Effect of Interaction Mechanisms on Facebook Dynamics Using a Common Knowledge Model

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Abstract. Web-based interactions allow agents to coordinate and to take actions (change state) jointly, i.e., to participate in collective action such as a protest, facilitating spread of contagion to large groups within networked populations. In game theoretic contexts, coordination requires that agents share common knowledge about each other. Common knowledge emerges within a group when each member knows the states and the types (preferences) of the other members, and critically, each member knows that everyone else has this information. Hence, these models of common knowledge and coordination on communication networks are fundamentally different from influence-based unilateral contagion models, such as those devised by Granovetter and Centola. Common knowledge arises in many settings in practice, yet there are few operational models that can be used to compute contagion dynamics. Moreover, these models utilize different mechanisms for driving contagion. We evaluate the three mechanisms of a common knowledge model that can represent web-based communication among groups of people on Facebook. We evaluate these mechanisms on five social (media) networks with wide-ranging properties. We demonstrate that different mechanisms can produce widely varying behaviors in terms of the extent of contagion spreading and the speed of contagion transmission.

1 Introduction

1.1 Background and Motivation

Infamous waves of uprisings (e.g., Black Lives Matter, Women's March, Occupy Wall Street) are commonly characterized by the significant use of social media to share information prior to, as well as during, protests to reach a critical number of participants. The goal of understanding how local online interactions through social networks can facilitate information sharing in a way that generates common knowledge and coordination within large groups has motivated the construction of models of mobilization. While the exemplar in this work is protests, other applications of mobilization are family decisions to evacuate in the face of hurricanes and forest fires, and to participate in demonstrations for equality. The results herein apply to these examples as well.

There are many influence-based threshold models of diffusion that have been proposed and evaluated, e.g., [16, 32, 19, 15, 26]. In a networked population, an agent or network node i transitions from an inactive state (state 0) to an active or state (state 1) if at least a threshold θ number of its neighbors (connections) are already in state 1. These models are used to explain different behaviors, such as the spread protests [15] and Twitter hashtags [26]. Watts argues for the use of threshold models in a wide range of scenarios [32]. In these models, agents make individual decisions to change state, irrespective of the decisions of their neighbors, and hence are referred to as *unilateral* models.

In contrast, in game-theoretic models of collective action, agents' decisions to transition to state 1 depend on their expectations of what others will do. That is, they need to know each others' willingness to participate (defined by the threshold θ) and this information needs to be *common knowledge* among a group of agents. Common knowledge (CK) emerges within a group when each member knows the states and attributes (e.g., preference, type) of the other members, and critically, each member knows that everyone else knows her attributes. Common knowledge enables a group of agents to *coordinate* their actions, thus enabling them to transition state simultaneously if it is mutually beneficial to do so.

In the context of collective action, e.g., protests, two CK models ([11] and [21]) combine social structure and individual incentives together in a coordination game of incomplete information and provide a rigorous formalization of common knowledge. The authors study which network structures are conducive to coordination, and the local spread of knowledge and collective action.

CK models are fundamentally different from unilateral models as (*i*) contagion can *initiate* in CK models—meaning that contagion can be generated when no contagion previously existed—whereas it does not in unilateral models (unless an agent's threshold is zero); (*ii*) CK models may utilize multiple mechanisms at graph geodesic distances of 1 and 2, whereas unilateral models most often use influence from distance-1 neighbors, and (*iii*) the characterizing (social) network substructure for threshold-based models is a star subgraph centered at the ego node making a decision, while those for CK models include distance-2 based stars and other substructures such as cliques [12] and bicliques [21] (i.e., complete bipartite graphs).

In this work, we evaluate the Common Knowledge on Facebook (CKF) model [21]. It models communication on Facebook (through "wall" or "timeline") as a means to generate CK and to facilitate coordination. Geodesic distance-2 communication is achieved as follows: two individuals i and j do not directly communicate, but each communicates with person k. This means that if, for example, i writes information about herself on k's wall, then j knows i's information by reading k's wall, without directly communicating with i. The information thus travels distance-2, from i to k to j. Multiple mechanisms are operative in the CKF model, including CK itself, network dynamics, and local and global interactions. Hence, it is of interest to understand the effects of mechanisms on the spread of contagions. We aim to develop computational models of the CKF model mechanisms to study these mechanisms individually and in combination,

to quantify their effects on the spread of collective action. Table 1 describes these mechanisms, which are formalized in Section 3.

Table 1. Communication mechanisms of the CKF model evaluated in this work, individually and in combination. These mechanisms may be operative in contagion initiation, propagation, or both. Mechanism abbreviations are denoted in [·].

Mechanism	Description
Common knowledge [CK]	This is a common knowledge mechanism characterized by *bicliques* in social networks. This mechanism can *initiate* contagion, and can drive contagion *propagation*. No seeded nodes with contagion are required.
Neighborhood dynamics [ND2]	This is influence (communication) produced by neighbors within distance-2 of an ego node. This mechanism *propagates* contagion.
Population dynamics [PD2]	Since agents (nodes) know both state and thresholds of agents within distance-2, an agent can infer information about the numbers of agents currently in state 1, even when these other agents are at geodesic distances of 4 or more. This mechanism *propagates* contagion.

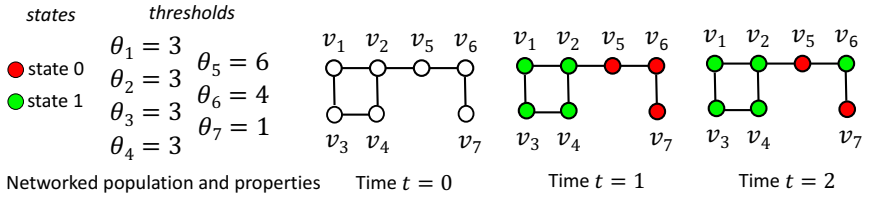


Fig. 1. Spread of contagion on a 7-node graph illustrating the mechanisms of Table 1. Each operative mechanism is evaluated independently, at each t. At $t = 2$, the *spread size* is 5 (5 nodes in green), and the *spread fraction* is 5/7. The dynamics resulting from the different mechanisms are discussed in the text.

Figure 1 provides an example illustrating all three mechanisms summarized in Table 1. In this network, there are 7 people with different thresholds. Based on the CKF model [21] summarized in Section 3, for agents to participate (i.e., transition to state 1), they need to share common knowledge with a group of people (they need to form a complete bipartite graph), and their thresholds should be less than the size of the common knowledge set (i.e., the group they share common knowledge with). In this example, agents 1, 2, 3, and 4 have threshold of 3, indicating that each needs to have at least 3 other people to participate (i.e., transition to state 1) for them to participate. These four people form a complete bipartite graph (a square) that allows them to generate common knowledge about their willingness to participate. They know each others' thresholds and know that it is sufficiently low for them to jointly participate and achieve mutual benefits. Hence, they transition to state 1 at $t = 1$. This is referred to as the *common knowledge* [CK] mechanism. On the other hand, agent 5, who shares common knowledge of thresholds with agents 1, 2, and 4 (through the 4-node star network centered at agent 2), has threshold of 6 which is not low enough for him to participate with the other 3 players that he shares CK with. Agent 5

also is part of CK node sets $\{2, 5, 6\}$ (a 3-node star centered at agent 5) and $\{5, 6, 7\}$ (a 3-node star centered at agent 6), but cannot transition to state 1 for the same reason. Similarly, persons 6 and 7 do not transition to state 1 at $t = 1$.

Since agent 2 is within distance-2 of agent 6 (friend-of-friend), agent 6 knows agent 2's threshold and state (action), through the Facebook wall or timeline of agent 5. At $t = 2$, agent 2's state is 1 and her fixed threshold is 3. Thus, agent 6 knows that at least four agents are in state 1. Agent 6's threshold is satisfied and she transitions to state 1. This is the *population dynamics* [PD2] mechanism.

Finally, at $t = 3$, person 7 will transition to state 1 as a result of the *neighborhood dynamics* [ND2] mechanism: it has one activated neighbor (agent 6) within distance-2 to meet its threshold of 1. All of the state transitions in this example are made formal in Section 3.

1.2 Contributions of This Work

Following others who study contagion dynamics on networks (e.g., [29]), we quantify contagion dynamics on five web-based social networks that range over 6× (i.e., over a factor of 6) in numbers of nodes 4× in numbers of edges, 4× in average degree, 13× in maximum degree, and 80× in average clustering coefficient. Thresholds range over 3×. We constructed agent-based models and a framework that can turn on and off any combination of mechanisms in simulations of contagion dynamics. (The CKF model is presented in Section 3; simulation process is given in Section 5.) There are also companion theoretical results, but owing to space limitations, these will be included in an extended version of the paper.

1. Effects of different contagion mechanisms on the spread evolution. We demonstrate that: (*i*) The [ND2] mechanism, as a driving force for contagion diffusion, is often relatively weak compared to the other mechanisms. For many networks and sets of simulation parameters, plots of fractions of nodes in state 1, as a function of time, show little difference between the effects of the [CK] mechanism alone, versus the [CK] and [ND2] mechanisms combined. However, there are cases (e.g., for the P2P network with $\theta = 8$ and $p_p = 0.2$), where the addition of [ND2] to [CK] increases the spread fraction by more than 50%. (*ii*) The [PD2] mechanism dominates the other two mechanisms for driving contagion in particular cases (e.g., for the Facebook (FB) network). In other cases, the [CK] mechanism dominates (e.g., for several cases for the Wiki network). As average degree decreases relative to threshold, the more the [PD2] mechanism can dominate. As average network degree increases, the more the [CK] mechanism dominates. This is because a star subgraph is a form of biclique, and the more nodes in a biclique, the more threshold assignments will cause state transition owing to CK. (*iii*) If [CK] and [PD2] mechanisms are already operative, then there is no increase in spread fraction if mechanism [ND2] is incorporated. (*iv*) There are combinations of simulation conditions (e.g., network, threshold, participation probability, CK model mechanisms) that can produce small or large spread size changes by varying only one of these inputs.

2. Sensitivity of contagion dynamics to average degree d_{ave}. The spread size (large or small) is driven by the magnitude of average degree relative to the

node threshold θ assigned uniformly to all nodes. In all five networks, spread size can be large (e.g., spread fraction > 0.5). If $d_{ave} > \theta$, then outbreak sizes are large; if $d_{ave} < \theta$, then outbreak sizes are lesser. We demonstrate a pronounced effect on spread size even when the magnitudes of d_{ave} are θ close.

2 Related Work

There are several studies that model web-based social media interactions, including the following. The spread of hashtags on Twitter is modeled using a threshold model in [26]. Diffusion on Facebook is modeled in [28], and a similar type of mechanism on Facebook is used to study the resharing of photographs [10]. None of these works uses the "wall" or "timeline" mechanism of Facebook that is modeled here in the CKF model. Several *unilateral* models and applications were identified in the Introduction. These are not repeated. Here, we focus on game-theoretic common knowledge models, in particular, the CKF model.

A couple of data mining studies have used Facebook walls, including an experimental study [13]. Features of cascades on Facebook are studied using user wall posts [18], but again, these are cascades of the conventional social influence type; there is no assessment of CK-based coordination. Most experimental studies are of unilateral interactions, where one user sends messages and influences to one or more others. Web-based experimental studies of unilateral influence phenomena include those on Twitter [15], Facebook [5, 14, 24, 10, 1], LinkedIn [9], Digg [17], Doodle [27], Stack Overflow [30], and Wikipedia [4], among others (e.g., [7, 8]). We argue that our work on CK is valuable in complementing the large number of studies, such as those cited here, on unilateral models and mechanisms.

There have been a few works on the CKF model, which was initially introduced in [21]. Details of the game-theoretic formulation are provided there. For example, the CKF model is not efficiently computable because finding all bicliques in a network is an NP-hard problem. This makes studying CKF on very large networks (e.g., with 1 million or more nodes) extremely difficult. An approximate and computationally efficient CKF model is specified in [22]. Studies involving both models include basic simulation results [21]. CK dynamics on networks that are devoid of key players is studied in [23]. *None* of these investigates the individual and combinations of mechanisms of Table 1.

3 Model

3.1 Preliminaries

This section provides a formal description of the Common Knowledge on Facebook (CKF) model [21] studied in this paper. The population is represented by a communication network $G(V, E)$. There is a node set $V = \{1, 2, \ldots, n\}$ of n nodes (people) and edge set E where an undirected edge $\{i, j\} \in E$ means that nodes $i, j \in V$ can communicate with each other. Each person $i \in V$ is in a state $a_{it} \in \{0, 1\}$: if $a_{it} = 1$, person i is in the active state (e.g., joining a protest), and $a_{it} = 0$ otherwise (e.g., staying at home). We use progressive dynamics [19], such that once in state 1, nodes do not transition back to 0. Each node i has a threshold θ_i that indicates its inclination/resistance to activate. Given person

i's threshold θ_i and the system state at t, denoted by $a_t = (a_{1t}, a_{2t}, \ldots, a_{nt})$, her utility is given by

$$U_{it} = \begin{cases} 0 & \text{if } a_{it} = 0 \\ 1 & \text{if } a_{it} = 1 \ \wedge \ \#\{j \in V : a_{jt} = 1\} \geq \theta_i \\ -z & \text{if } a_{it} = 1 \ \wedge \ \#\{j \in V : a_{jt} = 1\} < \theta_i \end{cases} \tag{1}$$

where $-z < 0$ is the penalty she gets if she activates and not enough people join her. Thus, a person will activate as long as she is sure that there is a sufficient number of people (in the population) in state 1 at t. A person always gets utility 0 by staying in state 0 regardless of what others do since we do not consider free-riding problems. When she transitions to the active state, she gets utility 1 if the total number of other people activating is at least θ_i. (Note that these "others" do not have to be neighbors of i, as in unilateral models.)

The CKF model describes Facebook-type (friend-of-friend) communication in which friends write to and read from each others' Facebook walls and this information is also available to their friends of friends. The mechanisms and its implications are described below. The communication network indicates that if $\{i, j\} \in E$, then node i (resp., j) communicates (θ_i, a_{it}) (resp., (θ_j, a_{jt})) to node j (resp., i) over edge $\{i, j\}$ at time t, and this information is available to j's neighbors. The communication network helps agents to coordinate by creating common knowledge at each t. Agents' presence on the network (online or offline) is captured by the participation probability $0 \leq p_p \leq 1$ for each node, which determines whether a node is participating in the contagion dynamics at each t; e.g., whether i is online or offline at t in Facebook.

3.2 Facebook Common Knowledge Model Mechanisms

Here we describe the three mechanisms in this model (cf. Table 1), and their implications. Figure 1 illustrates these mechanisms through an example. First of all, the CKF model describes a Facebook type communication which allows for distance-2 communication: two nodes, i and j, with $\{i, j\} \notin E$ can communicate by posting to and reading from the wall of a common neighbor k, provided $\{i, k\}, \{j, k\} \in E$. Thus, all $i \in V$ can communicate with all nodes $j \in V$ such that their geodesic distance is $|\{i, j\}| \leq 2$. All three mechanisms make use of this Facebook communication structure.

The **neighborhood dynamics [ND2]** mechanism (Table 1) is similar to the Granovetter [16] unilateral contagion model, but with interaction at distance-1 and -2. Let the neighbors j of i within distance-2 are defined by $N_i^2 = \{j : |\{i, j\}| \leq 2\}$. The [ND2] mechanism is given by

$$a_{it} = \begin{cases} 1 & \text{if } \ a_{i,t-1} = 1 \text{ or } |\{j \in N_i^2 : a_{j,t-1} = 1\}| \geq \theta_i \\ 0 & \text{otherwise.} \end{cases} \tag{2}$$

For the **common knowledge [CK]** mechanism of Table 1, the biclique subgraph is the structure necessary for creation of CK among a group of people [21], and allows them to jointly activate. We first compute all node-maximal bicliques in G, which is an NP-hard problem [3]. Let $M^{biclique}$ denote the set of nodes of

G that forms a biclique. Then, V in Equation (1) is replaced with $M^{biclique}$. At each t, Equation (1) is computed for each $i \in V$ in each CK set $M^{biclique}$ for which $i \in M^{biclique}$.

Finally, the **population dynamics [PD2]** mechanism indicates that a node i that is in state 0 can infer a minimum number of nodes already in state 1 if a neighbor j in N_i^2 is already in state 1, by knowing θ_j. Formally,

$$a_{it} = \begin{cases} 1 \text{ if } a_{i,t-1} = 1 \text{ or} \\ \qquad (\max \theta_j : j \in N_i^2, a_{j,t-1} = 1) + 1 \geq \theta_i \\ 0 \text{ otherwise.} \end{cases} \tag{3}$$

Assume $a_{i,t-1} = 0$. If $j \in N_i^2$ and $a_{j,t-1} = 1$, with θ_j, then i can infer that at least $\theta_j + 1$ nodes are in state 1. Now, if $\theta_i \leq \theta_j + 1$, then i will transition to state 1; i.e., $a_{it} = 1$.

At each time $t - 1$, all operative mechanisms are evaluated, independently, for each $i \in V$ in which $a_{i,t-1} = 0$. If any of the three mechanisms causes i to transition, then $a_{it} = 1$.

4 Social Networks

The web-based networks of this study are summarized in Table 2. FB is a Facebook user network [31], P2PG is a peer-to-peer network, Wiki is a Wikipedia network of voting for administrators, and Enron is an Enron email network [25]. All but the SF1 network are real (i.e., mined) networks. SF1 is a scale free (SF) network generated by a standard preferential attachment method [6] to fill in gaps of the real networks. For networks possessing multiple components, we use the giant component. These networks have wide-ranging properties and hence represent a broad sampling of web-based mined network features. Figure 2 shows the average degrees per network in the original graphs G, corresponding to geodesic distance of 1, and in the square of the graphs G^2 that are particularly relevant to CK model dynamics (forthcoming in Section 6).

Table 2. Characteristics of web-based social networks analyzed. If there are multiple connected components in a graph, we use only the giant component. Here, n and m are numbers of nodes and edges, respectively; d_{ave} and d_{max} are average and maximum degrees; c_{ave} is average clustering coefficient; and Δ is graph diameter. Properties are computed with the codes in [2].

Network	**Type**	n	m	d_{ave}	d_{max}	c_{ave}	Δ
FB	Facebook	43,953	182,384	8.30	223	0.115	18
P2P	Peer Comms.	10,876	39,940	7.34	103	0.00622	10
Enron	Email	33,696	180,811	10.7	1,383	0.509	17
SF1	Stylized	4,956	45,031	18.2	270	0.0780	8
Wiki	Online Voting	7,115	100762	28.3	1065	0.141	7

5 Agent-Based Model and Simulation Parameters

We conduct discrete time agent-based simulations based on the model described in Section 3 using the web-based networks given in Table 2. Table 3 summarizes

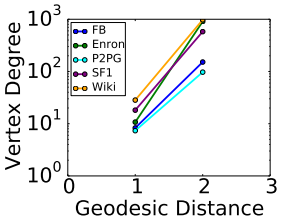


Fig. 2. Average vertex degree for geodesic distances 1 and 2 (i.e., for G^1 and G^2), which are relevant for the CK, ND2, and PD2 mechanisms for driving contagion through networks.

the parameters and their values associated with each simulation. A **simulation** consists of a set of 30 runs, where a **run** consists of the spread of contagion from an initial configuration (or state) with all nodes in state 0 at time $t = 0$, to a specified maximum time t_{max}. Differences among runs is stochasticity in models.

Table 3. Summary of contagion study parameters.

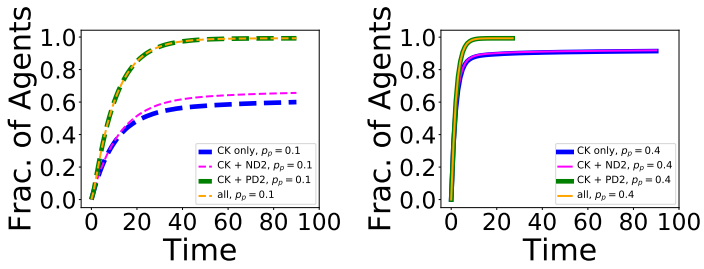
Parameter	**Description**
Agent Thresholds θ	Uniform threshold values for a simulation: all nodes in a network have the same value. Values range from $\theta = 8$ through $\theta = 29$.
Participation Probabilities p_p	Uniform value for all nodes in a simulation. Values in the range of 0.05 to 0.4.
Model Mechanisms	[CK], [ND2], and [PD2] mechanisms described in Table 1. [CK] is always operative to initiate contagion.
Seed Vertices	No specified seed vertices; all vertices initially in state 0. CK model initiates contagion without seeds.
Simulation Duration t_{max}	30 and 90 time steps.

6 Simulation Results

In this section, we present the results of our agent-based model simulations. All results provided are average results from 30 runs.

Effects of CKF model mechanisms on contagion dynamics. We analyze the effects of the [CK], [ND2], and [PD2] mechanisms (described in Table 1) and their combinations on the time histories of activated nodes for each network of the study. Figure 3 contains time histories for the fraction of nodes in state 1 over time for the Wiki network. In this simulation, all nodes have threshold $\theta \approx d_{ave} = 29$. The mechanism combinations are [CK] only, [CK] plus [ND2], [CK] plus [PD2], and [CK] plus [ND2] plus [PD2] (i.e., all) mechanisms. In Figure 3a, $p_p = 0.1$; in Figure 3b, $p_p = 0.4$. Several observations are important. First, the [ND2] mechanism does not contribute significantly to the driving force to transmit contagion in the system. This is seen in the first plot in that the magenta curve is only slightly above the blue curve, i.e., the addition of [ND2] to [CK] results in a small increase in spread fraction (i.e., fraction of agents in state 1). In comparing the orange and green curves in the left plot, we observe that adding [ND2] to [CK]+[PD2] does not increase spread fraction. The same two comparisons in the right plot (where $p_p = 0.4$) give the same conclusion. Second, the addition of the [PD2] mechanism to the [CK] mechanism can produce

significant increases in spread fraction (comparing blue and green curves). Third, in moving from the left to the right plot, the spread fractions increase, for a given time t, and the contagion spreads more rapidly, with increasing p_p. These findings are shown for all networks, as described below.

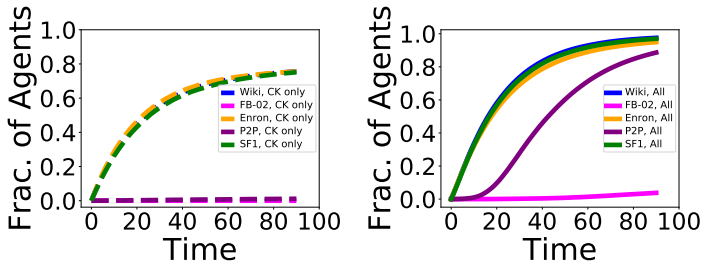


(a) $p_p = 0.1$ (b) $p_p = 0.4$

Fig. 3. Wiki network results for $\theta = d_{ave}$=29: (a) $p_p = 0.1$, (b) $p_p = 0.4$. Cumulative fraction of agents in state 1 is plotted as a function of time in simulation for combinations of different mechanisms (in Table 1). Each propagation mechanism is isolated for different simulations and is represented by a different curve; however, [CK] (labeled CK) is always operative. In (a), the blue ([CK] only) and magenta ([CK + [ND2]) curves are close together, indicating that for $p_p = 0.1$, the distance-2 classic diffusion mechanism [ND2] provides a relatively small increase to the overall contagion driving force. In (b), the blue ([CK] only) and magenta ([CK + [ND2]) curves overlay; this means that [ND2] provides no noticeable increase in driving force for contagion spreading. [PD2] (green and orange curves) in both plots provides significant additional driving force, since the green and orange curves are well above the blue and magenta curves. The *all mechanisms* (denoted *all* in legend) curves coincide with the [CK]+[PD2] curves since they (orange curves) overlay with the green curves. This means that, again, [ND2] does not provide much driving force to spread a contagion.

The effect of CK-only mechanism on contagion dynamics compared to the full model across networks. We analyze the fraction of activated nodes over time under the CK-only mechanisms and under all mechanisms of the CKF model combined. Figure 4 provides results for all networks, where the agent threshold in all networks is $\theta = 9$. In Figure 4a, the networks with greater outbreaks (Enron, Wiki, and SF1) have average degrees greater than θ, while those with lesser outbreaks (FB and P2P) have values of d_{ave} that are lesser that θ. It is worth noting that three networks have d_{ave} near θ. In Figure 4b, the addition of [ND2] and [PD2] driving forces results in a relatively greater increase in the spread size for P2P. Since FB has greater d_{ave} than P2P, one would surmise that FB should also show increased spreading in Figure 4b. The reason this is not the case is that P2P has far more nodes with degree 10 than does FB, and thus the driving force for $\theta = 9$ is greater in the P2P network.

Comparisons of final contagion spread at time $t = 30$ across networks. Finally, Fig. 5 provides spread fractions at $t = 30$ for four of the five networks under different combinations of mechanisms (specified on x-axis): from left to right, [CK] only, [CK]+[ND2], [CK]+[PD2], and all three mechanisms combined.



(a) CK mechanism only (b) All three mechanisms

Fig. 4. Cumulative fraction of agents in state 1 as a function of simulation time, for $p_p = 0.05$ and for $\theta = d_{ave}$=9: (a) only [CK] is active, (b) all mechanisms are active. See Table 1. The results show the sensitivity of outbreak size on average degree d_{ave}. In (a), d_{ave} for FB and P2P are slightly less than θ; these networks have small outbreaks due to CK only. SF1, Enron, and Wiki all have d_{ave}s greater than $\theta = 9$, one (Enron) only slightly, and the other two have d_{ave} appreciably greater than θ. In (b), the addition of [PD2] drives the contagion to greater magnitudes.

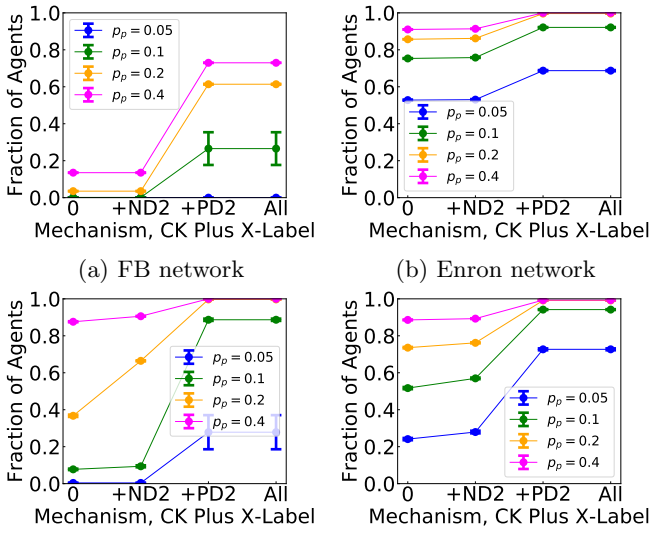
The uniform threshold for each network is its average degree, so that $\theta = d_{ave}$ is different across networks. In each plot, curves are for $p_p = 0.05$, 0.1, 0.2, 0.4.

The FB network of Figure 5a has the smallest spread sizes. The [CK] mechanism in isolation can drive contagion through appreciable fractions of the other three networks, depending on p_p. FB, and Enron in Figure 5b, show no effect of the [ND2] mechanism on spread fractions. However, P2P and Wiki in Figures 5c and 5d show positive contributions to spread size from the [ND2] mechanism. It is remarkable for P2P (Figure 5c) when $p_p = 0.2$. In all four plots, the [PD2] mechanism contributes significantly to the driving force for contagion spread (the positive slopes of curves from "+ND2" to "+PD2" on the x-axis), except perhaps when [CK] or [CK]+[ND2] produce very large spread sizes. Finally, we observe that the curves are flat in going from "+PD2" to "All" on the x-axis, where the difference is the addition of the [ND2] mechanism.

There is intuition for the lesser effectiveness of the [ND2] mechanism, relative to [PD2]. When p_p is low, a vertex in state 0 can have relatively fewer neighbors within distance-2 that are participating. The [ND2] mechanism counts the number of these neighbors that are in state 1, and hence the mechanism is weaker. In contrast, for [PD2], a node i in state 0 needs only *one* participating and active neighbor j within distance-2 that has a threshold $\theta_j + 1 \geq \theta_i$ in order for i to change state to 1. This is a stronger mechanism, and hence the spread is greater.

7 Conclusion

We evaluate the CKF contagion model on a set of networks with wide ranging properties, for a range of thresholds and participation probabilities. We model and investigate multiple mechanisms of contagion spread (initiation and propagation), as well as the full model. We find evidence that the [CK] and [PD2] mechanisms are the major driving forces for the contagion initiation and spread, compared to [ND2]. These types of results are being used to specify conditions



(a) FB network (b) Enron network

(c) P2P network (d) Wiki network

Fig. 5. CKF model results. Cumulative fraction of agents in state 1 at time $t = 30$ as a function of mechanisms and p_p (same legend for all plots) for $\theta = d_{ave}$: (a) FB, $\theta = 9$; (b) Enron, $\theta = 11$; (c) P2P, $\theta = 8$; and (d) Wiki, $\theta = 29$. The mechanisms on the x-axis always includes [CK] over all 30 time steps, where "0" corresponds to only the [CK] mechanism; "+ND2" means [CK] and [ND2]; "+PD2" means [CK] and [PD2]; and "All" means the full model. The error bars for y-axis values represent one stdev. The data illustrate that [PD2] provides a much greater driving force for contagion spread than does [ND2]. Although [CK] initiates contagion, [PD2] often generates a greater contribution to driving force than does [CK]. See for example P2P and $p_p = 0.1$.

for impending human subject experiments that will evaluate CK and its mechanisms (e.g., [20]), and will be used to assess the predictive ability of the models.

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