' \text{ for all } X' \subseteq \mathcal{X}\}$. The set of *minimal maximizing (or optimal) targeted populations* for $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ is given by $O_{M, \hat{\psi}}^{**} \stackrel{\text{def}}{=} \{X \in O_{M, \hat{\psi}}^* \mid |X| \leq |X'| \text{ for all } X' \in O_{M, \hat{\psi}}^*\}$. We write $O \in O_{M, \hat{\psi}}^*$ for a maximizing targeted population. By definition, $O_{M, \hat{\psi}}^{**} \subseteq O_{M, \hat{\psi}}^*$. We refer to each $X \notin O_{M, \hat{\psi}}^{**}$ as *sub-optimal* with respect to M and $\hat{\psi}$.

For the authoritarian mechanism *Auth* and relation *is-coworker-of* used in Section 1.1, each maximizing targeted population must contain $\{x_3, x_7, x_8\}$, so we have $O_{\text{Auth}, \hat{\psi}}^* = \{X \subseteq \mathcal{X} \mid \{x_3, x_7, x_8\} \subseteq X\}$. The set of optimal targeted populations is given by $O_{\text{Auth}, \hat{\psi}}^{**} = \{\{x_3, x_7, x_8\}\}$.

Lemma 6. *For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $O \in O_{M, \hat{\psi}}^*$, we have $\text{Isolates}(r_{c(M)}) \subseteq O$.*

Proof. The claim follows from Equation 3.2 and the definition of a maximizing targeted population. \square

Although the set $O_{M,\hat{\psi}}^*$ captures all sets of targeted individuals that maximize expected influence using $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, we are often limited in practice to considering maximizing targeted populations that are no larger than a particular size. Of course, this is a constraint specified in the statement of the influence maximization problem.

Given some integer $k > 0$, we define the set of *k-bounded maximizing targeted populations* for any $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ by $O_{M,\hat{\psi}}^{k*} \stackrel{\text{def}}{=} \{X \subseteq \mathcal{X} \mid X \succ_M^{\hat{\psi}} X' \text{ for all } X' \subseteq \mathcal{X} \text{ such that } |X|, |X'| \leq k\}$. The set of *k-bounded minimal maximizing (or optimal) targeted populations* is given by $O_{M,\hat{\psi}}^{k**} \stackrel{\text{def}}{=} \{X \in O_{M,\hat{\psi}}^{k*} \mid |X| \leq |X'| \text{ for all } X' \in O_{M,\hat{\psi}}^{k*}\}$. As an example, if we assume $k = 2$, then the set of *k-bounded optimal targeted populations* for the authoritarian mechanism *Auth* over relation *is-coworker-of* in Section 1.1 is $O_{Auth,\hat{\psi}}^{k**} = \{\{x_7, x_8\}\}$.

Isolated individuals do not provide any benefit to the propagation of information in a population. This is due to the fact that isolated individuals must be contained in an initial targeted population in order to contribute to an increase in expected influence (see Lemma 6). We therefore ignore isolated individuals in most of our discussions about optimality of targeted populations.

We restrict the sets of maximizing targeted populations to only those individuals that are non-isolated under $r_{c(M)}$ for any $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ by defining $\hat{O}_{M,\hat{\psi}}^* \stackrel{\text{def}}{=} \{X \subseteq \mathcal{X} \mid \exists X' \in O_{M,\hat{\psi}}^* \text{ such that } X = X' \setminus \text{Isolates}(r_{c(M)})\}$. The restatement in terms of optimal targeted populations is given by $\hat{O}_{M,\hat{\psi}}^{**} \stackrel{\text{def}}{=} \{X \subseteq \mathcal{X} \mid \exists X' \in O_{M,\hat{\psi}}^{**} \text{ such that } X = X' \setminus \text{Isolates}(r_{c(M)})\}$. As with other (minimal) maximizing targeted populations, we write $O \in \hat{O}_{M,\hat{\psi}}^*$ for a particular non-isolated (minimal) maximizing targeted population. Non-isolated (minimal) maximizing sets extend naturally to such *k-bounded* sets, which we denote by $\hat{O}_{M,\hat{\psi}}^{k*}$.

and $\hat{O}_{M,\hat{\psi}}^{k**}$.

6.4 Population Monotonicity

Influence mechanisms exhibit various properties that we can exploit in order to better inform our choice of optimal targeted population. One such property that some types of mechanisms exhibit is that the expected influence resulting from the use of those mechanisms is non-decreasing as the targeted population increases in size.

In Section 1.1, we used a targeted population $\{x_7\}$ and showed that it was able to influence only two other individuals using an authoritarian mechanism over the relation *is-coworker-of*. If we chose to include any other individual in the targeted population in addition to x_7 , then the expected influence would not decrease. We refer to this as population monotonicity and prove this property of an authoritarian mechanism in Theorem 18 in Section 7.2.1.

A mechanism $m \in \mathcal{M}$ is called *non-decreasing population monotonic* if and only if $\mathcal{I}(X, \{m\}, \hat{\psi}) \geq \mathcal{I}(X', \{m\}, \hat{\psi})$ for all $X' \subseteq X \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{e(\mathcal{M})}$. A mechanism $m \in \mathcal{M}$ is called *non-increasing population monotonic* if and only if $\mathcal{I}(X, \{m\}, \hat{\psi}) \leq \mathcal{I}(X', \{m\}, \hat{\psi})$ for all $X' \subseteq X \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{e(\mathcal{M})}$. We say $M \subseteq \mathcal{M}$ is non-decreasing population monotonic if and only if each $m \in M$ is non-decreasing population monotonic. Similarly, we say $M \subseteq \mathcal{M}$ is non-increasing population monotonic if and only if each $m \in M$ is non-increasing population monotonic. Mechanisms $M \subseteq \mathcal{M}$ are called *population non-monotonic* if and only if M is not non-decreasing or non-increasing population monotonic.

It follows from Equation 3.2, that the expected influence using any mechanism with the empty mechanism relation will be equal to the size of the targeted population. Such mechanisms satisfy our definition of non-decreasing population monotonicity.

Lemma 7. *For all $m \in \mathcal{M}$, if $r_m = \emptyset$, then m is non-decreasing population monotonic.*

Proof. Consider any $m \in \mathcal{M}$ such that $r_m = \emptyset$. For all targeted populations $X \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, it follows from Equation 3.2 that $\mathcal{I}(X, \{m\}, \hat{\psi}) = |X|$ since targeted individuals are unable to change states. This shows that m is non-decreasing population monotonic. \square

Theorem 3. *For all $m \in \mathcal{M}$, if $\mu_m(\hat{\psi}' \mid \hat{\psi}) > 0$ for all $\hat{\psi}, \hat{\psi}' \in \phi_m(\Psi_{\mathcal{X}})$, then m is non-decreasing population monotonic.*

Proof. Consider any $m \in \mathcal{M}$ such that $\mu_m(\hat{\psi}' \mid \hat{\psi}) > 0$ for all $\hat{\psi}, \hat{\psi}' \in \phi_m(\Psi_{\mathcal{X}})$. Let $(\hat{\psi}_m^t)_{t \geq 0}$ be the Markov chain induced by m . It follows directly from the definition of irreducibility that $(\hat{\psi}_m^t)_{t \geq 0}$ is irreducible. Therefore, a unique stationary distribution exists that is independent of the starting state and, hence, independent of the selection of targeted population. This gives $\mathcal{I}(X, \{m\}, \hat{\psi}'') = \mathcal{I}(X \cup X', \{m\}, \hat{\psi}'')$ for all $X, X' \subseteq \mathcal{X}$ and $\hat{\psi}'' \in \hat{\Psi}_{c(\mathcal{M})}$. It follows that m is non-decreasing population monotonic. \square

We can determine whether a mechanism is non-decreasing population monotonic through a locally-checkable condition by looking at the mechanism state transitions of each individual.

Corollary 2. *For all $m \in \mathcal{M}$, if $\mu_m^x(\hat{\psi})(\hat{\psi}'(x) \mid \hat{\psi}(x)) > 0$ for all $x \in \mathcal{X}$ and $\hat{\psi}, \hat{\psi}' \in \phi_m(\Psi_{\mathcal{X}})$, then m is non-decreasing population monotonic.*

Proof. The claim follows directly from Equation 3.3 and Theorem 3. \square

For any non-decreasing population monotonic mechanism, we can simply target the complete population in order to maximize expected influence. Although a smaller optimal targeted population may exist, we know that the complete population in this case will not produce less expected influence than any subset.

Theorem 4. *For all $m \in \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, if m is non-decreasing population monotonic, then $\mathcal{X} \in O_{\{m\}, \hat{\psi}}^*$.*

Proof. Assume any $m \in \mathcal{M}$ such that m is non-decreasing population monotonic. By definition of maximizing targeted population and non-decreasing population monotonicity, we have $\mathcal{I}(O, \{m\}, \hat{\psi}) = \mathcal{I}(O \cup X, \{m\}, \hat{\psi})$ for all $X \subseteq \mathcal{X}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $O \in O_{\{m\}, \hat{\psi}}^*$. It follows that $\mathcal{X} \in O_{\{m\}, \hat{\psi}}^*$. \square

6.5 Component Restricted Optimal Targeted Populations

Maximizing the spread of influence over the complete population is the typical goal of influence problems. We have already described types of relations that exhibit a natural disconnectedness in their structure; that is, they consist of more than a single maximal connected component. We restrict the expected influence function to any maximal connected component and define maximizing targeted populations in terms of a component restricted expected influence function.

For any $X \subseteq \mathcal{X}$, $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $X' \in \text{Components}^+(r_{c(M)})$, we write $\mathcal{I}_{X'}(X, M, \hat{\psi})$ for the expected influence restricted to the population in the maximal connected component X' . In this way, we have $\mathcal{I}_{\mathcal{X}}(X, M, \hat{\psi}) = \mathcal{I}(X, M, \hat{\psi})$. The propagation of information being restricted to a population $X' \in \text{Components}^+(r_{c(M)})$ gives $0 \leq \mathcal{I}_{X'}(X, M, \hat{\psi}) \leq |X'|$ for all $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. We have $\mathcal{I}_{X'}(X, M, \hat{\psi}) = 0$ whenever $X \cap X' = \emptyset$, which follows from our specification of $\mathcal{I}(\emptyset, M, \hat{\psi}) = 0$.

For any maximal connected component, we can determine targeted populations that maximize influence over that component. For any $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, the set of all maximizing targeted populations for the population in $X \in \text{Components}^+(r_{c(M)})$ is given by $O_{M, \hat{\psi}}^*(X) = \{X' \subseteq X \mid \mathcal{I}_X(X', M, \hat{\psi}) \geq \mathcal{I}_X(X'', M, \hat{\psi}) \text{ for all } X'' \subseteq X\}$. The set of all minimal maximizing (or optimal) targeted populations in the maximal connected component X is given by $O_{M, \hat{\psi}}^{**}(X) = \{X' \in O_{M, \hat{\psi}}^*(X) \mid |X'| \leq |X''| \text{ for all } X'' \in O_{M, \hat{\psi}}^*(X)\}$. These sets of (minimal) maximizing targeted populations for any given maximal connected component

extend naturally to k -bounded sets of targeted populations.

We show that the probabilities of influencing individuals within one maximal connected component are independent of the probabilities of influencing individuals in a different maximal connected component. This independence of influence exerted within different maximal connected components follows directly from our definition of a population measure function (Equation 3.3).

Theorem 5. *For all $M \subseteq \mathcal{M}$, $\hat{\psi}, \hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$, and $X, X' \in \text{Components}^+(r_{\mathbf{c}(M)})$, if $X \neq X'$, then $\mu_{\mathbf{c}(M)}^X(\hat{\psi})(\hat{\psi}'(X) \mid \hat{\psi}(X))$ is independent of $\mu_{\mathbf{c}(M)}^{X'}(\hat{\psi})(\hat{\psi}'(X') \mid \hat{\psi}(X'))$.*

Proof. The claim follows directly from Equation 3.3 since the transition probabilities of an individual are dependent only on its current state and on the states of its neighbors. \square

Theorem 6. *For all $X \subseteq \mathcal{X}$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, we have that $\mathcal{I}(X, M, \hat{\psi}) = \sum_{X' \in \text{Components}^+(r_{\mathbf{c}(M)})} \mathcal{I}_{X'}(X, M, \hat{\psi})$.*

Proof. Consider any $X \subseteq \mathcal{X}$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$. Let $\pi_{\mathbf{c}(M)}^{X'}$ be the stationary distribution and $o^{X'}$ be the objective function used to compute $\mathcal{I}_{X'}(X, M, \hat{\psi})$ for any $X' \in \text{Components}^+(r_{\mathbf{c}(M)})$. From Theorem 5, $\pi_{\mathbf{c}(M)}^{X'}$ is probabilistically independent of $\pi_{\mathbf{c}(M)}^{X''}$ for all $X', X'' \in \text{Components}^+(r_{\mathbf{c}(M)})$ whenever $X' \neq X''$. For all $X' \in \text{Components}^+(r_{\mathbf{c}(M)})$, $o^{X'}$ will count only individuals in X' since it follows from Theorem 5 that there is zero probability of any $x \in X'$ influencing any $x' \notin X'$. For all $\hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$, we have $\pi_{\mathbf{c}(M)}(\hat{\psi}') = \prod_{X' \in \text{Components}^+(r_{\mathbf{c}(M)})} \pi_{\mathbf{c}(M)}^{X'}(\hat{\psi}'(X'))$ and $o(\hat{\psi}') = \sum_{X' \in \text{Components}^+(r_{\mathbf{c}(M)})} o^{X'}(\hat{\psi}'(X'))$. We now have the following derivation of the claimed result in which we let $S_{\hat{\psi}'}^{X'} = \{\hat{\psi}'' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}}) \mid$

$\hat{\psi}''(X') = \hat{\psi}'(X')\}$:

$$\begin{aligned}
\mathcal{I}(X, M, \hat{\psi}) &= \sum_{\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})} o(\hat{\psi}') \cdot \pi_{c(M)}(\hat{\psi}') \\
&= \sum_{\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})} \left[\sum_{X' \in \text{Components}^+(r_{c(M)})} o^{X'}(\hat{\psi}'(X')) \right] \\
&\quad \cdot \left[\prod_{X' \in \text{Components}^+(r_{c(M)})} \pi_{c(M)}^{X'}(\hat{\psi}'(X')) \right] \\
&= \sum_{\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}})} \sum_{X' \in \text{Components}^+(r_{c(M)})} o^{X'}(\hat{\psi}'(X')) \cdot \left[\prod_{\hat{\psi}'' \in S_{\hat{\psi}'}^{X'}} \pi_{c(M)}(\hat{\psi}'') \right] \\
&= \sum_{X' \in \text{Components}^+(r_{c(M)})} \sum_{\hat{\psi}' \in \phi_{c(M)}(\Psi_{\mathcal{X}'})} o^{X'}(\hat{\psi}') \cdot \pi_{c(M)}^{X'}(\hat{\psi}') \\
&= \sum_{X' \in \text{Components}^+(r_{c(M)})} \mathcal{I}_{X'}(X, M, \hat{\psi}).
\end{aligned}$$

□

Corollary 3. For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(M)}$, $O \in O_{M, \hat{\psi}}^*$, and $O' \in \hat{O}_{M, \hat{\psi}}^*$, we have $\mathcal{I}(O, M, \hat{\psi}) = \mathcal{I}(O', M, \hat{\psi}) + |\text{Isolates}(r_{c(M)})|$.

Proof. The claim follows directly from Theorem 6 and Lemma 6. □

6.6 Targeted Population Size

The size of a targeted population, k , is given simply as a parameter of the influence maximization problem. There is no mention of what values for k will give the largest expected influence over all possible choices. The value we choose for k is not guaranteed to give the largest possible expected influence either for the complete population or within any given maximal connected component. We define what it means for k to be optimal and also give a notion of optimal k values for each maximal connected component in a population.

Definition 6. For any $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, we say that k is optimal (with respect to M) if and only if $k \geq |O|$ for some $O \in O_{M, \hat{\psi}}^{**}$.

Definition 7. For any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{c(M)})} O_{M, \hat{\psi}}^{**}(X)$, we say that k is component optimal (with respect to M) if and only if $k \geq \max_{1 \leq i \leq \eta^+(r_{c(M)})} |\vec{o}_i|$.

We refer to any $k > 0$ that is not (component) optimal as (component) sub-optimal.

Lemma 8. For all $M \subseteq \mathcal{M}$, if $k > 0$ is component optimal with respect to M , then $k \cdot \eta^+(r_{c(M)})$ is optimal with respect to M .

Proof. The claim follows directly from Definitions 6 and 7. □

Lemma 9. For all $M \subseteq \mathcal{M}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, if $k > 0$ is component optimal with respect to M , then $O_{M, \hat{\psi}}^{k**}(X) = O_{M, \hat{\psi}}^{**}(X)$ for all $X \in \text{Components}^+(r_{c(M)})$.

Proof. The claim follows directly from Definitions 6 and 7. □

There are mechanisms that will never influence anyone in a population to accept some influence information, no matter which individuals are targeted. A trivial example is a mechanism that is opposite to a contagion mechanism *Cont*, where each individual transitions with certainty to an individual mechanism state other than the influence information.

On the other hand, some mechanisms exhibit the opposite property, in that they will always be able to influence someone in a population. An example of such a mechanism is a contagion mechanism *Cont* as we specified in Section 3.7.5, where infected individuals remain infected. Other types of influence may only exhibit this property over some relations, which we show for authoritarian and group conformity types of influence in Section 7.2. We use this property of mechanisms to inform our choice of size for an optimal targeted population.

Definition 8. A mechanism $m \in \mathcal{M}$ is said to exert non-vanishing influence if and only if for all $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, we have $\mathcal{I}_X(X', \{m\}, \hat{\psi}) \geq 1$ for each $X \in \text{Components}^+(r_m)$ whenever $X' \cap X \neq \emptyset$.

That is, a mechanism is called non-vanishing if and only if there is an expectation that at least one individual will be influenced in each maximal connected component that contains at least one individual from the targeted population.

For a contagion mechanism, we can see from the definition of μ_{Cont} in Equation 3.9 that each $x \in \mathcal{X}$ will be influenced with certainty only if a neighbor is itself influenced. Moreover, each targeted individual will remain influenced. As a result, at least one individual will be influenced in each maximal connected component whenever we target at least one individual in that component. We prove this non-vanishing property of a contagion mechanism in Theorem 19 in Section 7.2.2.

Lemma 10. For all $m \in \mathcal{M}$, if $r_m = \emptyset$, then m is non-vanishing.

Proof. Consider any $m \in \mathcal{M}$ such that $r_m = \emptyset$. From Equation 3.2, we have $\mu_m^x(\emptyset)(\hat{\psi} \mid \hat{\psi}) = 1$ for all $x \in \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_m$. It follows that m is non-vanishing. \square

Definition 9. A set of mechanisms $M \subseteq \mathcal{M}$ is called a non-vanishing set (of mechanisms) if and only if each $m \in M$ is non-vanishing and $\mathcal{C}(M)$ is non-vanishing.

The objective of the influence maximization problem is to find a targeted population of a specified size $k > 0$ that maximizes the expected influence, but the problem statement says nothing about the best choice for the value of k . We may, for example, choose a larger k than is necessary to produce the same value for expected influence. Or, we may choose a value for k even though it is clear that larger values will produce larger expected influence. For any non-vanishing set of mechanisms, we give a lower bound on the size of optimal k values for the influence maximization problem.

Theorem 7. For all $M \subseteq \mathcal{M}$, if M is non-vanishing and $k < \eta^+(r_{c(M)})$, then k is sub-optimal with respect to M .

Proof. Consider any non-vanishing set of mechanisms $M \subseteq \mathcal{M}$, $k > 0$, $\hat{\psi} \in \hat{\Psi}_{c(M)}$, and $O \in O_{M, \hat{\psi}}^{k*}$. Assume $k < \eta^+(r_{c(M)})$. There must exist a maximal connected component $X \in \text{Components}^+(r_{c(M)})$ such that $\mathcal{I}_X(O, M, \hat{\psi}) = 0$. It follows that we can find a set $X' \subseteq X$ such that $\mathcal{I}(O \cup X', M, \hat{\psi}) > \mathcal{I}(O, M, \hat{\psi})$ since M is a non-vanishing set of mechanisms. \square

6.7 Decomposition of Targeted Populations

Distinguishing between the expected influence over the complete population and that within each maximal connected component allows us to characterize the corresponding sets of optimal targeted populations. In this section, we show that optimal targeted populations for the complete population are composed of optimal targeted populations for each maximal connected component. We then show that the absence of bridges between the relations of each individual mechanism in some set of mechanisms leads to optimal targeted populations for the set of mechanisms that are composed of those for each individual mechanism.

Theorem 8. For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(M)}$, and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{c(M)})} O_{M, \hat{\psi}}^*(X)$, we have $(\bigcup_{i=1}^{\eta^+(r_{c(M)})} \vec{o}_i) \in O_{M, \hat{\psi}}^*$.

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(M)}$, and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{c(M)})} O_{M, \hat{\psi}}^*(X)$ with the factors ordered in accordance with an enumeration given by $\langle \text{Components}^+(r_{c(M)}) \rangle = \langle X_1, X_2, \dots, X_{\eta^+(r_{c(M)})} \rangle$. Let $O = \bigcup_{i=1}^{\eta^+(r_{c(M)})} \vec{o}_i$. From Theorem 6, we have $\mathcal{I}(O, M, \hat{\psi}) = \sum_{i=1}^{\eta^+(r_{c(M)})} \mathcal{I}_{X_i}(O, M, \hat{\psi}) = \sum_{i=1}^{\eta^+(r_{c(M)})} \mathcal{I}_{X_i}(\vec{o}_i, M, \hat{\psi})$. Assume $O \notin O_{M, \hat{\psi}}^*$. It follows that there must exist some $X_i \in \text{Components}^+(r_{c(M)})$ and $X \subseteq X_i$ such that $\mathcal{I}_{X_i}(\vec{o}_i, M, \hat{\psi}) <$

$\mathcal{I}_{X_i}(X, M, \hat{\psi})$. However, this contradicts our construction with $\vec{o}_i \in O_{M, \hat{\psi}}^*(X_i)$. We therefore have that $O \in O_{M, \hat{\psi}}^*$, which gives the desired result. \square

We restate Theorem 8 in terms of optimal targeted populations simply by assuming optimal targeted populations are chosen for each maximal connected component.

Corollary 4. *For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{\mathbf{c}(M)})} O_{M, \hat{\psi}}^{**}(X)$, we have $(\bigcup_{i=1}^{\eta^+(r_{\mathbf{c}(M)})} \vec{o}_i) \in O_{M, \hat{\psi}}^{**}$.*

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{\mathbf{c}(M)})} O_{M, \hat{\psi}}^{**}(X)$ with the factors ordered in accordance with an enumeration given by $\langle \text{Components}^+(r_{\mathbf{c}(M)}) \rangle = \langle X_1, X_2, \dots, X_{\eta^+(r_{\mathbf{c}(M)})} \rangle$. Let $O = \bigcup_{i=1}^{\eta^+(r_{\mathbf{c}(M)})} \vec{o}_i$. From Theorem 6, we have $\mathcal{I}(O, M, \hat{\psi}) = \sum_{i=1}^{\eta^+(r_{\mathbf{c}(M)})} \mathcal{I}_{X_i}(O, M, \hat{\psi}) = \sum_{i=1}^{\eta^+(r_{\mathbf{c}(M)})} \mathcal{I}_{X_i}(\vec{o}_i, M, \hat{\psi})$. We showed in Theorem 8 that $O \in O_{M, \hat{\psi}}^*$. Assume now that O is maximizing but not minimal maximizing, that is, $O \notin O_{M, \hat{\psi}}^{**}$. It follows that there must exist some $X_i \in \text{Components}^+(r_{\mathbf{c}(M)})$ and $X \subseteq X_i$ such that $|X| < |\vec{o}_i|$ and $\mathcal{I}_{X_i}(\vec{o}_i, M, \hat{\psi}) = \mathcal{I}_{X_i}(X, M, \hat{\psi})$. However, this contradicts the optimality of \vec{o}_i since we have by construction that $\vec{o}_i \in O_{M, \hat{\psi}}^{**}(X_i)$. We therefore have that $O \in O_{M, \hat{\psi}}^{**}$, which gives the desired result. \square

The extension of Corollary 4 to k -bounded targeted populations follows by requiring that at most k individuals are targeted in each maximal connected component, where k is component optimal. This results in at most $k \cdot \eta^+(r_{\mathbf{c}(M)})$ individuals being targeted for the entire population when using $M \subseteq \mathcal{M}$.

Corollary 5. *For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, component optimal $k > 0$ with respect to M , and $\vec{o} \in \prod_{X \in \text{Components}^+(r_{\mathbf{c}(M)})} O_{M, \hat{\psi}}^{k**}(X)$, we have $(\bigcup_{i=1}^{\eta^+(r_{\mathbf{c}(M)})} \vec{o}_i) \in O_{M, \hat{\psi}}^{n**}$, where $n = k \cdot \eta^+(r_{\mathbf{c}(M)})$.*

Proof. The claim follows directly from Corollary 4 and Lemma 9. \square

We have not yet made any assumptions about the connectivity between the population topologies as viewed by each different influence mechanism from some set of mechanisms. There might be bridges between different maximal connected components of the relations corresponding to each of the different influence mechanisms, and these bridges might facilitate the propagation of influence information between mechanisms that exert otherwise independent influence.

For some applications, we may wish to influence a population over relations that partition the population. For example, people that identify themselves as Democrats represent a population that is distinct from people that identify themselves as Republicans. There are different entertainment news programs that cater to the viewpoints of each of these political identities. As such, we may wish to use different mechanism in order to influence each of these disjoint populations. This case corresponds to one in which there are no bridges.

We now present results on sets of mechanisms for which there are no bridges. We show that the absence of any bridges allows us to determine an optimal targeted population for a set of mechanisms by taking the union of the optimal targeted populations for each individual mechanism in that set. For any $m \in \mathcal{M}$, if there is no bridge between r_m and $r_{m'}$ for all $m, m' \in M$ with $m \neq m'$, then, in following discussion, we say simply that there are no bridges over M .

Theorem 9. *For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{**}$, if there are no bridges over M , then $(\bigcup_{i=1}^{|M|} \vec{o}_i) \in \hat{O}_{M, \hat{\psi}}^{**}$.*

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{**}$. Assume there are no bridges over M . Let $O = \bigcup_{i=1}^{|M|} \vec{o}_i$. Since there are no bridges, we have from Corollary 1 that $O = \bigcup_{i=1}^{\eta^+(r_{c(M)})} \vec{o}_i$ for some $\vec{o} \in \prod_{X \in \text{Components}(r_{c(M)})} \hat{O}_{M, \hat{\psi}}^{**}(X)$. It follows from Corollary 4 and Lemma 6 that $O \in \hat{O}_{M, \hat{\psi}}^{**}$, which gives the desired result. \square

Corollary 6. For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, component optimal $k > 0$ with respect to M , and $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{k_m^{**}}$ where $k_m = k \cdot \eta(r_m)$, if there are no bridges over M , then $(\bigcup_{i=1}^{|M|} \vec{o}_i) \in \hat{O}_{M, \hat{\psi}}^{n^{**}}$, where $n = \sum_{m \in M} k_m$.

Proof. The claim follows directly from Theorem 9 and Corollary 5. \square

The composition of an optimal targeted population for a set of mechanisms from optimal targeted populations for each mechanism in the set leads us to a related composition using the expected influence functions. We show that the expected influence for some sets of mechanisms can be determined from the expected influence for each mechanism in the set.

Theorem 10. For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in \hat{O}_{M, \hat{\psi}}^{**}$, and $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{**}$ with the factors ordered in accordance with an enumeration $\langle M \rangle = \langle m_1, m_2, \dots, m_{|M|} \rangle$, if there are no bridges over M , then $\mathcal{I}(O, M, \hat{\psi}) = \sum_{i=1}^{|M|} \mathcal{I}(\vec{o}_i, \{m_i\}, \hat{\psi})$.

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{**}$ with the factors ordered in accordance with an enumeration $\langle M \rangle = \langle m_1, m_2, \dots, m_{|M|} \rangle$. Assume there are no bridges over M . Let $O = \bigcup_{i=1}^{|M|} \vec{o}_i$. From Theorem 9, we know that $O \in \hat{O}_{M, \hat{\psi}}^{**}$. From Theorem 6, we have $\mathcal{I}(O, M, \hat{\psi}) = \sum_{X \in \text{Components}(r_{c(M)})} \mathcal{I}_X(O, M, \hat{\psi})$ and $\mathcal{I}(\vec{o}_i, \{m_i\}, \hat{\psi}) = \sum_{X \in \text{Components}(r_{m_i})} \mathcal{I}_X(\vec{o}_i, \{m_i\}, \hat{\psi})$ for each $m_i \in M$. Since there are no bridges, we have from Corollary 1 and the definition of the convex combination method in Equation 4.2 that $\mathcal{I}_X(O, M, \hat{\psi}) = \mathcal{I}_X(\vec{o}_i, \{m_i\}, \hat{\psi})$ for each $m_i \in M$ and $X \in \text{Components}(r_{m_i})$. It follows that

$$\begin{aligned} \mathcal{I}(O, M, \hat{\psi}) &= \sum_{X \in \text{Components}(r_{c(M)})} \mathcal{I}_X(O, M, \hat{\psi}) \\ &= \sum_{i=1}^{|M|} \sum_{X \in \text{Components}(r_{m_i})} \mathcal{I}_X(\vec{o}_i, \{m_i\}, \hat{\psi}) \\ &= \sum_{i=1}^{|M|} \mathcal{I}(\vec{o}_i, \{m_i\}, \hat{\psi}). \end{aligned}$$

□

Corollary 7. For all $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, component optimal $k > 0$ with respect to M , $\vec{o} \in \prod_{m \in M} \hat{O}_{\{m\}, \hat{\psi}}^{k_m^{**}}$ with the factors ordered in accordance with an enumeration $\langle M \rangle = \langle m_1, m_2, \dots, m_{|M|} \rangle$ where $k_m = k \cdot \eta(r_m)$, and $O \in \hat{O}_{M, \hat{\psi}}^{n^{**}}$ where $n = \sum_{m \in M} k_m$, if there are no bridges over M , then $\mathcal{I}(O, M, \hat{\psi}) = \sum_{i=1}^{|M|} \mathcal{I}(\vec{o}_i, \{m_i\}, \hat{\psi})$.

Proof. The claim follows directly from Theorem 10 and Corollary 6. □

6.8 Independence of Influence on Selection of Targeted Population

Under certain conditions, the Markov chains that express the underlying probabilistic state transitions in our model may exhibit behavior that trivializes solutions to the influence maximization problem. We relate well-known properties of Markov chains to properties of our model (e.g., influence neighborhoods, maximizing targeted populations) under which the expected influence is independent of the choice of targeted population. Knowledge of this independence can save considerable time trying to find an optimal targeted population.

Theorem 11. For any $M \subseteq \mathcal{M}$ and Markov chain $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$, if there exists $x \in \mathcal{X}$ such that $\delta_{\mathbf{c}(M)}(x) = \emptyset$, then $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$ is reducible.

Proof. Assume a set of influence mechanisms $M \subseteq \mathcal{M}$ and corresponding Markov chain $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$. Let $x \in \mathcal{X}$ be an individual such that $\delta_{\mathbf{c}(M)}(x) = \emptyset$. It follows from Equation 3.2 that $\mu_{\mathbf{c}(M)}^x(\emptyset)(\hat{\psi}' | \hat{\psi}) = 0$ for all $\hat{\psi}, \hat{\psi}' \in \hat{\Psi}_{\mathbf{c}(M)}$ such that $\hat{\psi} \neq \hat{\psi}'$. From Equation 3.3, we know that not every state is reachable from every state. This gives the claimed result. □

Theorem 12. For any $X' \subseteq X \subseteq \mathcal{X}$, $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, and Markov chain $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$, if $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$ is irreducible and aperiodic, then $|\mathcal{I}(X, M, \hat{\psi}) - \mathcal{I}(X', M, \hat{\psi})| = 0$.

Proof. The claim follows directly from the irreducibility and aperiodicity of $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$ and its long-term independence from any initial state distribution $\pi_{\mathbf{c}(M)}^0$. \square

Corollary 8. For any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathbf{c}(M)}$, and Markov chain $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$, if $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$ is irreducible and aperiodic, then $O_{M, \hat{\psi}}^* = \text{Pwr}(\mathcal{X})$.

Proof. The claim follows directly from Theorem 12. \square

Corollary 9. For any $M \subseteq \mathcal{M}$ and Markov chain $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$, if $(\hat{\psi}_{\mathbf{c}(M)}^t)_{t \geq 0}$ is irreducible and aperiodic, then any $k > 0$ is optimal with respect to M .

Proof. The claim follows directly from Corollary 8. \square

Chapter 7

Analysis of Influence Mechanisms

In this chapter, we characterize the behavior of the example types of influence mechanisms from Section 3.7 using mechanism properties that we have defined throughout this dissertation. We provide examples of information propagation when using one and two influence mechanisms from which we make observations about their mechanism properties. We use these examples to address the question of whether a single mechanism will give more or less expected influence than two mechanisms.

The question of whether multiple mechanisms will result in an increase in expected influence leads us to define two types of interactions between different mechanisms in terms of their impact on expected influence. These mechanism interactions are not only useful on their own by making clear any benefits offered by using multiple mechanisms, but they may also prove useful in the pursuit of stronger notions of mechanism interactions that we discuss in Chapter 8.

We conclude this chapter with an example construction of the transition matrices induced by two different influence mechanism measures. We show how the transition matrices induced by the individual transition measures are used to construct the population transition matrix.

This is followed by a construction of the individual and population transition matrices resulting from the uniform convex combination of the two influence mechanisms. We make use of the example matrices by showing how the expected influence is calculated for a specific targeted population.

7.1 Mechanism Interactions

In Chapter 6, we showed how various mechanism properties (e.g., non-vanishing influence, population monotonicity) and the population structure impact the determination of optimal targeted populations and, as a result, better inform us about the possible solutions for the influence maximization problem. In addition to their effects on optimal targeted populations, it is of similar interest to better understand how multiple mechanisms affect change in the expected influence over using any single mechanism in isolation. To this end, we give a formal statement of the types of interactions between mechanisms that lead to increases or decreases in expected influence.

7.1.1 Mechanism Interference

Although mechanism properties such as non-vanishing influence and the existence of bridges have been instrumental in stating some of our results, they alone do not specify in general whether any given set of mechanisms will provide a larger expected influence than any of its subsets. When using multiple mechanisms, we may have that the different mechanisms exhibit a synergistic effect when used in combination. We refer to such synergy as the interference type of sets of mechanisms.

Consider the authoritarian and group conformity mechanisms used for the example in Section 1.1 over the relations *is-coworker-of* and *is-friend-of*, respectively. For the specified tar-

geted population $\{x_7\}$, we showed that using both mechanisms and their corresponding relations leads to a greater spread of information throughout the population than is achieved using only an authoritarian mechanism over the relation *is-coworker-of*. We now formalize this kind of interference type in the following definitions.

Definition 10. If $\mathcal{I}(O, M, \hat{\psi}) \leq \mathcal{I}(O', M' \cup M, \hat{\psi})$ for all $k > 0$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M \cup M', \hat{\psi}}^{k*}$, then we say that M' positively interferes with M .

Definition 11. If $\mathcal{I}(O, M, \hat{\psi}) \geq \mathcal{I}(O', M' \cup M, \hat{\psi})$ for all $k > 0$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M \cup M', \hat{\psi}}^{k*}$, then we say that M' negatively interferes with M .

Definition 12. If $\mathcal{I}(O, M, \hat{\psi}) = \mathcal{I}(O', M' \cup M, \hat{\psi})$ for all $k > 0$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M \cup M', \hat{\psi}}^{k*}$, then M' and M are called non-interfering. If M' and M are not non-interfering, then they are called interfering.

A set of mechanisms $M \subseteq \mathcal{M}$ is called a *set of positively (negatively) interfering mechanisms* if each $M' \subseteq M$ positively (negatively) interferes with each $M'' \subseteq M$ whenever $M' \not\subseteq M''$. A set of mechanisms $M \subseteq \mathcal{M}$ is called a *set of non-interfering mechanisms* if each $M', M'' \subseteq M$ are non-interfering.

We further characterize interference types of mechanisms in terms of homogeneity. We say that a set of mechanisms M is *interference type homogeneous* if it is either a set of positively interfering mechanisms, a set of negatively interfering mechanisms, or a set of non-interfering mechanisms. Otherwise, we refer to M as *interference type heterogeneous*.

7.1.2 Mechanism Monotonicity

As with population monotonicity, in which expected influence using some set of mechanisms monotonically increases/decreases as we add more targeted individuals, there exist sets of

mechanisms that are similarly monotonic, in that, the expected influence monotonically increases/decreases as we include each additional mechanism from the set of mechanisms. We call this property mechanism monotonicity.

The expected influence function \mathcal{I} is *non-decreasing M -monotone* if and only if for all $M' \subseteq M'' \subseteq M$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, $k > 0$, $O \in O_{M', \hat{\psi}}^{k*}$, and $O' \in O_{M'', \hat{\psi}}^{k*}$, we have $\mathcal{I}(O, M', \hat{\psi}) \leq \mathcal{I}(O', M'', \hat{\psi})$. The expected influence function \mathcal{I} is *non-increasing M -monotone* if and only if for all $M' \subseteq M'' \subseteq M$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, $k > 0$, $O \in O_{M', \hat{\psi}}^{k*}$, and $O' \in O_{M'', \hat{\psi}}^{k*}$, we have $\mathcal{I}(O, M', \hat{\psi}) \geq \mathcal{I}(O', M'', \hat{\psi})$. If strict inequality holds, then we say simply that \mathcal{I} is increasing (or decreasing) M -monotone. We sometimes refer to \mathcal{I} as *M -monotone* if it is either non-decreasing or non-increasing M -monotone.

We capture a relation between interference type and mechanism monotonicity by showing that interference type homogeneity implies mechanism monotonicity.

Theorem 13. *For any $M \subseteq \mathcal{M}$, if M is a set of positively interfering mechanisms, then the expected influence function \mathcal{I} is non-decreasing M -monotone.*

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, and $k > 0$. Assume M is a set of positively interfering mechanisms. For any $M', M'' \subseteq M$, $O \in O_{M', \hat{\psi}}^{k*}$, and $O' \in O_{M' \cup M'', \hat{\psi}}^{k*}$, we have $\mathcal{I}(O, M', \hat{\psi}) \leq \mathcal{I}(O', M' \cup M'', \hat{\psi})$. By letting $M''' = M' \cup M''$, we have $\mathcal{I}(O, M', \hat{\psi}) \leq \mathcal{I}(O', M''', \hat{\psi})$, which satisfies our definition of non-decreasing M -monotonicity. \square

Theorem 14. *For any $M \subseteq \mathcal{M}$, if M is a set of negatively interfering mechanisms, then the expected influence function \mathcal{I} is non-increasing M -monotone.*

Proof. Consider any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, and $k > 0$. Assume M is a set of negatively interfering mechanisms. For any $M', M'' \subseteq M$, $O \in O_{M', \hat{\psi}}^{k*}$, and $O' \in O_{M' \cup M'', \hat{\psi}}^{k*}$, we have $\mathcal{I}(O, M', \hat{\psi}) \geq \mathcal{I}(O', M' \cup M'', \hat{\psi})$. By letting $M''' = M' \cup M''$, we have $\mathcal{I}(O, M', \hat{\psi}) \geq \mathcal{I}(O', M''', \hat{\psi})$, which satisfies our definition of non-increasing M -monotonicity. \square

Theorem 15. *For any $M \subseteq \mathcal{M}$, if M is a non-interfering set, then the expected influence function \mathcal{I} is M -monotone.*

Proof. The claim follows directly from the definitions of sets of non-interfering mechanisms and non-increasing/decreasing M -monotonicity. \square

Theorem 16. *For any $M \subseteq \mathcal{M}$, if M is a homogeneous set, then the expected influence function \mathcal{I} is M -monotone.*

Proof. The claim follows directly from Theorems 13, 14, and 15. \square

Our interest in the examples that follow is on the notion of interference type. However, we note the applicability of mechanism monotonicity to the stronger notions of mechanism interactions that motivate the discussions in the following Chapter 8.

7.2 Examples of Analysis

We now provide examples of analysis within our framework using some of the most widely studied types of influence: authoritarian, contagion, group conformity, and voter model. We begin with an example using only a single authoritarian type of influence mechanism. We then consider the mechanism interference when including a contagion mechanism in addition an authoritarian mechanism. For each of the types of mechanisms discussed, we prove some of the properties that it maintains (e.g., non-vanishing, population monotonicity).

7.2.1 Authoritarian Mechanism

The simplest example of analysis is the one in which we have only a single influence mechanism. This example corresponds to related influence models that are also restricted only to a

single influence mechanism (e.g., voter models, Asavathiratham’s Influence Model, and cascade models).

Consider once again the population $\mathcal{X} = \{x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8\}$ from the example in Section 1.1 (depicted in Figure 1.1). In this example, we assume an authoritarian mechanism *Auth* with a corresponding mechanism relation $r_{Auth} = is-coworker-of$. As we stated in Section 3.7.4, the indices of individuals in the individual enumeration $\langle \mathcal{X} \rangle$ correspond also to the level of authority assigned by the authority ordering \succ^a , that is, $x_i \succ^a x_j$ if and only if $i > j$. For simplicity, we assume $\hat{\Psi}_{Auth} = \{0, 1\}$ and sometimes refer to the mechanism states directly by their binary value.

Let $k = 1$ so that we are allowed to target only a single individual. Assume a targeted population $X = \{x_7\}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ where $\hat{\psi}[Auth] = 1$. Since we are dealing with binary mechanism states, this gives an initial state distribution such that $\pi_{Auth}^0(\hat{\psi}') = 1$ if and only if $\hat{\psi}'(x_7) = 1$ and $\hat{\psi}'(x_i) = 0$ for all $i \neq 7$. Our objective is to determine the expected number of influenced individuals given by $\mathcal{I}(\{x_7\}, Auth, \hat{\psi})$.

Each x_i with $x_7 \in \delta_{Auth}(x_i)$, except for x_4 (since $x_8 \in \delta_{Auth}(x_4)$), will transition from state 0 to state 1 with a probability of 1 since x_7 is more authoritative than each of the individuals to which it is related through *is-coworker-of* (see Equation 3.8). However, there is zero probability of making such a transition for each $x_i \in \{x_1, x_2, x_3, x_4, x_8\}$. This is due to the population structure under *is-coworker-of*, which requires the influence information isolated on one side of the cut-edge (x_8, x_4) to traverse this cut-edge in order to reach over half of the population. Since x_8 will never be influenced by x_4 using *Auth* over *is-coworker-of*, we are incapable of influencing more than half of the population when using targeted population $\{x_7\}$. With such deterministic state transitions under *Auth*, we have $\mathcal{I}(\{x_7\}, Auth, \hat{\psi}) = 3$ for all $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ (see Appendix A.1 for the individual transition matrices).

In general, no $x_i \in \{x_1, x_2, x_3, x_4, x_8\}$ will be influenced to adopt state 1 unless x_8 and x_3

are in the targeted population. Influencing the complete population \mathcal{X} to adopt state 1 using influence mechanism $Auth$ requires that x_3 (since it is disconnected under *is-coworker-of*), x_8 (since it commands the most authority in \mathcal{X}), and x_7 are in the targeted population. In other words, there is zero probability of influencing more than half of the population \mathcal{X} using $Auth$ when $k = 1$. This gives an optimal targeted population $\{x_3, x_7, x_8\} \in O_{Auth, \hat{\psi}}^{**}$ for all $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, where $\mathcal{I}(\{x_3, x_7, x_8\}, Auth, \hat{\psi}) = 8$.

We make a simple observation that, in terms of the underlying Markov chain, it is clear that not every state is reachable from every other state. This gives a reducible Markov chain on the population state space and the long-term behavior is therefore dependent on the initial state distribution.

This example brings to light two properties of an authoritarian influence mechanism. First, there is a chance of having zero expected influence when we target any individual that has a more authoritative neighboring individual. Second, the expected influence can not decrease as we target more individuals.

Theorem 17. *An authoritarian mechanism $Auth$ exerts vanishing influence whenever there exists $x \in \mathcal{X}$ such that $x' \succ^a x$ for all $x' \in \delta_{Auth}(x)$.*

Proof. Let $x \in \mathcal{X}$ be an individual such that $x' \succ^a x$ for all $x' \in \delta_{Auth}(x)$. It follows that $\mathcal{I}(\{x\}, Auth, \hat{\psi}) = 0$ for all $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ since x is incapable of influencing any of its neighbors and will itself be influenced to adopt a state other than $\hat{\psi}$. \square

Theorem 18. *An authoritarian mechanism $Auth$ is non-decreasing population monotonic.*

Proof. Without loss of generality, we assume any relation $r_{Auth} \in \mathcal{R}$ over which an authoritarian mechanism $Auth$ can exert influence consists of a single maximal connected component, i.e., $\eta^+(r_{Auth}) = 1$. Assume $Auth$ is not non-decreasing population monotonic. For any $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, there must exist $X, X' \subseteq \mathcal{X}$ such that $\mathcal{I}(X, Auth, \hat{\psi}) > \mathcal{I}(X \cup X', Auth, \hat{\psi})$.

If there does not exist $x \in X'$ such that $x \in \delta_{Auth}(x')$ for some $x' \in \mathcal{X}$ and $x \succ^a x'$, then $\mathcal{I}(X, Auth, \hat{\psi}) \leq \mathcal{I}(X \cup X', Auth, \hat{\psi})$, contradicting our assumption. Otherwise, let $x \in X'$ be an individual such that $x \in \delta_{Auth}(x')$ for some $x' \in \mathcal{X}$ and $x \succ^a x'$. It follows that $\mathcal{I}(X, Auth, \hat{\psi}) \leq \mathcal{I}(X \cup X', Auth, \hat{\psi})$, which also contradicts our assumption that *Auth* is not non-decreasing population monotonic. Thus, there is no X' that will result in a decrease in expected influence. \square

7.2.2 Authoritarian and Contagion Mechanisms

The use of multiple influence mechanisms for information propagation can have a substantial effect on the expected influence in comparison to the expected influence under only a single influence mechanism, especially when the multiple influence mechanisms apply to different relations. In this example, we show that we may be able to influence a larger number of individuals in a population through the use of multiple mechanisms than when we use only a single mechanism.

We use the same population $\mathcal{X} = \{x_1, x_2, x_3, x_4, x_5, x_6, x_7, x_8\}$ from the previous example, but we now use two mechanisms $M = \{Auth, Cont\}$. The relations we assume here for each mechanism are $r_{Auth} = is-coworker-of$ and $r_{Cont} = is-friend-of$ (see Figure 1.1). Note that we can apply different types of mechanisms to the same relation (e.g., contagion and group conformity types of mechanisms can be applied to *is-friend-of*). For simplicity, we assume $\hat{\Psi}_{Auth} = \hat{\Psi}_{Cont} = \{0, 1\}$. One natural question to ask is: Does the expected number of influenced individuals increase when we utilize more influence mechanisms?

Before we proceed with our example, we demonstrate some properties of a contagion mechanism *Cont*. As we discussed in Section 6.6, a contagion mechanism exerts non-vanishing influence.

Theorem 19. *A contagion mechanism $Cont$ exerts non-vanishing influence.*

Proof. From the definition of μ_{Cont} in Equation 3.9, we have that each $x \in \mathcal{X}$ will change states with certainty only if a neighboring individual has been influenced to adopt the specified influence information. We therefore have for all $X \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ that $\mathcal{I}(X, Cont, \hat{\psi}) \geq |X|$. It follows that $Cont$ exerts non-vanishing influence. \square

From Theorems 19 and 7, we know that any optimal targeted population must contain at least one individual from each maximal connected component. This can be used to inform our choice of k for the influence maximization problem since any $k < \eta^+(r_{Cont})$ will give a sub-optimal solution.

Theorem 20. *A contagion mechanism $Cont$ is non-decreasing population monotonic.*

Proof. It follows directly from the deterministic transitions of μ_{Cont} defined in Equation 3.9 that there can not exist a targeted population $X \subseteq \mathcal{X}$ and a subset $X' \subseteq X$ such that targeting X' results in a larger expected influence than that given by targeting X . \square

Continuing with the previous example using an authoritarian mechanism $Auth$, assume $k = 1$ and we have the same targeted population $\{x_7\}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ where $\hat{\psi}[Auth] = 1$. We have already shown in Section 7.2.1 that such a targeted population is incapable of influencing more than half of the population using $Auth$ over the relation *is-coworker-of*.

The different population structure given by relation *is-friend-of* allows x_3 to be influenced and does not have a cut-edge as under *is-coworker-of* that restricts the pathways for the spread of information. This suggests $\mathcal{I}(\{x_7\}, Auth, \hat{\psi}) \leq \mathcal{I}(\{x_7\}, \{Auth, Cont\}, \hat{\psi})$. We could compute these values directly for the given targeted population, but instead we are able to make a stronger claim by showing that $Cont$ positively interferes with $Auth$.

Theorem 21. *A contagion mechanism Cont positively interferes with an authoritarian mechanism Auth.*

Proof. Let $M = \{\text{Auth}, \text{Cont}\}$ and assume any $r_{\text{Auth}}, r_{\text{Cont}} \in \mathcal{R}$, $\hat{\psi} \in \hat{\Psi}_{c(M)}$, $k > 0$, $O \in O_{\text{Auth}, \hat{\psi}}^{k*}$, and $O' \in O_{M, \hat{\psi}}^{k*}$. By definition of the convex combination method in Equation 4.2, if $x \in \text{Isolates}(r_{\text{Auth}})$ for all $x \in \mathcal{X}$, then $\mathcal{I}(O, \text{Auth}, \hat{\psi}) \leq \mathcal{I}(O', M, \hat{\psi})$. Otherwise, there must exist a non-isolated maximal connected component $X \in \text{Components}(r_{\text{Auth}})$. If $x \in \text{Isolates}(r_{\text{Cont}})$ for all $x \in X$, then $X \in \text{Components}(r_{c(M)})$ and from Equation 4.2 we have $\mathcal{I}_X(O, \text{Auth}, \hat{\psi}) = \mathcal{I}_X(O', M, \hat{\psi})$. Otherwise, there must exist $X' \in \text{Components}(r_{\text{Cont}})$ such that $X'' = (X \cup X') \in \text{Components}(r_{c(M)})$. For any targeted individual $x \in X'' \cap O'$, if $x \in \delta_{\text{Auth}}(x')$, $x \succ^a x'$, and $x = \max_{\succ^a}(x'' \in \delta_{\text{Auth}}(x'))$, then the probability of x influencing x' is at least as great under $c(M)$ as under *Auth* alone since the influence probability under *Cont* is either certain (i.e., probability 1) or does not contribute at all to $c(M)$ (e.g., $\delta_{\text{Cont}}(x') = \emptyset$). If $x \in \delta_{\text{Auth}}(x')$, $x \succ^a x'$, and $x \neq \max_{\succ^a}(x'' \in \delta_{\text{Auth}}(x'))$, then the probability of x influencing x' is zero under *Auth* and, again, the probability of x influencing x' under *Cont* is either certain (i.e., probability 1) or does not contribute at all to $c(M)$ (e.g., $\delta_{\text{Cont}}(x') = \emptyset$). For each of these cases for the components in $\text{Components}(r_{c(M)})$, we have that the probability of x influencing x' is at least as great under $c(M)$ as under *Auth* alone. We therefore have from Theorem 6 that $\mathcal{I}(O, \text{Auth}, \hat{\psi}) \leq \mathcal{I}(O', M, \hat{\psi})$, which gives the desired result. \square

We note that the verification of our claimed improvement to expected influence under both types of mechanisms in Theorem 21 does not rely on computation. Instead, we make use of our formal framework for proving the claim. This highlights one of the benefits of using a framework such as the one we have developed.

r_{Conf} :

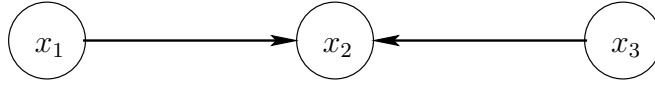


Figure 7.1: Depiction of a relation over which a group conformity mechanism $Conf$ can exert influence.

7.2.3 Group Conformity and Voter Model Mechanisms

We turn our attention now to group conformity and voter model types of mechanisms. We state conditions under which both types of mechanisms exert vanishing influence and show that they are non-decreasing population monotonic. Establishing these properties should prove useful in any future analysis of these types of mechanisms within our model. We use these same types of mechanisms in the following Section 7.3 for an example computation.

Theorem 22. *There exist relations over which a group conformity mechanism $Conf$ exerts vanishing influence.*

Proof. Consider the population $X = \{x_1, x_2, x_3\}$ depicted in Figure 7.1 that is connected by a relation r_{Conf} over which $Conf$ can exert influence. Let $\{x_2\}$ be a targeted population and $\hat{\psi}[Conf] = 1$ be the relevant influence information, where $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ and we are assuming $\hat{\Psi}_{Conf} = \{0, 1\}$. We have $\mathcal{I}_X(\{x_2\}, Conf, \hat{\psi}) = 0$, which shows that $Conf$ is non-vanishing over r_{Conf} (see Appendix A.2 for the transition matrices used to determine the expected influence).

□

Theorem 23. *There exist relations over which a voter model mechanism $Voter$ exerts vanishing influence whenever p_x is uniform for all $x \in \mathcal{X}$.*

Proof. The claim follows directly from Theorems 1 and 22.

□

The expected influence when using a group conformity mechanism $Conf$ can not decrease as we add more individuals to a targeted population.

Theorem 24. *A group conformity mechanism $Conf$ is non-decreasing population monotonic.*

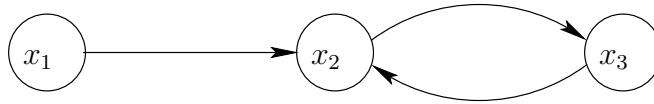
Proof. We prove the claim by contradiction. Assume a group conformity mechanism $Conf$ is not non-decreasing population monotonic. There must exist $X, X' \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ such that $\mathcal{I}(X, Conf, \hat{\psi}) > \mathcal{I}(X \cup X', Conf, \hat{\psi})$. From Equation 3.5, we have that the probability of influencing any $x \in \mathcal{X}$ grows in direct proportion with the number of neighbors $x' \in \delta_{Conf}(x)$ that have already been influenced to adopt $\hat{\psi}[Conf]$, regardless of the type of relation $r_{Conf} \in \mathcal{R}$. Thus, each additional targeted individual can not decrease the probability of influencing any other individual to adopt $\hat{\psi}[Conf]$. It follows that there does not exist $X, X' \subseteq \mathcal{X}$ such that $\mathcal{I}(X, Conf, \hat{\psi}) > \mathcal{I}(X \cup X', Conf, \hat{\psi})$, which contradicts our assumption. We conclude that $Conf$ is non-decreasing population monotonic. \square

Showing that a voter model mechanism $Voter$ is non-decreasing population monotonic follows from Theorem 24 by generalizing the proof to include non-uniform weighting distributions.

Theorem 25. *A voter model mechanism $Voter$ is non-decreasing population monotonic.*

Proof. The claim follows directly from Theorems 1 and 24 if we assume uniform neighborhood distributions p_x for each $x \in \mathcal{X}$. Otherwise, assume p_x is not necessarily uniform for each $x \in \mathcal{X}$. From Equation 3.7, the probability of influencing any $x \in \mathcal{X}$ increases as the number of influenced neighboring individuals increases, regardless of the type of relation $r_{Conf} \in \mathcal{R}$. Thus, there does not exist an assignment of distributions p_x to each $x \in \mathcal{X}$ such that $\mathcal{I}(X, Voter, \hat{\psi}) > \mathcal{I}(X \cup X', Voter, \hat{\psi})$ for any $X, X' \subseteq \mathcal{X}$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. We conclude that $Voter$ is non-decreasing population monotonic. \square

r_{Conf} :



r_{Voter} :

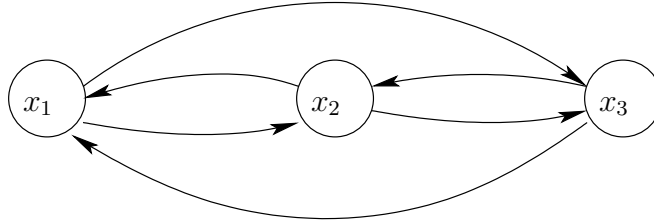


Figure 7.2: Graphs of r_{Conf} and r_{Voter} representing two different relations over the same set of individuals.

7.3 Example Computation

In previous examples, we used a population consisting of eight individuals, which was sufficient for recognizing some of the benefits of our model. However, the transition matrices resulting from a population of this size are too large to include in these examples. Assuming only binary states, we have 256×256 population transition matrices. Because of this, we present an example of computation for a smaller population of only three individuals.

Let $\mathcal{X} = \{x_1, x_2, x_3\}$ be a population consisting of three individuals. We use a group conformity and voter model mechanism with $M = \{Conf, Voter\}$, and assume their corresponding mechanism relations for this example are given by r_{Conf} and r_{Voter} (see depiction in Figure 7.2). For simplicity, assume $\hat{\Psi}_{Conf} = \hat{\Psi}_{Voter} = \{0, 1\}$, which gives a combined state space $\hat{\Psi}_{c(M)} = \{0, 1\}$.

Listed below are the individual transition matrices for each individual in the population that are conditioned on each neighborhood state, and the population transition matrix of the

corresponding population transition measure. The population transition matrix $[\mu_{Conf}]$ contains the transition probabilities using *Conf* and $[\mu_{Voter}]$ contains the transition probabilities using *Voter*. The canonical form for each of these matrices is such that the columns (and rows) are labeled in increasing value according to the numerical interpretation of the binary states. The population states are assumed to be consistent with the individual enumeration order $\langle \mathcal{X} \rangle = \langle x_1, x_2, x_3 \rangle$. For example, the rows of the population transition matrices $[\mu_{Conf}]$ and $[\mu_{Voter}]$ are labeled 000, 001, 010, \dots , 110, 111.

The individual transition matrices and population transition matrix under *Conf* are as follows:

$$[\mu_{Conf}^{x_1}(\emptyset)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Conf}^{x_2}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{Conf}^{x_2}(01)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix}$$

$$[\mu_{Conf}^{x_2}(10)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix} \quad [\mu_{Conf}^{x_2}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Conf}^{x_3}(0)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{Conf}^{x_3}(1)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Conf}] = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0.5 & 0 & 0.5 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.5 & 0 & 0.5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.5 & 0 & 0.5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0.5 & 0 & 0.5 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

A voter model mechanism requires that we specify a neighborhood weight distribution for each individual. Let $p_{x_1} = (0.2, 0.8)$, $p_{x_2} = (0.5, 0.5)$, and $p_{x_3} = (0.4, 0.6)$. We have, for example, that $p_{x_1}(x_3) = 0.8$ and $p_{x_3}(x_1) = 0.4$. Provided these neighborhood weight distributions, the individual transition matrices and population transition matrix under *Voter* are as follows:

$$[\mu_{Voter}^{x_1}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{Voter}^{x_1}(01)] = \begin{bmatrix} 0.2 & 0.8 \\ 0.2 & 0.8 \end{bmatrix}$$

$$[\mu_{Voter}^{x_1}(10)] = \begin{bmatrix} 0.8 & 0.2 \\ 0.8 & 0.2 \end{bmatrix} \quad [\mu_{Voter}^{x_1}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Voter}^{x_2}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{Voter}^{x_2}(01)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix}$$

$$[\mu_{Voter}^{x_2}(10)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix} \quad [\mu_{Voter}^{x_2}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Voter}^{x_3}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{Voter}^{x_3}(01)] = \begin{bmatrix} 0.4 & 0.6 \\ 0.4 & 0.6 \end{bmatrix}$$

$$[\mu_{Voter}^{x_3}(10)] = \begin{bmatrix} 0.6 & 0.4 \\ 0.6 & 0.4 \end{bmatrix} \quad [\mu_{Voter}^{x_3}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Voter}] = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0.1 & 0 & 0.1 & 0 & 0.4 & 0 & 0.4 & 0 \\ 0.32 & 0.48 & 0 & 0 & 0.08 & 0.12 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.2 & 0.3 & 0.2 & 0.3 \\ 0.3 & 0.2 & 0.3 & 0.2 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0.12 & 0.08 & 0 & 0 & 0.48 & 0.32 \\ 0 & 0.4 & 0 & 0.4 & 0 & 0.1 & 0 & 0.1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

Each entry in the population transition matrices $[\mu_{Conf}]$ and $[\mu_{Voter}]$ is computed by taking the product of the probability of each individual making the transition corresponding to their respective individual transition matrix, given the corresponding neighborhood state. Specifically

(see Equation 3.3), for all $\hat{\psi}, \hat{\psi}' \in \phi_{\mathbf{c}(M)}(\Psi_{\mathcal{X}})$ we have

$$\mu_{\mathbf{c}(M)}(\hat{\psi})(\hat{\psi}' | \hat{\psi}) = \prod_{x \in \mathcal{X}} \mu_{\mathbf{c}(M)}^x(\hat{\psi}(\delta_{\mathbf{c}(M)}(x)))(\hat{\psi}'(x) | \hat{\psi}(x)). \quad (7.1)$$

For example, the entry $[\mu_{\text{Voter}}]_{2,1} = \mu_{\text{Voter}}(010 | 001) = 0.48$ gives the probability of the population \mathcal{X} transitioning from state 001 to state 010 under the influence of *Voter*. The neighborhood states that condition the transitions for each individual are represented by the population state 001 since $\delta_{\text{Voter}}(\mathcal{X}) \subseteq \mathcal{X}$. For each individual, we have $\mu_{\text{Voter}}^{x_1}(10)(0 | 0) = 0.8$, $\mu_{\text{Voter}}^{x_2}(00)(0 | 1) = 1$, and $\mu_{\text{Voter}}^{x_3}(01)(1 | 0) = 0.6$. The population transition probability is given by

$$[\mu_{\text{Voter}}]_{2,1} = [\mu_{\text{Voter}}^{x_1}(10)]_{0,0} \cdot [\mu_{\text{Voter}}^{x_2}(00)]_{1,0} \cdot [\mu_{\text{Voter}}^{x_3}(01)]_{0,1} = 0.48. \quad (7.2)$$

The individual transition matrices and the resulting population transition matrix under both types of influence, denoted by $[\mu_{\mathbf{c}(M)}]$, are provided below. Each entry in the population transition matrix $[\mu_{\mathbf{c}(M)}]$ is computed using Equation 4.2.

$$[\mu_{\mathbf{c}(M)}^{x_1}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_1}(01)] = \begin{bmatrix} 0.2 & 0.8 \\ 0.2 & 0.8 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}^{x_1}(10)] = \begin{bmatrix} 0.8 & 0.2 \\ 0.8 & 0.2 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_1}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}^{x_2}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_2}(01)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}^{x_2}(10)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_2}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}^{x_3}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_3}(01)] = \begin{bmatrix} 0.2 & 0.8 \\ 0.2 & 0.8 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}^{x_3}(10)] = \begin{bmatrix} 0.8 & 0.2 \\ 0.8 & 0.2 \end{bmatrix} \quad [\mu_{\mathbf{c}(M)}^{x_3}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{\mathbf{c}(M)}] = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0.1 & 0 & 0.1 & 0 & 0.4 & 0 & 0.4 & 0 \\ 0.16 & 0.64 & 0 & 0 & 0.04 & 0.16 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.1 & 0.4 & 0.1 & 0.4 \\ 0.4 & 0.1 & 0.4 & 0.1 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0.16 & 0.04 & 0 & 0 & 0.64 & 0.16 \\ 0 & 0.4 & 0 & 0.4 & 0 & 0.1 & 0 & 0.1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

The population transition matrices that we have presented appear to show some block struc-

ture. In terms of the underlying Markov chain, this indicates reducibility. We avoid diversion into rigorous formal analysis of any obvious structure of the example transition matrices since our objective here is to demonstrate how the transition matrices are computed for one or more influence mechanisms within the framework of our influence model.

Now that the transition matrices have been specified, let us consider the expected influence computation using the targeted population $\{x_1, x_2\}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(M)}$ where $\hat{\psi}[M] = 1$. The binary state space for each mechanism, and their combination, gives us a deterministic initial state distribution $\pi_{Conf}^0 = \pi_{Voter}^0 = \pi_{c(M)}^0 = \langle 0, 0, 0, 0, 0, 0, 1, 0 \rangle$. Using this initial state distribution, we use the computational software program *Mathematica* to find a stationary distribution for each of the population transition matrices as follows:

$$\begin{aligned}\pi_{Conf}^0 \cdot \lim_{t \rightarrow \infty} [\mu_{Conf}]^t &= \langle 0, 0, 0, 0, 0, 0, 0, 1 \rangle \\ \pi_{Voter}^0 \cdot \lim_{t \rightarrow \infty} [\mu_{Voter}]^t &= \langle 0.394737, 0, 0, 0, 0, 0, 0, 0.605263 \rangle \\ \pi_{c(M)}^0 \cdot \lim_{t \rightarrow \infty} [\mu_{c(M)}]^t &= \langle 0.384615, 0, 0, 0, 0, 0, 0, 0.615385 \rangle.\end{aligned}$$

We use these stationary distributions to compute expected influence by summing over all possible states, the probability of being in a state multiplied by the number of influenced individuals in that state (Equation 5.1). This gives us $\mathcal{I}(\{x_1, x_2\}, Conf, \hat{\psi}) = 3$, $\mathcal{I}(\{x_1, x_2\}, Voter, \hat{\psi}) = 3 * 0.605263 = 1.815789$, and $\mathcal{I}(\{x_1, x_2\}, \{Conf, Voter\}, \hat{\psi}) = 3 * 0.615385 = 1.846155$.

We can see for this particular example that using both *Conf* and *Voter* gives a small increase in the expected influence over using *Voter* alone. However, it is clear that *Conf* is able to influence the entire population and the inclusion of *Voter* significantly decreases the expected influence over using *Conf* alone. There is more analysis that can be done using this small example, but this presentation is sufficient for our intended demonstration in this section.

Chapter 8

Diminishing Influence

Much of our work has focused on targeted populations. In fact, analysis of expected influence under maximizing targeted populations forms the majority of the related work. Notable among these are the results presented in [27, 28] on their definition of expected influence with respect to sets of targeted populations. They show that adding any $x \in \mathcal{X}$ to a targeted population $X \subseteq \mathcal{X}$ gives at least as large of an increase to expected influence as adding x to any superset $X' \supseteq X$. We refer to this property as *targeted population submodularity*, where submodularity is less formally referred to as diminishing returns.

We do not dismiss the importance of targeted population submodularity, but the use of multiple mechanisms in our model motivates our focus in this dissertation on the submodularity of expected influence with respect to sets of mechanisms, which we refer to as *mechanism submodularity*. Our aim in establishing mechanism submodularity is to identify sets of mechanisms $M \subseteq \mathcal{M}$ such that the inclusion of each additional mechanism $m \in M$ to some $M' \subseteq M$ gives at least as large of an increase to expected influence as adding m to any superset $M'' \supseteq M'$. This is especially important if we assume that each additional mechanism comes with a cost, or that we may only use a certain number of mechanisms or relations.

There is a tremendous amount of literature that details the problems and complexity results of minimizing a submodular function (see [18]). Recent related works [27, 28] make use of an expected influence function that exhibits targeted population submodularity, and view the influence maximization problem as one of maximizing a submodular function. Whereas minimization of a submodular function has been shown to be solvable in polynomial time [21], maximizing a submodular function has been shown in related models to be NP-Hard by reduction to the optimization version of the *Set Cover* problem [27].

The work we describe in this chapter represents our preliminary investigations into the mechanism submodularity or diminishing returns of our expected influence function. Our goal is to find a simple and efficient method for determining mechanism submodularity or diminishing returns of expected influence relative to a given set of mechanisms. Unfortunately, we are unable to establish mechanism submodularity of expected influence for anything other than for one type of sets of mechanisms. Unlike targeted population submodularity results in related work [27] in which individuals are indistinguishable aside from their current state and relational position in a population, mechanisms exhibit a wide range of properties that must be taken into consideration in order to establish mechanism submodularity. This motivates our consideration of alternative approaches that might give us diminishing returns or at least identify the most effective influence mechanisms for maximizing expected influence.

Among the alternatives to mechanism submodularity that we consider are orderings over sets of mechanisms that exhibit diminishing returns, a component restricted version of such diminishing mechanism orderings, a local bias measure of mechanisms for inferring magnitudes of change in expected influence, and Principal Component Analysis. We show that each of these is plagued by one or more pitfalls that hinders its practical usefulness, and we conclude with an existing method for testing submodularity.

8.1 Mechanism Submodularity

We do not always have the full set of mechanisms \mathcal{M} at our disposal for spreading information throughout a population. Some of the mechanisms may be irrelevant for an application (e.g., useless mechanism relation) or may be known to produce undesirable effects (e.g., interference type). Still, however, we are interested in the mechanism submodularity for the subset of mechanisms of interest. This leads us to define mechanism submodularity relative to any given set of mechanisms. In order to simplify the presentation, we define

$$\Delta_{\hat{\psi}}^k(M, M') \stackrel{\text{def}}{=} \mathcal{I}(O, M, \hat{\psi}) - \mathcal{I}(O', M', \hat{\psi}) \quad (8.1)$$

for any $M, M' \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, $k > 0$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M', \hat{\psi}}^{k*}$.

Definition 13. *Given any $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$ and $M \subseteq \mathcal{M}$, we say that \mathcal{I} is mechanism submodular with respect to M , or M -submodular, if and only if $\Delta_{\hat{\psi}}^k(M' \cup \{m\}, M') \geq \Delta_{\hat{\psi}}^k(M'' \cup \{m\}, M'')$ for all $M' \subseteq M'' \subseteq M$, $m \in M \setminus M''$, and $k > 0$.*

We have defined several properties of mechanisms and sets of mechanisms and have used these to characterize the expected influence and corresponding sets of optimal targeted populations. Establishing mechanism submodularity with respect to some set of mechanisms requires knowledge of the ways in which each of the subsets will interact with each other. For sets of non-interfering mechanisms, the expected influence function is mechanism submodular.

Theorem 26. *For any $M \subseteq \mathcal{M}$, if M is a set of non-interfering mechanisms, then \mathcal{I} is M -submodular.*

Proof. Consider any set of non-interfering mechanisms $M \subseteq \mathcal{M}$. We have by definition of non-interference that $\Delta_{\hat{\psi}}^k(M', M'') = 0$ for all $M', M'' \subseteq M$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, and $k > 0$, which satisfies M -submodularity in Definition 13. \square

We expect that other properties of mechanisms, as well as other conditions on the mechanism properties we have already defined, will lead to more general mechanism submodularity results. Theorem 13 shows that positive interfering sets of mechanisms are mechanism monotonic, and we might be able to place additional constraints on such sets in order to achieve mechanism submodularity. Although mechanism submodularity under more general assumptions about the mechanisms would be of much interest, we turn our attention now to alternative notions of diminishing returns.

8.2 Mechanism Orderings

We define an ordering over a set of mechanisms such that each additional mechanism included in accordance with the ordering will give the largest possible increase in expected influence over the alternatives.

Definition 14. Let \succsim be any linear ordering over $M \subseteq \mathcal{M}$ with elements indexed such that $m_i \succsim m_{i+1}$ and $M_i = \bigcup_{l=1}^i \{m_l\}$. We say that \succsim is a *diminishing mechanism ordering* (under $k > 0$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$) if and only if $\Delta_{\hat{\psi}}^k(M_{i+1}, M_i) \geq \Delta_{\hat{\psi}}^k(M_{i+j+1}, M_{i+j})$ for all $i \geq 1$ and $j \geq 0$. We say that \succsim is a *non-decreasing* (or *non-increasing*) *monotonic mechanism ordering* (under $k > 0$ and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$) if and only if $\Delta_{\hat{\psi}}^k(M_{i+1}, M_i) \geq 0$ (or $\Delta_{\hat{\psi}}^k(M_{i+1}, M_i) \leq 0$) for all $i \geq 1$.

We introduce a simple greedy approach for constructing mechanism orderings. Let $\succsim_{\mathcal{I}}^{\hat{\psi}, k}$ denote the *greedy mechanism ordering* over $M \subseteq \mathcal{M}$ induced by \mathcal{I} using influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ and $k > 0$. We define $\succsim_{\mathcal{I}}^{\hat{\psi}, k}$ such that $m_i \succsim_{\mathcal{I}}^{\hat{\psi}, k} m_{i+1}$ if and only if $\Delta_{\hat{\psi}}^k(M_i \cup \{m_{i+1}\}, M_i) \geq \Delta_{\hat{\psi}}^k(M_i \cup \{m\}, M_i)$ for all $m \in M \setminus M_i$, where M_i is the set of mechanisms including and preceding m_i under a linearization of the ordering constructed thus far.

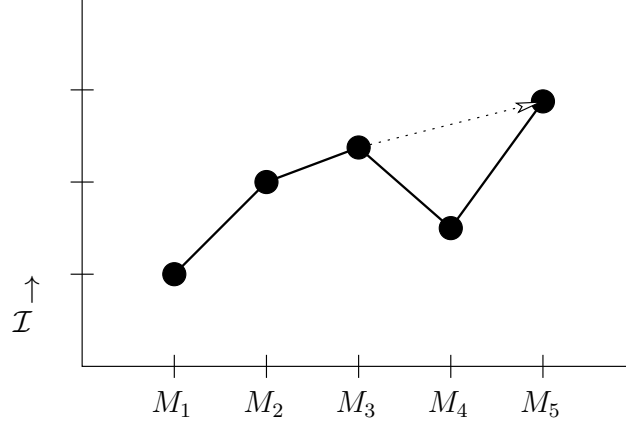


Figure 8.1: Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value.

A greedy mechanism ordering need not be a diminishing or monotonic mechanism ordering. To see this, consider a linearization $\succsim_{\mathcal{I}}^l$ of a greedy mechanism ordering $\succsim_{\mathcal{I}}^{\hat{\psi}, k}$ over $M = \{m_1, m_2, m_3, m_4, m_5\}$. Assume we have $m_1 \succsim_{\mathcal{I}}^l m_2 \succsim_{\mathcal{I}}^l m_3 \succsim_{\mathcal{I}}^l m_4 \succsim_{\mathcal{I}}^l m_5$ and that expected influence is non-decreasing everywhere except $\mathcal{I}(O', M_4, \hat{\psi}) < \mathcal{I}(O, M_3, \hat{\psi}) < \mathcal{I}(O', M_5, \hat{\psi})$ for any choice of $O \in O_{M_3, \hat{\psi}}^{k*}$, $O' \in O_{M_4, \hat{\psi}}^{k*}$, and $O'' \in O_{M_5, \hat{\psi}}^{k*}$. That is, we have a “dip” in expected influence with the inclusion of m_4 using a greedy mechanism ordering (see Figure 8.1).

We are able to avoid “dips” in a greedy mechanism ordering when using a homogeneous set of mechanisms (see Figures 8.2, 8.3, and 8.4).

Theorem 27. *For all $k > 0$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, if M is an interference-type homogeneous set, then any linearization of the greedy mechanism ordering $\succsim_{\mathcal{I}}^{\hat{\psi}, k}$ over M is a monotonic mechanism ordering.*

Proof. Consider any $k > 0$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. Without loss of generality, assume

M is a positively interfering set. Let M_i denote the set of all mechanisms preceding m_{i+1} in a linearization $\succsim_{\mathcal{I}}^l$ of the greedy mechanism ordering $\succsim_{\mathcal{I}}^{\hat{\psi},k}$. Since M is a set of positively interfering mechanisms, there does not exist $m \in M$ and $k > 0$ such that $\Delta_{\hat{\psi}}^k(M_i \cup \{m\}, M_i) < 0$. We conclude that $\succsim_{\mathcal{I}}^l$ is a (non-decreasing) monotonic mechanism ordering. \square

Corollary 10. *For all $k > 0$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, the existence of a monotonic linearization of the greedy mechanism ordering $\succsim_{\mathcal{I}}^{\hat{\psi},k}$ over M does not imply that M is an interference-type homogeneous set.*

Proof. Assume any $M \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $k > 0$. Without loss of generality, assume a non-decreasing monotonic linearization $\succsim_{\mathcal{I}}^l$ of the greedy mechanism ordering $\succsim_{\mathcal{I}}^{\hat{\psi},k}$ over $M \subseteq \mathcal{M}$. By definition, an ordering of $m_i \succsim_{\mathcal{I}}^l m_{i+1}$ does not preclude the existence of a mechanism $m \in M \setminus M_i$ such that $\Delta_{\hat{\psi}}^k(M_i \cup \{m\}, M_i) < 0$. It follows that M is not an interference-type homogeneous set. \square

Given any interference-type homogeneous set of mechanisms, we have shown a simple greedy mechanism ordering that exhibits monotonicity over expected influence values; however, we have not stated conditions that guarantee a diminishing mechanism ordering. In any case, determination of interference-type homogeneity of a set of mechanisms still requires computation of the expected influence over the complete population. The main motivation for finding diminishing mechanism orderings is to avoid having to use more mechanisms than are useful (or affordable, if we assume a cost with each mechanism). By having to compute expected influence for each subset of mechanisms, we lose any advantage offered by diminishing mechanism orderings. In the following section, we define mechanism orderings using component restricted expected influence.

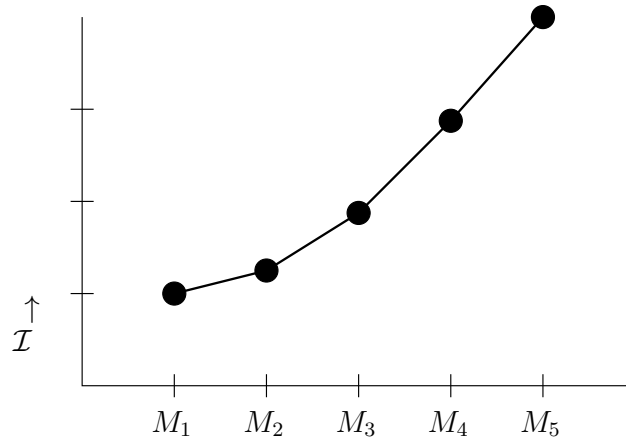


Figure 8.2: Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering over a set of positively interfering mechanisms. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity, but not diminishing returns.

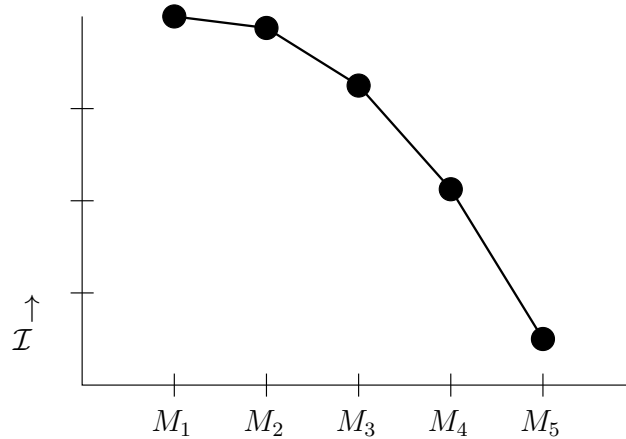


Figure 8.3: Illustration of one possible shape for the expected influence corresponding to a greedy mechanism ordering over a set of negatively interfering mechanisms. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity, but not diminishing returns.

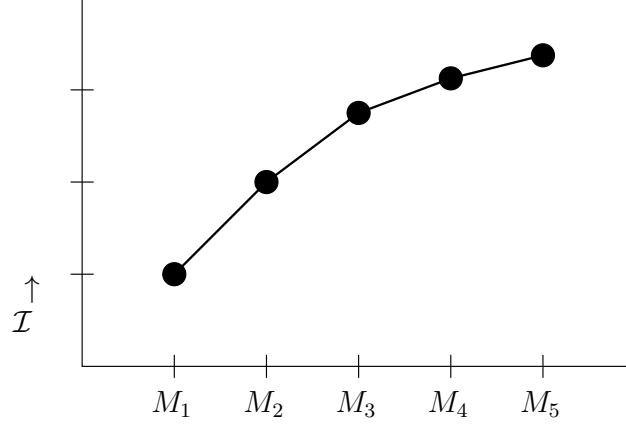


Figure 8.4: Illustration of one possible shape for the expected influence corresponding to a diminishing mechanism ordering. The horizontal axis represents the sets of mechanisms obtained by the inclusion of each additional mechanism in accordance with the ordering and the vertical axis represents the corresponding expected influence value. The greedy mechanism ordering exhibits monotonicity and diminishing returns.

8.3 Component Restricted Mechanism Orderings

Our work in Chapter 6 shows that expected influence functions and optimal targeted populations can be decomposed into computations on maximal connected components of a population. We utilize that work in this section by showing that mechanism orderings can be similarly decomposed.

To begin with, we show that orderings of mechanisms based on strict comparisons of their component restricted expected influence are preserved in the expected influence for the complete population.

Theorem 28. *For all $k > 0$, $M, M' \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M', \hat{\psi}}^{k*}$ if $\mathcal{I}_X(O, M, \hat{\psi}) \geq \mathcal{I}_X(O', M', \hat{\psi})$ for all $X \in \text{Components}^+(r_{c(M \cup M')})$, then $\mathcal{I}(O, M, \hat{\psi}) \geq \mathcal{I}(O', M', \hat{\psi})$.*

Proof. The claim follows directly from Theorem 6. □

We define a *component restricted mechanism ordering* over any $M \subseteq \mathcal{M}$ induced by component restricted expected influence functions using $k > 0$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, denoted by $\succ_r^{\hat{\psi},k}$. We define $m_i \succ_r^{\hat{\psi},k} m_{i+1}$ if and only if we have $\mathcal{I}_X(O, M_i \cup \{m_{i+1}\}, \hat{\psi}) \geq \mathcal{I}_X(O', M_i \cup \{m\}, \hat{\psi})$ for all $m \in M$, $O \in O_{M_i \cup \{m_{i+1}\}, \hat{\psi}}^{k*}$, $O' \in O_{M_i \cup \{m\}, \hat{\psi}}^{k*}$, and $X \in \text{Components}^+(r_{c(M_i \cup \{m_{i+1}, m\})})$, where M_i is the set of mechanisms including and preceding m_i under a linearization of the ordering thus far.

A greedy mechanism ordering and a component restricted mechanism ordering may have respective linearizations that are equal, for some $M \subseteq \mathcal{M}$, $k > 0$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. Such equal linearizations are not guaranteed to exist for all $M \subseteq \mathcal{M}$ since the strict pairwise comparisons that define the component restricted mechanism orderings may leave some mechanisms unordered even though they are ordered by the greedy mechanism ordering. We state this existence of equal linearizations for some sets of mechanisms in the following.

Theorem 29. *There exist $M \subseteq \mathcal{M}$, $k > 0$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and linearizations $\succ_{\mathcal{I}}^l$ of $\succ_{\mathcal{I}}^{\hat{\psi},k}$ and \succ_r^l of $\succ_r^{\hat{\psi},k}$ over M such that $\succ_{\mathcal{I}}^l = \succ_r^l$.*

Proof. Consider any $M \subseteq \mathcal{M}$, $k > 0$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. From Theorem 6, if $\mathcal{I}_X(O, M', \hat{\psi}) \geq \mathcal{I}_X(O', M'', \hat{\psi})$ for all $X \in \text{Components}^+(r_{c(M' \cup M'')})$, $O \in O_{M', \hat{\psi}}^{k*}$, and $O' \in O_{M'', \hat{\psi}}^{k*}$, then we have $\mathcal{I}(O, M', \hat{\psi}) \geq \mathcal{I}(O', M'', \hat{\psi})$ for all $O \in O_{M', \hat{\psi}}^{k*}$ and $O' \in O_{M'', \hat{\psi}}^{k*}$. The claim follows now from the definitions of a greedy mechanism ordering $\succ_{\mathcal{I}}^{\hat{\psi},k}$ and a component restricted greedy mechanism ordering $\succ_r^{\hat{\psi},k}$ over M . \square

We can achieve the same conclusion as in Theorem 28 without requiring the mechanism orderings induced by each component restricted expected influence function to be the same.

Theorem 30. *For all $k > 0$, $M, M' \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M', \hat{\psi}}^{k*}$, if for all $X' \in \text{Components}^+(r_{c(M \cup M')})$ there exists $X \in \text{Components}^+(r_{c(M \cup M')})$ such that*

$\mathcal{I}_{X'}(O, M, \hat{\psi}) < \mathcal{I}_{X'}(O', M', \hat{\psi})$ implies $[\mathcal{I}_X(O, M, \hat{\psi}) - \mathcal{I}_X(O', M', \hat{\psi})] \geq [\mathcal{I}_{X'}(O, M', \hat{\psi}) - \mathcal{I}_{X'}(O', M, \hat{\psi})]$, then $\mathcal{I}(O, M, \hat{\psi}) \geq \mathcal{I}(O', M', \hat{\psi})$.

Proof. Consider any $k > 0$, $M, M' \subseteq \mathcal{M}$, $\hat{\psi} \in \hat{\Psi}_{\mathcal{C}(\mathcal{M})}$, $O \in O_{M, \hat{\psi}}^{k*}$, and $O' \in O_{M', \hat{\psi}}^{k*}$. Assume for all $X \in \text{Components}^+(r_{\mathcal{C}(M \cup M')})$ there exists $X' \in \text{Components}^+(r_{\mathcal{C}(M \cup M')})$ such that $\mathcal{I}_X(O, M, \hat{\psi}) > \mathcal{I}_X(O', M', \hat{\psi})$ implies $[\mathcal{I}_X(O, M, \hat{\psi}) - \mathcal{I}_X(O', M', \hat{\psi})] \geq [\mathcal{I}_{X'}(O, M', \hat{\psi}) - \mathcal{I}_{X'}(O', M, \hat{\psi})]$. All such X and X' give $[\mathcal{I}_X(O, M, \hat{\psi}) + \mathcal{I}_{X'}(O, M, \hat{\psi})] \geq [\mathcal{I}_X(O', M', \hat{\psi}) + \mathcal{I}_{X'}(O', M', \hat{\psi})]$. The claim now follows from Theorem 6. \square

Theorem 30 may afford us a slight increase in the efficiency of computing mechanism orderings since we are not necessarily required to evaluate the component restricted expected influence for every maximal connected component. If the difference in the expected influence summed over all maximal connected components checked so far is greater than the size of the last unchecked maximal connected component, then the mechanism ordering induced by the expected function over the complete population will not change.

To clarify this point, assume any $M \subseteq \mathcal{M}$. For each $X_i \in \text{Components}^+(r_{\mathcal{C}(M)})$, we write $n_i = |\phi_{\mathcal{C}(M)}(\Psi_{X_i})|$ to denote the size of the population mechanism state space for that component. The expected influence computation for each $X_i \in \text{Components}^+(r_{\mathcal{C}(M)})$ takes time on the order of $O(n_i^3)$ (assuming inverse power method, see Section 5.1). Under the conditions stated in Theorem 30, we may be able to avoid computation of component restricted expected influence for one or more maximal connected components. If we let n^* denote the size of the population mechanism state space for the maximal connected components that we can avoid, then we can compute expected influence taking time on the order of $O((n - n^*)^3)$, where $n = |\phi_{\mathcal{C}(M)}(\Psi_{\mathcal{X}})|$.

Regardless of potential efficiency improvement when using the component restricted mechanism ordering approach, such decomposition of mechanism orderings is worthwhile only

when the number of maximal connected components under the union of each mechanism's relation is significantly less than one. For the cases approaching one component under the combined relation, we gain nothing from decomposing the computation of the mechanism ordering since we still end up computing expected influence for the complete connected population. The significance of any improvements is still questionable for cases in which the number of maximal connected components under the combined relation is less than one since there may be a large number of isolated individuals or very small maximal connected components, both of which offer marginal improvement.

As with the greedy mechanism ordering approach, the component restricted ordering approach also fails to provide us with an efficient approach for establishing diminishing returns that does not require computation of expected influence for the complete population.

8.4 Local Bias Magnitude

Having considered two mechanism ordering approaches that fall short of our goal of establishing diminishing returns and that are expressed in terms of the global behavior of expected influence, we turn our attention now to finding a locally-checkable property that allows us to make comparative judgments about the magnitudes of expected influence under different mechanisms.

We have identified an approach for making local judgments of global expected influence behavior that uses a measure for each individual of the bias of a mechanism to transition toward an individual mechanism state, where the individual mechanism state of interest corresponds to the influence information. Although it seems natural that this type of local bias of a mechanism toward an individual mechanism state should be consistent with the induced global process, we show that our notion of bias magnitude does not give any computational advantage over using

the expected influence function on the complete population.

In the following, we give a formal development of our notion of local bias magnitude of mechanisms. We present this formalization only to make clear the issues that prevent such a notion from offering any advantage over computation of expected influence on the complete population.

We determine the bias of a mechanism toward a mechanism state from the long-term state distribution probabilities for individuals. Given a mechanism $m \in \mathcal{M}$, the transition probabilities over $\hat{\Psi}_m$ of an individual $x \in \mathcal{X}$ are given by the individual measure assignment function μ_m^x . The transition probabilities for each individual are conditioned on the mechanism states of the neighboring individuals, which are themselves conditioned on their neighbors and so on. The long-term state distribution for $x \in \mathcal{X}$ is therefore dependent upon each $x' \in X$, where $X \in \text{Components}^+(r_m)$ is the maximal connected component containing x .

Let π_m^0 be an initial state distribution over $\phi_m(\Psi_{\mathcal{X}})$ for the complete population. Each initial state distribution π_m^0 projects onto an initial state distribution over $\phi_m(\Psi_X)$ for each $X \in \text{Components}^+(r_m)$, which we denote by $\pi_m^0[X]$. Such a projection is given for each $\hat{\psi} \in \phi_m(\Psi_X)$ by $\pi_m^0[X](\hat{\psi}) = \sum_{\hat{\psi}' \in Q} \pi_m^0(\hat{\psi}')$, where $Q = \{\hat{\psi}'' \in \phi_m(\Psi_{\mathcal{X}}) \mid \hat{\psi}''(X) = \hat{\psi}\}$. For each $X \in \text{Components}^+(r_m)$, we use $\pi_m^0[X]$ to compute a stationary distribution $\pi_m[X]$ of the Markov chain induced by μ_m^X .

Formally, we define the *local bias magnitude* function $b : \mathcal{X} \times \hat{\Psi}_{c(\mathcal{M})} \rightarrow (\mathcal{M} \rightarrow \mathbb{R})$ for each $x \in \mathcal{X}$, $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, and $m \in \mathcal{M}$ as:

$$b_{\hat{\psi}}^x(m) \stackrel{\text{def}}{=} \begin{cases} \sum_{\hat{\psi}' \in S} \pi_m[X](\hat{\psi}') & \text{if } X \neq \emptyset, \\ 0 & \text{otherwise,} \end{cases} \quad (8.2)$$

where $X = [x]_{r_m}$ and $S = \{\hat{\psi}'' \in \phi_m(\Psi_X) \mid \hat{\psi}''(x) = \hat{\psi}[\{m\}](x)\}$.

At this point, we might make comparisons between the local bias magnitudes of each individual under the influences of different mechanisms with the goal of making claims about the differences in expected influence under each different mechanism. Unfortunately, the local bias magnitude function does not give a completely locally-checkable method for determining mechanism orderings. The long-term state distribution probabilities of each individual are dependent upon the states of every other individual in the maximal connected component in which they are a member. This requires us to solve the Markov chain corresponding to each maximal connected component, which does not support our motivation for considering local bias magnitude in the first place, and presents the same problems as with the component restricted mechanism orderings.

8.5 Principal Component Analysis

Each of the approaches that we have taken to establish diminishing mechanism orderings falls short of our goal of finding a simple and efficient method for determining such orderings. We now explore a different approach by viewing each subset of mechanisms as a different dimension over which expected influence is computed and apply Principal Component Analysis (PCA) in an attempt to find the dimensions (i.e., subsets of mechanisms) that give the largest variance (i.e., impact on expected influence). Despite the usefulness of PCA for dimensionality reduction, it is unable to give us a diminishing mechanism ordering. We give a brief construction using PCA in order to make clear why it does not work for our purposes.

Given $M \subseteq \mathcal{M}$, $k > 0$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$, let $D(M)$ be the $|Pwr(M)| \times |Pwr(M)|$ matrix defined such that each entry $D(M)_{ij} = \Delta_{\hat{\psi}}^k(M_i \cup M_j, M_i)$, where the indices for the sets of mechanisms are consistent with the mechanism set enumeration $\langle Pwr(\mathcal{M}) \rangle$. Each entry $D(M)_{ij}$ gives the value of adding mechanisms M_j to mechanisms M_i . We have $D(M)_{ij} =$

$D(M)_{ii}$ for all $M_j \subseteq M_i$. We write $D'(M)$ to denote the matrix obtained from $D(M)$ by subtracting the mean along each row vector $D(M)_i$ from each entry in $D(M)_i$. This gives a mean of zero along each row vector and is required for PCA.

We compute the $|Pwr(M)| \times |Pwr(M)|$ covariance matrix $D_c(M)$ from $D'(M)$. Each entry $D_c(M)_{ij} = cov(D'(M)_i, D'(M)_j)$ gives the covariance of the row vector $D'(M)_i$ corresponding to mechanisms M_i with the row vector $D'(M)_j$ corresponding to mechanisms M_j . If $D_c(M)_{ij} > 0$, then the value of adding any set of mechanisms to M_j tends to increase whenever adding the same set to M_i increases. If $D_c(M)_{ij} < 0$, then the value of adding any set of mechanisms to M_j tends to decrease whenever adding the same set to M_i increases. And, if $D_c(M)_{ij} = 0$, then M_i and M_j are uncorrelated.

The eigenvectors and their corresponding eigenvalue are computed from $D_c(M)$. We order the eigenvectors by decreasing eigenvalue and choose the first n eigenvectors as our *principal components*, where n is chosen such that these first n eigenvectors adequately capture the largest variance of the original data. The principal components represent the new linearly transformed dimensions of the original data.

Although PCA does provide a very simple method for dimensionality reduction, the eigenvectors that give the new dimensions do not allow us to specify the set of mechanisms to which they correspond. This is due to these new dimensions being expressed as linear transformations of the original dimensions (i.e., sets of mechanisms). As with the other approaches we have addressed in this chapter, PCA still requires computation of expected influence for all possible sets of mechanisms. As such, we might as well make our determination of the best set of mechanisms with those computations and avoid the additional work of PCA altogether.

8.6 Violated Squares Approach

The best method to date that we have found for testing submodularity of a set function is defined in [42] using what are called *violated squares*. The method is rather simple and uses a geometric construction on an n -cube. Although the basic approach is straightforward, we avoid rehashing its full presentation in [42]. Instead, we restate their approach as it applies to our framework.

Assume some $k > 0$, $M \subseteq \mathcal{M}$, and $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$. Let Q_n denote the n -cube, where $n = |M|$. The vertices are labeled as sets of mechanisms and edges connect sets of mechanisms that differ in only one element. For any $M'' \subseteq M' \subseteq M$ and $M''' \subseteq M \setminus M'$, the set $\{M', M'', M' \cup M''', M'' \cup M'''\}$ is called a *square*. A square is called a *violated square* if $\Delta_{\hat{\psi}}^k(M'' \cup M''', M'') < \Delta_{\hat{\psi}}^k(M' \cup M''', M')$. That is, a violated square does not satisfy the submodularity condition.

The problem now is to determine how many violated squares exist. If no violated squares exist, then it follows that expected influence is M -submodular when using the specified k and $\hat{\psi}$. Otherwise, the existence of any violated squares reveals that expected influence is not M -submodular. It is shown in [42] that only a small number of violated squares can require a large number of changes in order to make a function submodular.

The violated squares approach presented in [42] for testing submodularity can easily be applied within our framework to test the M -submodularity of expected influence for any $M \subseteq \mathcal{M}$. As with the other approaches we have presented in this chapter, the violated squares approach still requires computation of expected influence for all subsets of mechanisms of some $M \subseteq \mathcal{M}$ over the complete population.

Chapter 9

Concluding Remarks

The proliferation of digital means of communication has led correspondingly to an increase in the number of digital representations of social networks. At first thought, many people may confine their ideas of a social network to the massive, popular forms that are common today. However, social networks take on a much finer distinction in real-life. For example, one's family can be thought of as forming one social network, whereas one's work colleagues form another social network.

Being able to express formally the many different social networks in which people exist has allowed researchers in a variety of fields to study phenomena that was not feasible prior to the development of digital social network representations. One such phenomenon that has received considerable attention over the past decade is the propagation of information across a population, where such propagation is taken as an result of the influence between individuals. A better understanding of how influence affects information propagation has many important applications and has led to the statement of different influence problems.

In this dissertation, we have focused on what is called the influence maximization problem. For this problem, we assume that a set of individuals (i.e., targeted population) is given some

information (i.e., influence information) with the objective of maximizing the spread of that information throughout the population.

Existing approaches to the influence maximization problem have assumed that individuals are placed within a single social network and that they are able to influence each other through a single influence mechanism. The goal then is to find the best targeted population of a certain size that maximizes the expected number of influenced individuals in the long-term. Such approaches take a limited view of the potential types of influence in a population.

We have presented in this dissertation a new model for studying influence problems, such as the influence maximization problem, that leverages multiple influence mechanisms and multiple social relations for propagating information across a population. The usage of multiple social relations reflects more accurately the social structures of a population. We provide evidence from the social psychology literature that supports the formulation of our influence model.

Our results have focused on the impact of multiple mechanisms and their corresponding relations on optimal targeted populations for the influence maximization problem. We define several mechanism properties that we use to characterize different types of influence mechanisms, and show how different types of mechanism impact expected influence and the determination of optimal targeted populations.

Unlike most related works, we have not assumed a connected population. We showed that the maximal connected components of a population place a lower bound on the size of an optimal targeted population when using mechanisms that exert non-vanishing influence. We also showed that targeted populations for the complete population are composed from those of the maximal connected components. We build upon this result by showing that targeted populations for sets of mechanisms that do not contain any bridges between their mechanism relations can similarly be composed from the targeted populations for each individual mechanism from

the set of mechanisms.

We gave formal definitions for several well-known types of influence within our model (e.g., group conformity, authoritarian, contagion), and we established properties for each of these types of mechanisms using some of the mechanism properties we have defined.

We concluded with a presentation of our preliminary results on what we refer to as mechanism submodularity. We showed that expected influence is mechanism submodular for mechanisms that exert non-interfering influence, but did not state general conditions for satisfying mechanism submodularity. We explored several alternatives to mechanism submodularity, some of which may prove fruitful in future work, but each fell short of our goal of finding a simple and efficient method for determining mechanism submodularity or diminishing mechanism orderings.

9.1 Future Work

The large number of parameters in the specification of our model required us to make many assumptions so that we could focus on the problems of interest. The properties of other types of influence mechanisms can be analyzed, other combination methods can be used, or other influence problems can be studied within our model's framework. We conclude in this section with a discussion of just a few of the possible areas for future work.

The use of a combination method is essential for specifying how individuals react to the effects of multiple influences, and also provides a natural interpretation as the behavioral type of an individual. We assumed a single combination method and, hence, a behavioral (sub-)type homogeneous population. Our interpretation of combination methods as behavioral types requires elaboration, but we expect that improvements to the information propagation shown in other works [13, 14] would be reflected within our model as well.

Another extension to our statement of behavioral types is the potential for viewing behavioral types in a hierarchical manner so that we could discuss behavioral types of sets of individuals. We maintain the individual behavioral types, but observe that individuals may be members of various groups, organizations, or corporations (see, for example, [22, 24, 39, 54]). Aggregate population behavioral types could be leveraged to better understand the potential for influencing individuals that belong to certain communities, or for characterizing these various groups in terms of their influence susceptibilities.

We have assumed that the mechanism relations do not change over time, but we do recognize that social relations may continuously evolve. For example, the *is-coworker-of* and *is-friend-of* may evolve as employees leave or are hired, or as friendship is formed or broken. Use of dynamic relations must also consider time-dependent transition probabilities and the induced time-heterogeneous Markov chains. This would introduce additional complexity, but it would also result in a more accurate reflection of social relations.

An extension to the results we presented in Section 6.7 is to consider the decomposition of optimal targeted populations in the presence of bridges. Any bridges that exist between different mechanism relations might lead to an increase in expected influence, but they might also lead to an increase in the size of an optimal targeted population over the ones for each individual mechanism. On the other hand, the size of an optimal targeted population for the set of mechanism might decrease over those for each individual mechanism. Determining the mechanism properties that guarantee an increase in expected influence along with a decrease in the size of an optimal targeted population provides an interesting problem for future work.

Another research pursuit that is of great interest deals with the inversion of our influence model. Our focus has been placed on modeling how individuals are influenced and understanding how such influence affects the expected spread of information throughout a population. Our current pursuit could offer insight into modeling the inverse process of determining the

originating source(s) of influence. That is, given the current state of a population, how were the individuals influenced to transition into their current states and what individuals are the originators of exerting that influence? Modeling this inverse process has important applications in areas such as computer security.

REFERENCES

- [1] Marianne Akian, Stéphane Gaubert, and Laure Ninove. Multiple equilibria of nonhomogeneous markov chains and self-validating web rankings. *arXiv:0712.0469v1 [math.PR]*, December 4, 2007.
- [2] Chalee Asavathiratham. *The Influence Model: A Tractable Representation for the Dynamics of Networked Markov Chains*. PhD thesis, Massachusetts Institute of Technology, October 2000.
- [3] Chalee Asavathiratham, Sandip Roy, Bernard Lesieutre, and George Verghese. The influence model. *IEEE Control Systems Magazine*, December 2001.
- [4] Solomon E. Asch. Opinions and social pressure. *Scientific American*, 193(5):31–35, November 1955.
- [5] Lars Backstrom, Dan Huttenlocher, Jon Kleinberg, and Xiangyang Lan. Group formation in large social networks: Membership, growth, and evolution. In *Proceedings of the Twelfth International Conference on Knowledge Discovery and Data Mining (KDD)*, Philadelphia, Pennsylvania, USA, pages 44–54, August 20-23, 2006.
- [6] Christopher L. Barrett, Stephen Eubank, and Madhav V. Marathe. An interaction-based approach to computational epidemiology. In *Proceedings of the Twenty-Third AAAI Conference on Artificial Intelligence, Chicago, Illinois, USA*, pages 1590–1593. AAAI Press, July 13-17, 2008.
- [7] André Campos, Virginia Dignum, Frank Dignum, Alberto Signoretti, Anne Magály, and Sérgio Fialho. A process-oriented approach to model agent personality (extended abstract). In Decker, Sichman, Sierra, and Castelfranchi, editors, *Proceedings of the Eighth International Conference on Autonomous Agents and Multiagent Systems (AAMAS)*, Budapest, Hungary, pages 1141–1142. International Foundation for Autonomous Agents and Multiagent Systems, May 10-15, 2009.
- [8] Tim Carnes, Chandrashekhar Nagarajan, Stefan M. Wild, and Anke van Zuylen. Maximizing influence in a competitive social network: A follower’s perspective. In *Proceedings of the Ninth International Conference on Electronic Commerce (ICEC)*, Minneapolis, Minnesota, USA, pages 351–360. ACM, August 19-22, 2007.
- [9] Claudio Castellano, Santo Fortunato, and Vittorio Loreto. Statistical physics of social dynamics. *arXiv:0710.3256v2 [physics.soc-ph]*, May 11, 2009.
- [10] Robert B. Cialdini and Noah L. Goldstein. Social influence: Compliance and conformity. *Annual Review of Psychology*, 55:591–621, 2004.

- [11] Robert T. Clemen and Robert L. Winkler. Combining probability distributions from experts in risk analysis. *Risk Analysis*, 19(2):187–203, April 1999.
- [12] Peter Clifford and Aidan Sudbury. A model for spatial conflict. *Biometrika*, 60(3):581–588, 1973.
- [13] Sebastiano A. Delre, Wander Jager, and Marco A. Janssen. Diffusion dynamics in small-world networks with heterogeneous consumers. *Computational & Mathematical Organization Theory*, 13(2):185–202, June 2007.
- [14] Jean-Dominique Deuschel and Andreas Greven, editors. *Interacting Stochastic Systems*. Springer, 2005.
- [15] Pedro Domingos and Matt Richardson. Mining the network value of customers. In *Proceedings of the Seventh ACM SIGKDD International Conference on Knowledge Discovery and Data Mining, San Francisco, California, USA*, pages 57–66. ACM, August 26-29, 2001.
- [16] Leon Festinger. A theory of social comparison processes. *Human Relations*, 7(2):117–140, May 1954.
- [17] Noah E. Friedkin. A formal theory of social power. *Journal of Mathematical Sociology*, 12(2):103–126, 1986.
- [18] Satoru Fujishige. *Submodular Functions and Optimization*, volume 58 of *Annals of Discrete Mathematics*. Elsevier, second edition, 2005.
- [19] Aram Galstyan and Paul R. Cohen. Influence propagation in modular networks. In *AAAI Spring Symposium on Social Information Processing, Palo Alto, California, USA*, pages 21–23. AAAI Press, March 26-28, 2008.
- [20] George R. Goethals and R. Eric Nelson. Similarity in the influence process: The belief-value distinction. *Journal of Personality and Social Psychology*, 25(1):117–122, 1973.
- [21] M. Grötschel, L. Lovász, and A. Schrijver. The ellipsoid method and its consequences in combinatorial optimization. *Combinatorica*, 1(2):169–197, 1981.
- [22] Albert O. Hirschman. *Exit, Voice, and Loyalty: Responses to Decline in Firms, Organizations, and States*. Harvard University Press, 1970.
- [23] Richard A. Holley and Thomas M. Liggett. Ergodic theorems for weakly interacting infinite systems and the voter model. *Annals of Probability*, 3(4):643–663, 1975.
- [24] Bernardo A. Huberman and Tad Hogg. Communities of practice: Performance and evolution. Technical report, Xerox Research Park, 1994.

- [25] Dean L. Isaacson and Richard W. Madsen. *Markov Chains Theory and Applications*. John Wiley & Sons, Inc., 1976.
- [26] Robert A. Jacobs. Methods for combining experts' probability assessments. *Neural Computation*, 7(5):867–888, September 1995.
- [27] David Kempe, Jon Kleinberg, and Éva Tardos. Maximizing the spread of influence through a social network. In *Proceedings of the Ninth International Conference on Knowledge Discovery and Data Mining (KDD)*, pages 137–146. ACM Press, 2003.
- [28] David Kempe, Jon Kleinberg, and Éva Tardos. Influential nodes in a diffusion model for social networks. In *Proceedings of the Thirty-Second International Colloquium on Automata, Languages and Programming (ICALP), Lisbon, Portugal*, volume 3580 of *Lecture Notes in Computer Science*, pages 1127–1138. Springer Berlin / Heidelberg, July 11-15, 2005.
- [29] W. O. Kermack and A. G. McKendrick. A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London*, 115(772):700–721, August 1927.
- [30] Masahiro Kimura and Kazumi Saito. Approximate solutions for the influence maximization problem in a social network. In *Knowledge-Based Intelligent Information and Engineering Systems*, volume 4252 of *Lecture Notes in Computer Science*, chapter Artificial Intelligence for Decision Making, pages 531–536. Springer Berlin / Heidelberg, October 9-11, 2006.
- [31] R. Lambiotte and S. Redner. Dynamics of vacillating voters. *Journal of Statistical Mechanics: Theory and Experiment*, L10001, October 2007.
- [32] David A. Levin, Yuval Peres, and Elizabeth L. Wilmer. *Markov Chains and Mixing Times*. American Mathematical Society (AMS), 2009.
- [33] Thomas M. Liggett. *Stochastic Interacting Systems: Contact, Voter and Exclusion Processes*, volume 324 of *Grundlehren der mathematischen Wissenschaften*. Springer, 1999.
- [34] William N. McPhee. *Formal Theories of Mass Behavior*. The Free Press of Glencoe, 1963.
- [35] Gordon W. Meyer. Social information processing and social networks: A test of social influence mechanisms. *Human Relations*, 47(9):1013–1047, 1994.
- [36] Elchana Mossel and Sebastien Roch. On the submodularity of influence in social networks. In *Proceedings of the Thirty-Ninth ACM Symposium on Theory of Computing (STOC), San Diego, California, USA*. ACM, June 11-13, 2007.

- [37] J. R. Norris. *Markov Chains*. Cambridge Series on Statistical and Probabilistic Mathematics. Cambridge University Press, 1997.
- [38] L.-E. Öller. A method for pooling forecasts. *Journal of the Operational Research Society*, 29(1):55–63, January 1978.
- [39] Avi Pfeffer, Subrata Das, David Lawless, and Brenda Ng. Global/local dynamic models. In Manuela Veloso, editor, *Proceedings of the Twentieth International Joint Conference on Artificial Intelligence (IJCAI), Hyderabad, India*, pages 2580–2585. IJCAI / AAAI Press, January 6-12, 2007.
- [40] Roopesh Ranjan and Tilmann Gneiting. Combining probability forecasts. Technical Report 543, Department of Statistics, University of Washington, October 2008.
- [41] Kazumi Saito, Ryohei Nakano, and Masahiro Kimura. Prediction of information diffusion probabilities for independent cascade model. In *Knowledge-Based Intelligent Information and Engineering Systems*, volume 5179 of *Lecture Notes in Computer Science*, pages 67–75. Springer Berlin / Heidelberg, 2008.
- [42] C. Seshadhri and Jan Vondrák. Is submodularity testable? In *Innovations in Computer Science 2011 (ICS 2011), Beijing, China*, pages 195–210. Tsinghua University Press, January 7-9, 2011.
- [43] V. Sood and S. Redner. Voter model on heterogeneous graphs. *arXiv:cond-mat/0412599v2*, April 3, 2005.
- [44] M. Stone. The opinion pool. *Annals of Mathematical Statistics*, 32(4):1339–1342, 1961.
- [45] Jerry Suls, Rene Martin, and Ladd Wheeler. Three kinds of opinion comparison: The triadic model. *Personality and Social Psychology Review*, 4(3):219–237, 2000.
- [46] Sarah Tanford and Steven Penrod. Social influence model: A formal integration of research on majority and minority influence processes. *Psychological Bulletin*, 95(2):189–225, March 1984.
- [47] S. Hoya White, A. Martín del Ray, and G. Rodríguez Sánchez. Using cellular automata to simulate epidemic diseases. *Applied Mathematical Sciences*, 3(20):959–968, 2009.
- [48] Andrew W. Wicker. Interest-matching comparisons using CP-nets. Master’s thesis, Department of Computer Science, North Carolina State University, December 2006.
- [49] Andrew W. Wicker and Jon Doyle. Interest-matching comparisons using CP-nets. In *Proceedings of the Twenty-Second AAAI Conference on Artificial Intelligence, Vancouver, British Columbia, Canada*, pages 1914–1915. AAAI Press / MIT Press, July 22-26, 2007.

- [50] Andrew W. Wicker and Jon Doyle. Comparing Preferences Expressed by CP-networks (Extended Abstract). In *AAAI Workshop on Advances in Preference Handling, Chicago, Illinois, USA*, pages 128–133. AAAI Press / MIT Press, July 13-17, 2008.
- [51] Andrew W. Wicker and Jon Doyle. Comparing preferences expressed by CP-networks. (*In Preparation*), 2012.
- [52] Fang Wu, Bernardo A. Huberman, Lada A. Adamic, and Joshua R. Tyler. Information flow in social groups. *Physica A: Statistical and Theoretical Physics*, 337(1-2):327–335, June 2004.
- [53] Hua Yuan and Guoqing Chen. Network virus-epidemic model with the point-to-group information propagation. *Applied Mathematics and Computation*, 206(1):357–367, December 2008.
- [54] Ding Zhou, Eren Manavoglu, Jia Li, C. Lee Giles, and Hongyuan Zha. Probabilistic models for discovering e-communities. In *Proceedings of the Fifteenth International World Wide Web Conference (WWW), Edinburgh, Scotland*, pages 173–182. ACM, May 23-26, 2006.

APPENDIX

Appendix A

Example Matrices

A.1 Individual Transition Matrices for Section 7.2.1

The individual transition matrices used to compute the expected influence values in Section 7.2.1 are provided below. We do not include the large 256×256 population transition matrix for lack of space, but it can be constructed from the individual transition matrices.

$$[\mu_{Auth}^{x_1}(00)] = [\mu_{Auth}^{x_1}(10)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_1}(01)] = [\mu_{Auth}^{x_1}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_2}(00)] = [\mu_{Auth}^{x_2}(10)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_2}(01)] = [\mu_{Auth}^{x_2}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_3}(\emptyset)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_4}(0000)] = [\mu_{Auth}^{x_4}(0010)] = [\mu_{Auth}^{x_4}(0100)] = [\mu_{Auth}^{x_4}(0110)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_4}(1000)] = [\mu_{Auth}^{x_4}(1010)] = [\mu_{Auth}^{x_4}(1100)] = [\mu_{Auth}^{x_4}(1110)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_4}(0001)] = [\mu_{Auth}^{x_4}(0011)] = [\mu_{Auth}^{x_4}(0101)] = [\mu_{Auth}^{x_4}(0111)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_4}(1001)] = [\mu_{Auth}^{x_4}(1011)] = [\mu_{Auth}^{x_4}(1101)] = [\mu_{Auth}^{x_4}(1111)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_5}(000)] = [\mu_{Auth}^{x_5}(010)] = [\mu_{Auth}^{x_5}(100)] = [\mu_{Auth}^{x_5}(110)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_5}(001)] = [\mu_{Auth}^{x_5}(011)] = [\mu_{Auth}^{x_5}(101)] = [\mu_{Auth}^{x_5}(111)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_6}(000)] = [\mu_{Auth}^{x_6}(010)] = [\mu_{Auth}^{x_6}(100)] = [\mu_{Auth}^{x_6}(110)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Auth}^{x_6}(001)] = [\mu_{Auth}^{x_6}(011)] = [\mu_{Auth}^{x_6}(101)] = [\mu_{Auth}^{x_6}(111)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_7}(000)] = [\mu_{Auth}^{x_7}(010)] = [\mu_{Auth}^{x_7}(100)] = [\mu_{Auth}^{x_7}(110)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_7}(001)] = [\mu_{Auth}^{x_7}(011)] = [\mu_{Auth}^{x_7}(101)] = [\mu_{Auth}^{x_7}(111)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_8}(000)] = [\mu_{Auth}^{x_8}(010)] = [\mu_{Auth}^{x_8}(100)] = [\mu_{Auth}^{x_8}(110)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Auth}^{x_8}(001)] = [\mu_{Auth}^{x_8}(011)] = [\mu_{Auth}^{x_8}(101)] = [\mu_{Auth}^{x_8}(111)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

A.2 Counter-example for Proof of Theorem 22

The individual transition matrices and population transition matrix used in the proof of Theorem 22 are as follows:

$$[\mu_{Conf}^{x_1}(\emptyset)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} \quad [\mu_{Conf}^{x_2}(00)] = \begin{bmatrix} 1 & 0 \\ 1 & 0 \end{bmatrix}$$

$$[\mu_{Conf}^{x_2}(01)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix} \quad [\mu_{Conf}^{x_2}(10)] = \begin{bmatrix} 0.5 & 0.5 \\ 0.5 & 0.5 \end{bmatrix}$$

$$[\mu_{Conf}^{x_2}(11)] = \begin{bmatrix} 0 & 1 \\ 0 & 1 \end{bmatrix} \quad [\mu_{Conf}^{x_3}(\emptyset)] = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}$$

$$[\mu_{Conf}] = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.5 & 0 & 0.5 & 0 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0.5 & 0 & 0.5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0.5 & 0 & 0.5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 & 0.5 & 0 & 0.5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{bmatrix}$$

Using targeted population $\{x_2\}$ and influence information $\hat{\psi} \in \hat{\Psi}_{c(\mathcal{M})}$ with $\hat{\psi}[Conf] = 1$, we have an initial state distribution $\pi_{Conf}^0 = \langle 0, 0, 1, 0, 0, 0, 0, 0 \rangle$. We find a stationary distribution given by $\pi_{Conf}^0 \cdot \lim_{t \rightarrow \infty} [\mu_{Conf}]^t = \langle 1, 0, 0, 0, 0, 0, 0, 0 \rangle$. The expected influence is now easily computed as $\mathcal{I}(\{x_2\}, Conf, \hat{\psi}) = 0$.