Semi-Supervised Learning with Competitive Infection Models

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Abstract

The goal in semi-supervised learning is to effectively combine labeled and unlabeled data. One way to do this is by encouraging smoothness across edges in a graph whose nodes correspond to input examples. In many graph-based methods, labels can be thought of as propagating over the graph, where the underlying propagation mechanism is based on random walks or on averaging dynamics. While theoretically elegant, these dynamics suffer from several drawbacks which can hurt predictive performance.

Our goal in this work is to explore alternative mechanisms for propagating labels. In particular, we propose a method based on dynamic infection processes, where unlabeled nodes can be “infected” with the label of their already infected neighbors. Our algorithm is efficient and scalable, and an analysis of the underlying optimization objective reveals a surprising relation to other Laplacian approaches. We conclude with a thorough set of experiments across multiple benchmarks and various learning settings.

1 Introduction

The supervised learning framework underlies much of the empirical success of machine learning systems. Nonetheless, results in unsupervised learning have demonstrated that there is much to be gained from unlabeled data as well. This has prompted considerable interest in the semi-supervised learning setting, where the data includes both labeled and unlabeled examples. Methods for semi-supervised learning (SSL) are especially useful for applications in which unlabeled examples are ample, but labeled examples are scarce or expensive.

One of the most wide-spread approaches to SSL, and our focus in this paper, is the class of graph-based methods. In these, part of the problem input is a graph that specifies which input points should be considered close. Graph-based methods assume that proximity in the graph implies similarity in labels. There are many variations on this idea [5, 7, 36, 40], each using smoothness and graph distance differently. However, they all share the intuition that the classification function should be smooth with respect to the graph.

One way for encouraging smoothness is by optimizing an objective based on the graph Laplacian. This is prevalent in classic SSL methods such as Label Propagation (LabelProp) [46] and its variants [45, 4, 41], as well as in recent deep graph embedding methods [44, 35]. In some cases, the Laplacian objective can be interpreted as the probability that a random walk terminates at a certain state. In others, the objective can be expressed as a quadratic form which can be optimized by iterative local averaging of labels. The optimization process can hence be thought of as propagating labels under a certain averaging dynamic process, whose steady state corresponds to the optimum. Due to their elegance, computational properties, and empirical power, random walks and local averaging have become the standard mechanisms for propagating information in many applications. Nonetheless, they have several shortcomings, which we address here.

First, many of the guarantees of such methods hold only for undirected graphs. For directed graphs, the Laplacian is not necessarily PSD, meaning that the objective is no longer convex, and that the quadratic smoothness interpretation breaks down. Optimization in directed graph Laplacians is much harder and far less understood [42], and sampling is computationally prohibitive, slow to converge, and unstable [28, 29].

Second, such methods were originally designed for graphs that approximate the density of the data in feature space. As such, they can fail when applied to real graphs, especially large networks with a commu-
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InfProp (accuracy: 0.91, mean: 0.86)
LabelProp (accuracy: 0.40, mean: 0.55)
ShortPaths (accuracy: 0.79, mean: 0.77)

Figure 1: For graphs with weakly inter-connected components, infection dynamics (our method, left) propagate labels better than random walks (middle) or shortest paths (right). Labeled nodes are outlined, shapes denote true labels, and probabilistic predictions are encoded by CMY color values. See supp. material for more details.

Figure 1: For graphs with weakly inter-connected components, infection dynamics (our method, left) propagate labels better than random walks (middle) or shortest paths (right). Labeled nodes are outlined, shapes denote true labels, and probabilistic predictions are encoded by CMY color values. See supp. material for more details.

InfProp is motivated by the idea that different graph types may require different dynamics for efficient propagation of information. It is inspired by propagation dynamics found in the natural and social worlds, and draws on the successful application of infection models in different contexts [25, 20, 14]. InfProp is especially efficient for graphs with highly intra-connected but lightly inter-connected components, a characteristic of many real-world networks. Fig. 1 illustrates this for a small synthetic random network with three clusters (see supplementary material for details). As can be seen, InfProp propagates information correctly, even when the seed set is very small. In comparison, LabelProp provides uninformative and almost uniform predictions which are prone to error, and shortest paths over the weighted graph err due to cross-cluster links.

InfProp uses infection probabilities for labeling; these, however, turn out to be \#P hard to compute exactly. We therefore provide a fully polynomial-time randomized approximation scheme (FPRAS). Our solution exploits an equivalence between the infection process and shortest paths in random graphs. The resulting algorithm is easy to parallelize, making the method highly scalable. It also extends to various learning settings, such as multilabel prediction and active SSL.

In Sec. 5 we analyze the optimization objective underlying the propagation of labels via infection dynamics, highlighting an intriguing connection graph Laplacians. Our analysis shows that InfProp can be viewed as optimizing a quadratic objective, in which weights are seed-specific and related in an intricate manner to the underlying diffusion process. We conclude with an extensive set of experiments in multiple learning settings which demonstrate the effectiveness of our approach.

2 Propagating Labels with Infections

In this section we present our infection-based method for semi-supervised learning. We are given as input a
The core idea of our method is to propagate labels from labeled to unlabeled nodes using infection dynamics. The process is initialized with all seed nodes in an infected state and all unlabeled nodes in a null state $\emptyset$. Then, a stochastic model of infection dynamics is used to determine how the infectious state of nodes in the graph changes over time, typically as a function of the states of neighboring nodes. To support multiple label classes, we consider competitive infection models. In these, seeds $s \in S$ are initially infected with their true labels $y_s$, and compete in infecting unlabeled nodes.

We focus on the transductive setting, where the goal is to predict the labels of all non-seed nodes $u \in U$. The models we consider are stochastic and converge to a steady state. This means that, after some time point, the labels of all nodes will not change anymore (we refer to this as process termination, or steady state). Since the process itself is stochastic, each instantiation will result in a different value for the labels at termination. For a given infection model, let $Y_{v\ell}$ be the binary random variable indicating whether node $v$ is infected by label $\ell$ at steady state.\footnote{For multilabel tasks, $\mathcal{Y}$ is the set of seed node identities, and $f$ becomes a weighted sum of their labels.} Since our goal is to reason about the labels of the nodes, it will be natural to utilize the infection dynamics to generate probabilistic predictions. For each node $v$, our method outputs a distribution over labels $f_v$. Each entry $f_v(y)$ corresponds to the probability that $v$ has value $y$ at steady state, as a function of the seed set $S$ and its labels:

$$f_v(S, y) = \Pr[Y_{v\ell} = 1] = \mathbb{E}[Y_{v\ell}]$$

(1)

Note that $\ell$ can take values in $1, \ldots, L$ but also $\ell = 0$ for $\emptyset$. The entry $f_v(0)$ therefore describes the (possibly non-negative) probability that $v$ remained uninfected.

Computing $f$ exactly is known to be \#P-hard even for simple infection models [14]. Hence, like many other infection-based methods [25, 17, 16], we resort to a Monte-Carlo approach and estimate $f$ by averaging over infection outcomes $Y$. Our final predictor $\hat{f}$ is:

$$\hat{f}_v(S, y) = \frac{1}{N} \sum_{i=1}^{N} Y_{v\ell}^{(i)}$$

(2)

where $Y_{v\ell}^{(i)}$ is an indicator for the $i^{th}$ random instance.

In principle, outcomes $Y$ can be evaluated by simulating the infection dynamics. This however is not straightforward for several of the models we consider, such as those with continuous time. In the next section we describe some infection models, and show how $\hat{f}$ can be efficiently computed for them using an alternative graphical representation of the infection process.

We conclude by stating an approximation bound for $f$. As we can calculate $\hat{f}$ efficiently (see next section) this implies that our method yields an efficient approximation scheme for the true infection probabilities.

**Proposition 1.** For every $\epsilon, \delta \in [0, 1]$, if $N \geq \frac{1}{\delta^2 \epsilon^2} \log \frac{2n(L+1)}{\delta}$, then with probability of at least $1 - \delta$, Algorithm 2 returns $\hat{f}$ such that $\|\hat{f} - f\|_{\max} \leq \epsilon$.

**Proof.** Note that each $Y_{v\ell}$ is a random variable in $\{0, 1\}$. Furthermore, $\hat{f}$ is an average of $Y$, and $f$ is the corresponding expectation. The result is obtained by applying the Hoeffding and union bounds. \hfill $\square$

# 2.1 Competitive Infection Models for Graph Labeling

As mentioned above, our SSL method relies on an infection process where nodes of the graph are “infected” with labels. There are many variants of infection processes (see [13]); we describe some relevant ones below.

## 2.1.1 The Independent Cascade model

Since its introduction in [19], the simple but powerful Independent Cascade (IC) model has been used extensively. The original IC model, briefly reviewed below, is a discrete-time, network-dependent interpretation of the classic Susceptible-Infected-Recovered (SIR) epidemiological model [26]. At time $t = 0$, seed nodes are initialized to an infected state, and all other nodes to a susceptible state. If node $u$ is infected at time step $t$, then at time $t+1$ it attempts to infect each of its non-infected out-neighbors $v \in Nei(u)$, and succeeds with probability $p_{uv}$. If successful, we refer to the edge $(u, v)$ as active or activated, mark the infection time of $v$ as $\tau_v = t+1$, and set $v$’s infecter to be $u$, which we denote by $\rho(v) = u$. The model is therefore parametrized by the set of all edge infection probabilities $\{p_{uv} \mid (u, v) \in E\}$ (given as input via $p_{uv} = W_{uv}$). Once a node becomes infected, it remains in this state. As infections are probabilistic, not all nodes are necessarily infected. The process terminates either when all nodes are infected, or (more commonly) when all infection attempts at some time step are unsuccessful.

The IC model describes the propagation of a single infectious content. Hence, it can tell us only when and how a node is infected, but not by what. This motivated a class of competitive infection models which support multiple content types. Several competitive
IC variants have been proposed [8, 10, 12, 24]. The common theme in these is that nodes inherit the label of their earliest infecter (with tie-breaking when needed). All of these are supported by our method. In the supplementary material we show how our approach can also be applied to threshold models [25].

2.1.2 Continuous Time Dynamics

While simple and elegant, the IC dynamics are somewhat limited in their expressive power. One important generalization is the Continuous-Time IC model (CTIC) [20]. This model is well suited for SSL as it is flexible, does not require tie-breaking, and allows for incorporating node priors. In this model, a successful infection attempt entails an “incubation period”, after which the node becomes infected. Hence, if $u$ succeeds in infecting $v$ at time $\tau_{uv} \in \mathbb{R}^+$, it draws an incubation time $\delta_{uv} \sim D(\theta_{uv})$, and $v$ can become infected at time $\tau_{uv} = \tau_u + \delta_{uv}$. As in the IC model, $v$ inherits the label of its earliest infecter $\rho(v) = \arg\min_s \tau_{uv}$. The competitive CTIC model generalizes the competitive IC model for an appropriate choice of $D$, where $\delta$ is set to 1 with probability $p_c$, and $\infty$ with probability $1 - p_c$. We therefore consider a general mixture distribution of activations and incubation times $D(p, \theta)$, where $\delta$ is sampled w.p. $p_c$, and set to $\infty$ w.p. $1 - p_c$. Since infections are determined by the earliest successful attempt, the shortest-paths interpretation and algorithm (Sec. 2.2.1) hold for the random graph $G^\delta = (V, E, \delta)$.

2.2 Computing Infections Efficiently

For infection models as in Sec. 2.1, we would like to calculate predictions $\hat{f}$ as in Eq. (2). A naive approach would be to do this by simulating the infection process $N$ times and averaging. This, however, is inefficient for discrete-time IC, requires continuous time simulation for CTIC, and does not apply to general models. We hence provide an equivalent efficient alternative below.

### Algorithm 1 BasicInfProp($G, S, y, p, N$)

1: for $i = 1, \ldots, N$ do
2: Initialize $Y^{(i)}_{ul} \leftarrow 0$ for all $u \in U, \ell \in Y \cup \emptyset$
3: for $(u, v) \in E$ do
4: $W_{uv} \leftarrow 1$ with probability $p_{uv}$, and $\infty$ o.w.
5: for $s \in S$ do
6: $\text{dist}[s][\cdot] \leftarrow \text{Dijkstra}(G, W, s)$
7: for $u \in U$ do
8: $Y^{(i)}_{u, \alpha(u)} \leftarrow 1$ where $\alpha(u) \in \arg\min_s \text{dist}[s][u]$
9: Return $\hat{f} = \frac{1}{N} \sum_{i=1}^{N} Y^{(i)}$

2.2.1 Infections as Shortest Paths

We now present an alternative view of the sampling process, which facilitates efficient implementation and extensions. Consider first the discrete time IC process. For a single instantiation of the process, recall that if $u$ succeeds in infecting $v$, the edge $(u, v)$ is considered active. We use the set of active edges $A \subseteq E$ (sampled throughout the instantiation until termination) to construct the active graph $G^A = (V, E, W^A)$ with weights $W^A_e = 1$ for $e \in A$ and $W^A_e = \infty$ for $e \in E \setminus A$. An important observation is that node $v$ is infected at termination if there exists a path in $G^A$ from some seed node $s \in S$ to $v$ with finite weight. We refer to this as an active path. Since $v$’s actual infection time $\tau_v$ is set by the earliest successful infection, it is also the length of the shortest active path from some $s \in S$.

The above formulation allows for replacing time with graph distances. Let $d_A(u, v)$ be the distance from $u$ to $v$ in $G^A$. Due to the recursive nature of label assignment, it follows that $v$ inherits its label from the $s \in S$ whose distance to $v$ is shortest. We refer to $s$ as $v$’s ancestor, denoted by $\alpha(v)$, and set $\alpha(v) = \emptyset$ when there are no paths from $S$ to $v$. Infection outcomes $Y_{uv}$ can now be expressed using distances:

$$Y_{uv} = \mathbb{I}\{\ell = y_{\alpha(u)}\}, \quad \alpha(v) = \arg\min_{s \in S} d_A(s, v) \tag{3}$$

Recall that our motivation here was to compute $Y$ without simulating the dynamics. Since distances $d_A$ depend on edge activations, it is not yet clear why Eq. (3) is useful. An important result by [25] shows that ancestors can be computed over a simpler random graph model. Specifically, let $\tilde{A} \subseteq E$ be a random edge set, where each edge $(u, v) \in E$ is sampled independently to be in $\tilde{A}$ with probability $p_{uv}$. Then, for an appropriately defined $G^A$ and $d_{\tilde{A}}$, we have:

$$\alpha(v) = \arg\min_{s \in S} d_{\tilde{A}}(s, v) \tag{4}$$

Thus, to compute each $Y_{uv}^{(i)}$ (and hence $\hat{f}$), it suffices to sample edges independently, and compute shortest paths on $G^A$, bypassing the need for simulation. Under this view, $f$ can be thought of as an ensemble of shortest-path predictors, whose weights are set by the dynamics. Algorithm 1 provides a simple implementation of this idea for the discrete time IC model. After sampling edges, the algorithm computes shortest paths (using Dijkstra) from each $s \in S$ to all $u \in U$. Then, each node $u$ is assigned the label of its ancestor $\alpha(u)$. This approach applies to a large class of infection models that admit to a similar graphical form [25].
2.2.2 Improved Efficiency via Modified Dijkstra

Recall that for a single infection instance, a node inherits its label from the closest seed node. Based on this, Algorithm 1 offers a direct approach for computing \( f \), where shortest paths are computed from each of the \( k \) seed nodes to every unlabeled node \( v \in U \) using \( k \) calls to Dijkstra. While correct, this method suffers an unnecessary factor of \( k \) on its runtime. To reduce this overhead, we change Dijkstra’s initialization and updates, so that only a single call would suffice. Algorithm 2 implements this idea for the general CTIC model (Sec. 2.1.2) and allows for node priors (Sec. 4). The correctness of the algorithm is stated below, and a proof is provided in the supplementary material.

**Proposition 2.** Algorithm 2 correctly computes the estimated infection probabilities \( f \) in Eq. (1).

The worst-case complexity of Dijkstra, and hence of each iteration in Algorithm 2, is \( O(m+n \log n) \). Other implementations of Dijkstra which support further parallelization or GPUs [30] can also be modified for our setting. Nonetheless, the practical run time of Algorithm 2 can be, and typically is, much better, for two reasons. First, note that only the subset of active edges are traversed (and sampled on the fly), and only nodes which are reachable from \( S \) are processed. The infection parameters \( p \) therefore induce a trade-off between the influence diameter of \( S \) and the run time (empirical demonstration in Fig. 3 (left)). Second, many settings require “hard” predictions \( \hat{y} \in \mathcal{Y} \), typically set by \( \hat{y}_v = \arg \max_{y \in \mathcal{Y}} f_{uv} \). Hence, for \( \hat{y}_v \) to be correct, it suffices that \( f_{uv \hat{y}_v} \geq f_{uv} \) for all \( y \in \mathcal{Y} \), which does not require the full convergence stated in Proposition 1 (empirical demonstration in Fig. 3 (right)).

In this section we showed how infection outcomes can be computed efficiently. It is therefore only natural to ask - what is it that infections optimize? In the next section we show that \( f \) is in fact the solution to a quadratic optimization objective, whose weights intricately depend on the infection dynamics.

3 What do infections optimize?

Many SSL methods propose an optimization objective which encodes some notion of smoothness. For instance, the classic LabelProp algorithm [46] encourages adjacent nodes to agree on their predicted labels by minimizing a quadratic penalty term:

\[
f_{LP} = \arg \min_{f'} \sum_{\ell \in \mathcal{Y}} \sum_{u,v} W_{uv} (f'_{uv} - f_{uv})^2
\]  

for predictions \( f' \) and symmetric weights \( W \), subject to \( f'_{uv} = \mathbb{1}_{(y=v)} \) for all \( s \in S \). In this section we show that InfProp has a related interpretation. Specifically, we show that the InfProp predictions \( f \) minimize the quadratic objective in Eq. (13).

While similar in structure, the fundamental difference between Eqs. (5) and (13) lies in how the weights are determined. In LabelProp (and variants), edge weights are given as input, and are typically set according to some feature-based similarity measure. In this sense, each \( W_{uv} \) is a local function of the features of \( u \) and \( v \). In contrast, weights in Eq. (13) are set in a global manner. As we show next, each weight is a function of the infection dynamics, of the specific seed set \( S \), and, if available, of the features of all \( \mathcal{Y} \) nodes. To demonstrate this, and to see why Eq. (13) holds, it will be helpful to analyze InfProp from a spectral perspective.

3.1 A Laplacian Interpretation for InfProp

An interesting property of LabelProp is that its objective can be expressed via the graph Laplacian. For a directed weighted graph, the normalized Laplacian is:

\[
\mathcal{L}_{LP} = I - \tilde{W}
\]  

where \( \tilde{W} = D^{-1}W \), \( D \) is diagonal with \( D_{uv} = \sum_v W_{uv} \) (and \( W \) is symmetric). The output of LabelProp can be computed by solving the system \( \mathcal{L}_{LP} f' = 0 \) for the unlabeled nodes. We now show that the infection-based predictions of InfProp also correspond to the solution of a certain Laplacian system which is determined by the seed set and the infection dynamics.

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**Algorithm 2 InfProp \((G, S, y, D, q, N)\)**

1: for \( i = 1, \ldots, N \) do
2: Initialize \( Y_{i}^{(i)} \leftarrow 0 \) for all \( u \in U, \ell \in \mathcal{Y} \cup \emptyset \)
3: for \( v \in U \) do
4: \( \text{dist}[v] \leftarrow \infty, \ y[v] \leftarrow \emptyset \)
5: for \( s \in S \) do
6: \( \text{dist}[s] \leftarrow 0, \ y[s] \leftarrow y_s \)
7: push \( s \) into min-queue \( Q \)
8: while \( Q \) is not empty do
9: pop \( v \) from \( Q \) \( \triangleright \text{break ties randomly} \)
10: for \( u \in \text{Nei}(v) \) do
11: sample \( \delta_{vu} \sim D(\theta, p) \) \( \triangleright \text{incubation time} \)
12: if \( \delta_{vu} = \infty \) then continue
13: \( \text{alt} \leftarrow \text{dist}[v] + W_{uv} + q_u(y[v]) \) \( \triangleright \text{penalize} \)
14: if \( \text{alt} < \text{dist}[u] \) then
15: \( \text{dist}[u] = \text{alt} \)
16: \( y[u] \leftarrow y[v] \) \( \triangleright u \text{ inherits label from parent } v \)
17: push/update \( u \) in \( Q \) with \( \text{dist}[u] \)
18: \( Y_{u,y[v]}^{(i)} \leftarrow 1 \) for all \( u \in U \)
19: Return \( f = \frac{1}{N} \sum_{i=1}^{N} Y^{(i)} \)
Consider a single infection instance, and denote by $T_{uv}(S)$ the random variable indicating whether $u$ was infected by $v$ for seed $S$, namely $T_{uv}(S) = 1_{\{v \in \rho(u)\}}$. We refer to the matrix $T$ as the infector matrix. Further denote by $T$ the expected infector matrix $T(S) = E[T(S)]$. We use this to define the following Laplacian:

$$\mathcal{L}(S) = I - T(S)$$

(7)

Note that $\mathcal{L}$ is defined over the same graph $G$, but need not be symmetric. We now show that $\mathcal{L}$ is indeed a Laplacian matrix, and that it can be used to infer $f$.

**Lemma 1.** The infection-based predictions $f$ in Eq. (2) are also the solution to the Laplacian system:

$$\mathcal{L}(S)f = b(S)$$

(8)

where:

$$b_{u\ell}(S) = \sum_v b_{v\ell}^{(S)}, \quad b_{v\ell}^{(S)} = cov[T_{vu}(S), Y_{u\ell}]$$

For conciseness, we defer the full proof to the supplementary material, and show here a useful special case.

**Lemma 2.** If $T$ and $Y$ are uncorrelated, then the infection-based predictions $f$ in Eq. (2) are also the solution to the homogeneous Laplacian system:

$$\mathcal{L}(S)f = 0$$

(9)

**Proof.** We first show that $\mathcal{L}$ is a graph Laplacian, namely that the sum of each row in $T$ is equal to the corresponding diagonal element in $I$, which is 1. Since rows in $T$ have only one non-zero entry of value one, each row in $T$ is positive and sums to one. Note that $T_u$ provides a distribution over the infectors of $u$.

We now prove Eq. (9). By definition, the label of each node at steady state is set to be that of its infector, namely $Y\ell = Y_{\ell\rho(\ell)}$ for all $u$ and $\ell$, or simply $Y = T Y$. Using Eq. (2) and applying expectation, we have:

$$f(S) = E[Y] = E[T(S)Y]$$

(10)


3.2 InfProp as Optimization

We next use the Laplacian insight above to provide an objective minimized by the InfProp solution. Begin by noting that for LabelProp, the solution of Eq. (6) coincides with the solution of the following objective:

$$f_{LP} = \text{argmin}_{f'} \|\mathcal{L}_{LP} f'\|_F^2$$

$$= \text{argmin}_{f'} \sum_{\ell} \sum_u \left( f'_{u\ell} - \sum_v \bar{W}_{uv} f'_{v\ell} \right)^2$$

(11)

where minimization is only over the unlabeled nodes, and $\| \cdot \|_F$ denotes the Frobenius norm. This gives an alternative quadratic objective which bounds Eq. (5) and directly expresses the steady-state of LabelProp’s averaging dynamics. In a similar fashion, we can derive an equivalent formulation of $f$ in Eq. (8) via:

$$f(S) = \text{argmin}_{f'} \|\mathcal{L}(S) f' - b(S)\|_F^2$$

(12)

Expanding and denoting $w_{uv}^{(S)} = T_{uv}(S)$ provides the general objective of our method:

$$\min_{f'} \sum_u \sum_{\ell} \left( f'_{u\ell} - \sum_v (w_{uv}^{(S)} f'_{v\ell} + b_{u\ell}^{(S)}) \right)^2$$

(13)

Note that Eq. (13) and Eq. (11) are structurally equivalent up to the bias terms, which disappear under the conditions of Lemma 2. The critical difference is that the weights in Eq. (13) are now functions of the dynamics and seed set, rather than just scalars given as input. Through their dependence on $T$ and $Y$, the weights and bias terms in Eq. (13) are in fact functions of the dynamics. In this sense, $w_{uv}^{(S)}$ quantifies how well $v$ relays information from $S$ to $u$, which depends on the entire graph. Similarly, the term $b_{u\ell}^{(S)}$ quantifies consistency between the identity of $v$’s infector ($v$) and the inherited label ($\ell$). This means that frequent yet indecisive infectors are penalized, while reliable nodes remain unbiased.

Finally, note that the optimization interpretation above does not offer a better optimization scheme, since calculating the weights $w(S)$ and $b(S)$ would require sampling. Hence, our InfProp sampling algorithms from Sec. 2.2 would be a simpler approach.

4 Other Learning Settings

In this section we briefly describe how our method extends to other learning settings used in our experiments. For more details please see the supp. material.

Incorporating features and priors: Many network-based datasets include additional node features or priors. Our method incorporates priors directly into the CTIC dynamics by penalizing incubation times. Denote by $\rho_{v\ell}$ the prior for labeling $v$ with $\ell$, and let $q : [0, 1] \rightarrow \mathbb{R}$ be a penalty function. If $u$ succeeds in infecting $v$ with $\ell$, the incubation time $\delta_{uv}$ is penalized by an additional $q(\rho_{v\ell})$. For a monotone decreasing $q$, high priors induce low penalties, and vice versa. Although penalties are deployed locally, they delay the propagation of the penalized label across the graph in a global manner.
Confidence and active learning: Recall that $v$ remains uninfected with probability $f_v$. Hence, $\sigma_v(S) = 1 - f_v$ serves as a natural measure of confidence. We use this as a selection criteria for an active setting where the goal is to choose a seed set of size $k$. The objective we consider coincides with the well-studied notion of influence [25], which is monotone and submodular and admits to an efficient greedy approximation scheme. Our method thus offers a tractable alternative to existing active SSL methods [23, 18, 21].

5 Related Work

Methods for SSL are often based on assumptions regarding the structure of the unlabeled data. One such assumption is smoothness, which states that examples that are close are likely to have similar labels. In the classic Label Propagation algorithm [46], adjacent nodes in the graph are encouraged to agree on their labels via a quadratic penalty. Some variants add regularization terms [4], allow for label uncertainty [41], or include normalization and unanchored seeds [45].

The above methods are designed for graphs that approximate the data density via similarity in feature space, and are typically constructed from samples. Recent SSL methods are geared towards tasks where graphs are an additional part of the input. Motivated by deep embeddings [31], these methods embed the nodes of a graph into a low-dimensional vector space, which can then be used in various ways. When the data includes only the graph, the embeddings can be used as input for an off-the-shelf predictor [35]. When the data includes additional node features, the embedding can act as a regularizer for a standard loss over the labeled nodes [44, 27]. In contrast to classic methods, these methods propagate features rather than labels.

An alternative method for utilizing graphs is to consider shortest paths as a measure of closeness. The authors of [1] show that Laplacians and shortest paths are special cases of “resistance distances”, and propose (but do not evaluate) a new regularizer. Other methods construct ad-hoc graphs whose shortest paths approximate density-based distances [34, 6]. A recent work [15] proposes a method for SSL in directed graphs based on distance diffusion. As they consider distances from unlabeled to labeled nodes, each instance is computationally intensive, and requires an approximation scheme. In contrast, we consider distances from labeled to unlabeled nodes, which can be computed efficiently. While for a specific setting (symmetric weights and a certain link function) both models overlap, in this paper we consider a more general setup.

Our method draws on the rich literature of infection models and diffusion processes over networks. These have been used for describing the propagation of information, innovation, behavioral norms, and others, and have been utilized in works in influence maximization [25], network inference [20], influence maximization [25] estimation [17, 16] and prediction [37], and personalized marketing [14].

6 Experiments

We evaluated our method on various learning tasks over three benchmark dataset collections, which include networked data for multiclass learning with features [39] and without features [38], and multilabel learning [33]. The datasets include diverse networks such as social networks, citation and co-authorship graphs, product and item networks, and hyperlink

Figure 3: Activation tradeoff and convergence

Figure 2: Results on the CoRA dataset for various learning settings.
graphs (see supplementary material for dataset summary statistics).

Our experimental setup follows the standard graph-based semi-supervised learning evaluation approach. Specifically, in each instance we draw a seed set of size $k$ uniformly at random, acquire its labels, and then use the graph and labeled seed set to generate labels for all nodes. We repeat this procedure for 10 random seed set selections and for various values of $k$ (where $k$ is set to be a fixed proportion of the number of nodes in the graph) and report average results.

We compared our method to current state-of-the-art baselines, which include spectral methods as well as deep embedding methods. For tasks which do not include features, these included LABELPROP [46], ADOPTION [4], MAD [41], and the feature-agnostic deep method DEEPWALK [35]. For tasks which do include features, we compared to the prior-supporting spectral method LLGC [45], the recent feature-based deep method PLANETOID [44], LABELPROP as a graph-only baseline, logistic regression (LOGREG) as a features-only baseline, and a baseline where labels are set by shortest paths in $G$ (SHORTPATHS). For the active setting (Fig. 2), we compared our approach (GREEDY) to METIS [22], to choosing high-degree nodes (HiDeg), and to random seeds (RAND).

For our method (INFPROP) we used exponential incubation times $\delta \sim \text{Exp}(\theta)$. As in many works (e.g., [25, 15]), we used $\theta_{uv} = 1/d_u$ for all node pairs $(u, v) \in E$, where $d_u$ is the out-degree of $u$. We set the number of random instances to $N = 1000$. Fig. 3 (right) demonstrates accuracy and convergence as a function of $N$. We show results for two variants: INF-Prop, where we set activation probabilities to $p = 1$ for all edges, and INFPROP$_{0.5}$, where $p = 0.5$. In addition to providing a confidence measure, INFPROP$_{0.5}$ is much faster, while on average achieving 0.99% of the performance of INFPROP. Fig. 3 (left) demonstrates the tradeoff in accuracy and runtime when varying $p$.

The methods we consider naturally output probabilistic “soft” labels as predictions. We therefore evaluate performance using both probabilistic (for multi-class) or order-based (for multi-label) performance measures, as well as performance measures for “hard” labels, which were generated by choosing the label with the highest value. Tables 1 and 2 include results for all datasets for $k = 1\%$ of the data. Fig. 2 shows results for various values of $k$ on the CoRA dataset (which appears in all benchmarks). As shown, INFPROP consistently performs well across all settings.

### 7 Conclusions

In this work we presented an SSL method where labels propagate over the graph using dynamic infection models. These models have a strong connection to short-path ensembles and to graph Laplacians, allow for efficient computation, and show empirical potential. Our work was motivated by the idea that different graph types may require different dynamics, which led us to consider alternatives to random walks and averaging dynamics. We used a competitive CTIC variant, but other infection models (and other dynamics in general) can be considered. The choice of dynamics can serve as a means for expressing prior knowledge and for encoding structure and dependencies.

The models we use have very few tunable parameters. Nonetheless, one can consider highly parametrized models. Such parameters can be used to control infection probabilities, be node or label specific, relate to features, and even adjust the dynamics themselves. The stochastic nature of the models and the nonlinearity of the dynamics makes learning these parameters a challenging task, which we leave for future work.

### Acknowledgments

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**Table 1: Results for experiments on data without features.**

<table>
<thead>
<tr>
<th>Labeling Type</th>
<th>Multiclass (Accuracy / MSE)</th>
<th>Multilabel (AUC / Top-1)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CoRA</td>
<td>DBLP</td>
</tr>
<tr>
<td>InfProp</td>
<td>0.59 / 0.56</td>
<td>0.75 / 0.42</td>
</tr>
<tr>
<td>InfProp$_{0.5}$</td>
<td>0.58 / 0.64</td>
<td>0.74 / 0.46</td>
</tr>
<tr>
<td>ShortPaths</td>
<td>0.53 / 0.87</td>
<td>0.63 / 0.74</td>
</tr>
<tr>
<td>LabelProp</td>
<td>0.41 / 0.74</td>
<td>0.60 / 0.59</td>
</tr>
<tr>
<td>Adsorption</td>
<td>0.42 / 0.99</td>
<td>0.54 / 0.99</td>
</tr>
<tr>
<td>MAD</td>
<td>0.45 / 0.99</td>
<td>0.20 / 1.00</td>
</tr>
<tr>
<td>DeepWalk</td>
<td>0.29 / 0.86</td>
<td>0.77 / 0.62</td>
</tr>
</tbody>
</table>

**Table 2: Results on data with features.**

<table>
<thead>
<tr>
<th>Labeling Type</th>
<th>CiteSeer</th>
<th>CoRA</th>
<th>PubMed</th>
</tr>
</thead>
<tbody>
<tr>
<td>INFProp</td>
<td>0.47 / 0.72</td>
<td>0.62 / 0.59</td>
<td>0.74 / 0.46</td>
</tr>
<tr>
<td>INFProp$_{0.5}$</td>
<td>0.48 / 0.74</td>
<td>0.60 / 0.57</td>
<td>0.72 / 0.41</td>
</tr>
<tr>
<td>SHORTPATHS</td>
<td>0.39 / 0.73</td>
<td>0.44 / 0.72</td>
<td>0.68 / 0.51</td>
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<tr>
<td>LOGREG</td>
<td>0.44 / 0.78</td>
<td>0.37 / 0.81</td>
<td>0.45 / 0.65</td>
</tr>
<tr>
<td>LABELPROP</td>
<td>0.39 / 0.77</td>
<td>0.38 / 0.78</td>
<td>0.40 / 0.67</td>
</tr>
<tr>
<td>LLGC</td>
<td>0.45 / 0.71</td>
<td>0.49 / 0.69</td>
<td>0.44 / 0.67</td>
</tr>
<tr>
<td>PLANETOID$^{2}$</td>
<td>0.41 / 0.94</td>
<td>0.53 / 0.89</td>
<td>0.68 / 0.64</td>
</tr>
</tbody>
</table>

$^{2}$Results differ from [44] since their evaluation is based on a specific seed, chosen by a different procedure, evaluated on 1000 samples, and early-stopped differently.
References


