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Performance modeling of epidemic routing

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Abstract

In this paper, we develop a rigorous, unified framework based on ordinary differential equations (ODEs) to study epidemic routing and its variations. These ODEs can be derived as limits of Markovian models under a natural scaling as the number of nodes increases. While an analytical study of Markovian models is quite complex and numerical solution impractical for large networks, the corresponding ODE models yield closed-form expressions for several performance metrics of interest, and a numerical solution complexity that does not increase with the number of nodes. Using this ODE approach, we investigate how resources such as buffer space and the number of copies made for a packet can be traded for faster delivery, illustrating the differences among various forwarding and recovery schemes considered. We perform model validations through simulation studies. Finally we consider the effect of buffer management by complementing the forwarding models with Markovian and fluid buffer models.

Keywords: Delay tolerant networks; Disruption tolerant networks; Wireless ad hoc networks; Epidemic routing; Performance modeling; Ordinary differential equations

1. Introduction

Epidemic routing [28] has been proposed as an approach for routing in sparse and/or highly mobile networks in which there may not be a contemporaneous path from source to destination. It adopts a so-called "store-carry-forward" paradigm – a node receiving a packet buffers and carries that packet as it moves, passing the packet on to new nodes that it encounters. Analogous to the spread of infectious diseases, each time a packet-carrying node encounters a node that does not have a copy of that packet, the carrier is said to *infect* this new node by passing on a packet copy; newly infected nodes, in turn, behave similarly. The destination receives the packet when it first meets an infected node. When the traffic load is very low, epidemic routing is able to achieve minimum delivery delay at the expense of increased use of resources such as buffer space, bandwidth, and transmission power. However this also leads to link and/or storage congestion when the network

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is loaded. Variations of epidemic routing have recently been proposed that exploit the tradeoff between delivery delay and resource consumption, including K-hop schemes [23,6], probabilistic forwarding [17,8], and spray-and-wait [26,25]. These different schemes differ in their "infection process", i.e., the spreading of a packet in the network. They need to be combined with a so-called "recovery process" that deletes copies of a packet at infected nodes, following the successful delivery of the packet to the destination. Different recovery schemes have been proposed: some are simply based on timers, others actively spread in the network the information that a copy has been delivered to the destination [8].

Early efforts evaluating the performance of epidemic routing schemes used simulation [28,10,17]. More recently, Markovian models have been developed to study the performance of epidemic routing [24,6,8], 2-hop forwarding [6], and spray-and-wait [26,25]. Recognizing the similarities between epidemic routing and the spread of infectious diseases, [24,8] used ordinary differential equation (ODE) models adapted from infectious disease-spread modeling [3] to study the source-to-destination delivery delay under the basic epidemic routing scheme, and then adopted Markovian models to study other performance metrics.

In this paper, we develop a rigorous, unified framework, based on ordinary differential equations (ODE), to study epidemic routing and its variations. The starting point of our work is [6], where the authors consider common node mobility models (e.g., random waypoint and random direction mobility) and show that nodal inter-meeting times are nearly exponentially distributed when transmission ranges are small compared to the network area, and node velocity is sufficiently high. This observation suggests that Markovian models of epidemic routing can lead to quite accurate performance predictions; indeed [6] develops Markov chain models for epidemic routing and 2-hop forwarding, deriving the average source-to-destination delivery delay and the number of extant copies of a packet at the time of delivery. An analytical study of such Markov chain models is quite complex for even simple epidemic models, and more complex schemes have defied analysis thus far. Moreover, numerical solution of such models becomes impractical when the number of nodes is large.

We develop ODEs as a fluid limit of Markovian models such as [6], under an appropriate scaling as the number of nodes increases. Through the paper we show that ODE is a valid tool for investigating epidemic style routing. In fact this approach allows us to derive closed-form formulas for the performance metrics considered in [6], obtaining matching results. More importantly, we are also able to use the ODE framework to further model the recovery process, to study more complex variants of epidemic routing, and to model the performance of epidemic routing with different buffer management schemes under buffer constraints. While different recovery processes are studied also in [8] using Markov chains, model simulation is first needed to determine a number of model parameters. Many of our ODE models can be analytically solved, providing closed-form formulas for the performance metrics of interest; in cases where we resort to numerical solution, the computational complexity does not increase with the number of nodes. The drawback of our ODE models is that they provide the moments of the various performance metrics of interest, while numerical solution of Markov chain models can provide complete distributions (e.g., for the number of packet copies in the system). Simulation results show good agreement with the predictions of our ODE models.

The main purpose of the paper is to show how ODE models can be advantageously employed to study the performance of various epidemic style routing schemes, rather than to provide final conclusions about the merits of specific schemes. Nevertheless we have obtained insights into different epidemic routing schemes through our models. In particular, we have identified rules of thumb for configuring these schemes, we have shown the existence of a linear relation between total number of copies sent and the buffer occupancy under certain schemes, and we have demonstrated that the relative benefit of different recovery schemes depends strongly on the specific infection process. Finally our analysis of buffer-constrained epidemic routing suggests that sizing node buffers to limit packet loss is not vital as long as appropriate buffer management schemes are used.

The remainder of this paper is structured as follows. Basic epidemic routing and our basic ODE model are described and derived in Section 2, allowing one to characterize the source-to-destination delivery delay, the number of copies made for a packet, and the average buffer occupancy. In Section 3, it is shown how the ODE model can be easily extended to three important variations of

basic epidemic routing (K-hop forwarding, probabilistic forwarding and limited-time forwarding), to the global timeout scheme for deleting anti-packets, and to include signaling overheads. In Section 4, we perform validation for these models through simulation. We use these extended models to characterize the tradeoff between delivery delay and resource consumption (buffer occupancy, number of copies made) in Section 5. In Section 6, we integrate the ODE models with Markov and fluid buffer models to study the effect of finite buffers, and compare different buffer management strategies. In Section 7, we review related works and compare our work with them. Finally, in Section 8 we summarize the paper.

2. Basic epidemic routing

In this section we develop our ODE model for basic epidemic routing [28], after briefly describing epidemic routing and the scenario we are considering. We then use the model to study three different recovery techniques for deleting packet copies after the delivery of the packet.

We consider a set of $N+1$ nodes, each with a finite transmission range, moving in a closed area, and different source–destination pairs. We say that two nodes "meet" when they come within transmission range of each other, at which point they can exchange packets. Let us focus on a single packet. The analogy with disease spreading is useful in describing epidemic routing. The source of the packet can be viewed as the first carrier of a new disease, the first *infected* node, which copies the packet to (infects) every node it meets. These new infected nodes act in the same way. As a result, the population of *susceptible* nodes (i.e., nodes without a copy of the packet) decreases over time. Once a node carrying the packet meets the destination, it passes the packet on to the destination, deletes the packet from its own buffer, and retains "packet-delivered" information (an "anti-packet") which will prevent it from receiving another copy of this packet in the future. Such a node is said to have *recovered* from the disease. Here the recovery process simply relies on meeting with the destination. We will shortly consider more sophisticated recovery schemes.

Consider now many packets spreading at the same time in the network. We assume that when two nodes meet they can exchange an arbitrary number of packets, and each node has enough buffer to store all packets (the latter assumption is relaxed in Section 6), thus allowing different infections to be considered independently. We also assume a mechanism exists so that nodes never exchange a packet if both nodes are already carrying a copy of that packet (more details in Section 3.3).

2.1. ODE models for basic epidemic routing

As noted earlier, [6] showed that the pairwise meeting time between nodes is nearly exponentially distributed, if nodes move in a limited region (of area A) according to common mobility models (such as the random waypoint or random direction model [2]) and if their transmission range (d) is small compared to A, and their speed is sufficiently high. The authors also derived the following estimation of the pairwise meeting rate β:

$$\beta \approx \frac{2wdE[V^*]}{A}, \tag{1}$$

where w is a constant specific to the mobility model, and $E[V^*]$ is the average relative speed between two nodes. Under this approximation, [6] showed that the evolution of the number of infected nodes can be modeled as a Markov chain.

We introduce our modeling approach starting from the Markov model for basic epidemic routing before the delivery of a copy to the destination. Given $n_I(t)$, the number of infected nodes at time t, the transition rate from state n_I to state n_I+1 is $r_N(n_I) = \beta n_I(N-n_I)$, where N is the total number of nodes in the network (excluding the destination). If we rewrite the rates in a "density dependent form", as $r_N(n_I) = N\lambda(n_I/N)\,(1-n_I/N)$ and assume that $\lambda = N\beta$ is constant, we can apply Theorem 3.1 in [16] to prove that, as N increases, the fraction of infected nodes (n_I/N) converges asymptotically to the solution of the following equation:[1]

$$i'(t) = \lambda i(t)(1-i(t)), \quad \text{for } t \geqslant 0 \tag{2}$$

with initial condition $i(0) = \lim_{N\to\infty} n_I(0)/N$. The average number of infected nodes then converges to $I(t) = Ni(t)$ in the sense of Footnote 1. The following equation can be derived for $I(t)$ from Eq. (2):

$$I'(t) = \beta I(N-I) \tag{3}$$

with initial condition $I(0) = Ni(0)$. Such an ODE, which, as we have shown, results as a fluid limit of a Markov model as N increases, has been commonly used in epidemiology studies, and was first applied

[1] Formally, $\forall \epsilon > 0$, $\lim_{N\to\infty} \text{Prob}\{|\sup_{s\leqslant t}\{n_I(s)/N - i(s)\}| > \epsilon\} = 0$.

to epidemic routing in [24] as a reasonable approximation.

We remark that (1) the initial population of infected nodes must scale with N, and (2) the pairwise meeting rate must scale as $1/N$. Eq. (1) provides insight into the physical interpretation of this meeting rate scaling: in particular if the area A increases with N, keeping node density constant, then β scales with $1/A$, i.e., $1/N$. In the following we will consider Eq. (3) with initial condition $I(0) = 1$, which corresponds to an initial fraction of infected nodes $i(0) = 1/N$. Despite the "small" number of initial infected nodes, we will see via our simulation results that the approximation is a good one. We also note that Eq. (3), as well as other related equations we will derive shortly, can also be obtained in a different manner from Markovian models by neglecting terms related to higher moments (the details are given in Appendix C).

2.2. Delay under epidemic routing

Let T_d be the packet delivery delay, i.e., the time from when a packet is first generated at the source to the time when it is first delivered to the destination, and denote its Cumulative Distribution Function (CDF) by $P(t) = \text{Prob}(T_d < t)$. Under the same scaling and approximations considered earlier, we can derive the following equation for $P(t)$: $P'(t) = \lambda i(1 - P)$, where $i(t)$ is the solution of Eq. (2). Let us consider $P_N(t)$, the CDF of T_d when the number of nodes in the system is $N + 1$, i.e., there are N nodes plus one destination node. We have

$$\begin{aligned}
&P_N(t+\mathrm{d}t) - P_N(t)\\
&\quad = \text{Prob}\{t \leqslant T_d < t+\mathrm{d}t\}\\
&\quad = \text{Prob}\{\text{destination meets an infected node in } [t, t+\mathrm{d}t] | T_d > t\}\\
&\qquad \times \text{Prob}\{T_d > t\}\\
&\quad = \text{Prob}\{\text{destination meets one of the } n_I(t) \text{ infected nodes in}\\
&\qquad [t, t+\mathrm{d}t]\} \times (1 - P_N(t))\\
&\quad = E[\text{Prob}\{\text{destination meets one of the } n_I(t) \text{ infected nodes in}\\
&\qquad [t,\ t\ +\ \mathrm{d}t] | n_I(t)\}] \times (1 - P_N(t)) \approx E[\beta n_I(t)\,\mathrm{d}t](1 - P_N(t))\\
&\quad = \beta E[n_I(t)](1 - P_N(t))\,\mathrm{d}t = \lambda E\left[\frac{n_I(t)}{N}\right](1 - P_N(t))\mathrm{d}t.
\end{aligned}$$

Note that $n_I(t)$ is the number of infected nodes at time t, given that the destination has not received a copy of the packet. It implicitly accounts for the condition $T_d > t$. The following holds for $P_N(t)$:

$$\frac{\mathrm{d}P_N}{\mathrm{d}t} = \lambda E\left[\frac{n_I(t)}{N}\right](1 - P_N(t)).$$

As N increases, $E[n_I(t)/N]$ converges to $i(t)$, and $P_N(t)$ converges to the solution of

$$P'(t) = \lambda i(t)(1 - P(t)).$$

For a finite population of size N we can consider:

$$P'(t) = \beta I(t)(1 - P(t)). \tag{4}$$

Eq. (4) was proposed in [24], based on an analogy with a Markov process. Solving Eqs. (3) and (4) with $I(0) = 1$, $P(0) = 0$ yields

$$I(t) = \frac{N}{1 + (N-1)\mathrm{e}^{-\beta N t}}, \quad P(t) = 1 - \frac{N}{N - 1 + \mathrm{e}^{\beta N t}}.$$

From $P(t)$, the average delivery delay can be explicitly found as

$$\mathrm{E}[T_d] = \int_0^\infty (1 - P(t))\,\mathrm{d}t = \ln N/(\beta(N-1)). \tag{5}$$

The average number of copies of a packet in the system when the packet is delivered to the destination under epidemic routing, $E[C_{\text{ep}}]$, can also be derived, as it coincides with the average number of infected nodes in the system, apart from the source, when the packet is delivered (details given in Appendix E): $E[C_{\text{ep}}] = \int_0^\infty I(t)P'(t)\,\mathrm{d}t - 1 = \frac{N-1}{2}$.

Using a Markov chain model, [6] obtained the same results for the number of copies, computed the Laplace–Stieltjes Transform (LST) of the delay, and from the LST found the following asymptotic expression for the average delay as $N \to \infty$: $\frac{1}{\beta(N-1)}(\ln N + \gamma + \mathrm{O}(\frac{1}{N}))$, matching Eq. (5). We note that the derivation is much simpler using our ODE model.

2.3. Recovery from infection

In the last section, we studied the delivery delay, and the number of copies made at delivery time under epidemic routing. In this section, we study the recovery schemes proposed in [8].

Clearly, once a node delivers a packet to the destination, it should delete the packet from its buffer to save storage space and prevent the node from infecting other nodes. Moreover, to avoid being reinfected by the packet, a node can keep track of packet delivery. We refer to this information stored at the node as "anti-packet", and refer to this scheme of handling already-delivered packets as the IMMUNE scheme. A more aggressive approach toward deleting obsolete copies is to propagate anti-packets among nodes. An anti-packet can be propagated only to infected nodes (which we will refer to

Table 1
Summary of closed-form expressions obtained for different schemes

Schemes	$I(t)$ / $P(t)$	$E[T_d]$	$E[C]$,$E[G]$	$E[Q]$
Epidemic	$I(t)=\frac{N}{1+(N-1)e^{-\beta Nt}}$ $P(t)=1-\frac{N}{N-1+e^{\beta Nt}}$	$\frac{\ln N}{\beta(N-1)}$	$E[C]=\frac{N-1}{2}$ $E[G]\approx N-1$(IM) $E[G]=\frac{N-3}{2}+\frac{\sqrt{N^2-2N+5}}{2}$(IM_TX)	$\approx N\lambda/\beta(IM)$ $\approx \lambda\frac{N-1+\sqrt{N^2-2N+5}}{2\beta}$(IM_TX)
2-hop	$I(t)=N-(N-1)e^{-\beta t}$ $P(t)=1-e^{N-1-\beta Nt-(N-1)}e^{-\beta t}$	$\frac{1}{\beta}\sqrt{\frac{\pi}{2}}\frac{1}{\sqrt{N-1}}$	$E[C]=\sqrt{\frac{\pi}{2}}\sqrt{N}, G=\frac{N-1}{2}$(IM)	$\frac{\lambda(N+1)}{2\beta}$(IM)
Prob. forwarding	$I(t)=\frac{N}{1+(N-1)e^{-p\beta Nt}}$ $P(t)=1-\left(\frac{N}{N-1+e^{p\beta Nt}}\right)^{1/p}$	$\left[\frac{\ln(N)}{\beta(N-1)},\frac{\ln(N)}{\beta p(N-1)}\right]$	$E[C]=\frac{p(N-1)}{1+p}$	
Limited-time forwarding (no reinfection)	$I(t)=\frac{a_2+e^{\beta(a_2-a_1)t}+A}{A+e^{\beta(a_2-a_1)t}}$ $a_{1,2}=\frac{(\beta N-\mu)\mp\sqrt{(\beta N-\mu)^2+4\beta\mu}}{2\beta}$ $a_1<0, a_2>0, A=\frac{a_2-1}{1-a_1}$	$\underset{N\to\infty}{\sim}\frac{1}{\beta}\frac{\ln(N-\frac{\mu}{\beta})}{N-\frac{\mu}{\beta}}$ $\underset{\mu\to\infty}{\sim}\frac{\mu-N\beta}{\beta\mu}$ $\underset{N=\frac{\mu}{\beta}\to\infty}{\sim}\frac{\pi}{2\beta\sqrt{N-2}}$		
Global timeout	$P(t)=1-\frac{N}{N-1+e^{\beta Nt}}, t\leqslant T$ $P(t)=1-e^{\beta b(T-t)}$ $\frac{N}{N-1+e^{\beta NT}}, t>T, b=I(T)$	$\frac{\ln(N)}{\beta(N-1)}-\frac{\ln(1+(N-1)e^{-\beta NT})}{\beta(N-1)}+\frac{1}{\beta e^{\beta NT}}$	$E[C]=\frac{N-1}{2}-\frac{N^2(N-1)}{2(e^{\beta TN}+N-1)^2}$	
Global timeout(2)	$P(t)=1-\frac{N}{N-1+e^{\beta Nt}}, t\leqslant T$ $P(t)=1-\frac{Ne^{\beta(T-t)}}{N-1+e^{\beta NT}}, t>T$	$\frac{\ln(N)}{\beta(N-1)}-\frac{\ln(1+(N-1)e^{-\beta NT})}{\beta(N-1)}$ $+\frac{N}{\beta(e^{\beta TN}+N-1)}$	$E[C]=\frac{N-1}{2}-\frac{N^2(N-1)}{2(e^{\beta TN}+N-1)^2}$	

as the IMMUNE_TX scheme), or to both infected and susceptible nodes (VACCINE scheme). We study the following two metrics for epidemic routing under these different recovery schemes. One is the average number of times a packet is copied during its lifetime, excluding the copy to the destination, denoted as $E[G]$. This value is greater than or equal to $E[C]$, because more copies can be made after the delivery to the destination. This metric is strongly related to the bandwidth requirement, and transmission power consumption of a specific scheme. The other is the average buffer occupancy at each node $E[Q]$, for which we are going to derive an expression under a specific traffic pattern. The two metrics are related each other and they both depend on the specific recovery process.

In order to study these two metrics, similar to our earlier analysis in Section 2.1, we can derive ODEs that take into account the recovery processes as the limit of Markov models (details are deferred to Appendix A), with the additional consideration that we need to scale the number of destinations n_D in a manner similar to the scaling of the number of initially infected nodes, i.e., $\lim_{N\to\infty} n_D/N = d$. For example, if we consider the IMMUNE scheme, the number of infected and recovered nodes should be respectively close to $I(t)$ and $R(t)$, which are solutions of the following equations:

$$I'(t) = \beta I(N - I - R) - \beta ID, \tag{6}$$

$$R'(t) = \beta ID, \tag{7}$$

where D is the number of destinations, and we consider $I(0) = 1$, $R(0) = 0$, $D = 1$. This model allows us to evaluate the average number of times that a packet is copied during its lifetime, $E[G_{ep}]$. In fact the total number of copies made for a packet equals the number of nodes that have ever been infected, i.e., $E[G_{ep}] = \lim_{t\to\infty}(I(t) + R(t)) - I(0)$. A good approximation for $E[G_{ep}]$ can be found through the previous equations by expressing I as a function of R, without the need to solve for $I(t)$ and $R(t)$. Analogous ODEs can be derived for the IMMUNE_TX and VACCINE schemes, and a closed formula can be derived for $E[G_{ep}]$ for the IMMUNE_TX scheme. Numerical solutions are needed for the VACCINE scheme (see Table 1 for closed-form results and Appendix B for the detailed derivations).

We next consider the average buffer occupancy $E[Q]$, in the case of $N + 1$ unicast flows, with each node being the source of one flow and destination for one other flow. The packet generation process in each flow is a Poisson process with rate λ. Denote by L the average packet lifetime (the time from when the packet is generated by the source node to when all copies of the packet are removed from the system). The average number of copies of a packet in the system during its lifetime is given by $\int_0^\infty I(t)\,dt/L$, where $I(t)$ is the solution to the ODEs that include the recovery process. As the total arrival rate of new packets to the system is $(N + 1)\lambda$, by Little's law, the average number of packets in the system is $(N + 1)\lambda L$. Therefore the average total buffer occupancy in the whole network is given by $E[Q_{total}] = (\int_0^\infty I(t)dt/L)(N + 1)\lambda L = \int_0^\infty I(t)\,dt(N + 1)\lambda$, and the per-node buffer occupancy is thus $E[Q] = \lambda \int_0^\infty I(t)dt$.

Modeling a node's buffer as an $M/M/\infty$ queue gives the same result and shows a linear relationship between the average buffer occupancy and the number of copies made under the IMMUNE scheme. In fact, given that each packet is copied $E[G_{ep}]$ times, each flow generates relay traffic at rate $E[G_{ep}]\lambda$, and the total rate of relay traffic in the network is $E[G_{ep}]\lambda(N + 1)$ (as there are $N + 1$ flows). This traffic is equally divided among the $N + 1$ nodes, hence the arrival rate of relay packets to each node is $E[G_{ep}]\lambda$, and the total packet arrival rate is $\lambda(1 + E[G_{ep}])$. If a copy is deleted only when the node meets the destination,[2] the service rate is $1/\beta$ and the average buffer occupancy is $E[Q] = \frac{\lambda}{\beta}(1 + E[G_{ep}])$.

3. Extended model

The schemes in the previous section all share the same infection process: they propagate a packet among nodes in a flooding/epidemic manner, but differ in the way they counteract the infection after the packet has been delivered to the destination. As results in Table 1 show, this can lead to substantial differences in terms of buffer occupancy and the total number of copies made for a packet. Depending on the specific applications, it might be preferable to trade off timely delivery for savings in resource consumption, by changing the way packets are propagated among nodes. We describe in Section 3.1 K-hop forwarding, probabilistic forwarding and limited-time forwarding that allow us to achieve such tradeoff. In Section 3.2, we introduce the global timeout scheme that naturally addresses the

[2] This is the case under IMMUNE for the basic epidemic routing, and also for the probabilistic and K-hop forwarding schemes we will consider.

problem of deleting anti-packets. We discuss how ODE models can be used to model signaling overhead in Section 3.3. All the ODEs models we propose can be derived as limits of Markovian models, similarly to what we have shown in Section 2.1. We do not detail the derivations, but only stress the peculiarities (if any) to be taken into account when applying the limiting theorem.

3.1. Trade-off schemes

3.1.1. K-hop forwarding

Under K-hop forwarding, a packet can traverse at most K hops to reach the destination. We can use ODE models to model the K-hop forwarding scheme, as we demonstrate for $K = 2$. Under 2-hop forwarding, the source copies the packet to every node it meets until it meets the destination; relay nodes do not copy the packet to any other node except the destination. As the packet spreads at a rate proportional to the number of susceptible nodes, the following equations model the delivery delay:

$$I'(t) = \beta(N - I),$$
$$P'(t) = \beta I(1 - P)$$

with initial condition: $I(0) = 1$ and $P(0) = 0$. Note that in order to derive the previous equations from the Markovian model similarly to what we did in Section 2.1, we need to let the number of source nodes scale with N.

This ODE system can be solved explicitly, from which we can then derive an asymptotic expression for the average delivery delay and the average number of copies until delivery (see Table 1 for the results and Appendix D for the derivation). These results again match those obtained in [6] using a Markov Chain model.

We can apply analysis similar to Section 2.3 to study the number of copies made and the average buffer occupancy for given recovery schemes. For IMMUNE recovery, we obtain more accurate model through the following derivations. Let $G_{2\text{hop}}(N)$ be the number of times a packet is copied during its life time (excluding the copy to the destination) for 2-hop forwarding. For each packet, the source node copies the packet to every relay node it meets before it meets the destination. Therefore $G_{2\text{hop}}(N)$ equals the number of nodes the source node meets before meeting the destination. As the inter-meeting times between pairs of nodes are i.i.d. exponential random variables, the destination node is equally likely to be the ith node to meet the source node, for $i = 1, \ldots, N$. Therefore we have $\Pr(G_{2\text{hop}}(N) = i) = \frac{1}{N}$, for $i = 0, \ldots, N-1$, and hence $E[G_{2\text{hop}}(N)] = \frac{N-1}{2}$. Given $G_{2\text{hop}}(N)$, we can derive the average buffer occupancy using a $M/M/\infty$ model with the departure rate β, using an approach similar to what we described in Section 2.3.

3.1.2. Probabilistic forwarding

Probabilistic forwarding is similar to epidemic routing except that when two nodes meet, each node accepts a relay packet with probability p. When $p = 0$, the probabilistic forwarding degenerates to direct source–destination delivery, and when $p = 1$, epidemic routing is performed. Varying p in the range $(0, 1)$ allows a trade-off between storage/transmission requirements and delivery delay. We can model the delivery delay using the following ODEs:

$$\frac{dI}{dt} = \beta p I(N - I),$$
$$\frac{dP}{dt} = \beta I(1 - P)$$

with $I(0) = 1$, $P(0) = 0$. We derived a closed-form solution for this ODEs, from which we then derived bounds for the average delay, and close-form formula for the number of copies at delivery time (Table 1). Similar to basic epidemic routing case, we derived a ODE model to study G_{prob} and the average buffer occupancy under probabilistic forwarding.

3.1.3. Limited-time forwarding

Under limited-time forwarding, when a node accepts a packet copy, it starts a timer with duration drawn from an exponential distribution with rate μ. When the timer expires, the copy is deleted from the buffer. The choice of timeout value allows us to trade off the delivery delay against storage and number of transmissions. In order to guarantee the eventual delivery of each packet, a node does not time out a packet for which it is the original source. When a packet copy in a node times out, the node can either store an anti-packet (so that it will not be infected by the packet again), or keep no information (in which case it become susceptible to the packet again).

The former scheme can be studied by the following ODEs, where $T(t)$ is the number of timed out nodes at time t. As above these ODEs can be derived as limit of Markovian models.[3]

[3] There is no need to scale the timer rate μ, while we need to scale β as we noted in Section 2.1.

$$\frac{dI}{dt} = \beta I(N - I - T) - \mu(I - 1),$$
$$\frac{dT}{dt} = \mu(I - 1),$$
$$\frac{dP}{dt} = \beta I(1 - P).$$

We numerically solved this ODEs to calculate the average delivery delay, $E[T_d]$. Similar to epidemic routing, by extending the ODEs to include recovery processes, we are able to evaluate numerically the average number of copies made for a packet $E[G]$ and the average buffer occupancy $E[Q]$.

The latter scheme can be studied using the following ODEs:

$$\frac{dI}{dt} = \beta I(N - I) - \mu(I - 1),$$
$$\frac{dP}{dt} = \beta I(1 - P).$$

The ODEs can be solved explicitly and an asymptotic expression for the average delay can be found (see Table 1 for the results, and Appendix D for details).

We found that if $\mu \geqslant N\beta$ the number of infected nodes goes to zero as $t \to \infty$. In this case limited-time forwarding can perform recovery via timeout and there is no need for explicitly transmitted anti-packets, the epidemic spreading will eventually die out in this case. The asymptotic delay for $\mu = N\beta$ equals $\frac{\pi}{2\beta\sqrt{N-2}}$ (see Appendix D).

3.2. Handling anti-packets: global timeout scheme

Under the recovery schemes, IMMUNE, IMMUNE_TX and VACCINE (Section 2.3), nodes store and propagate anti-packets to delete obsolete packet copies in order to save buffer space and number of copies sent for a packet. Although anti-packets are typically much smaller than data packets, a way is needed to delete anti-packets: otherwise, the buffer space taken up by anti-packets will grow infinitely. In this section, we describe a global timeout scheme for deleting anti-packets.

Under the global timeout scheme, as the name suggests, there is a global timer associated with each packet: acting upon the copies and anti-packets for the packet stored at all the nodes. Before the timer expires, the packet is propagated according to the forwarding scheme employed. When the timer expires, all anti-packets will be deleted; the infected nodes keep their copies of the packet, but can only forward the copy to the destination. Notice that as there is no relaying after time T, nodes do not need to keep anti-packets from then on.

As [24] suggested, a global timer can be implemented as follows.[4] The source node sets a TTL (Time-To-Live) field to duration T for each packet generated. The TTL field is decreased as time passes. Whenever the packet is copied to another node, the new copy's TTL field is set to the remaining TTL field of the old copy; when an anti-packet is generated at the destination, its TTL field is set to the same value as the data packet being delivered.

The global timeout scheme is similar to spray and wait [26] in that both schemes have two phases: epidemic style forwarding phase and direct delivery phase. While spray and wait limits the spreading by specifying the maximum number of copies, our scheme limits the spreading by setting a duration. We will see in Section 5 that varying the timeout value T allows a tradeoff between delivery delay and resource consumption.

We now demonstrate how ODEs can be used to model this global timeout scheme using the example of epidemic routing with IMMUNE recovery. As usual, let $I(t)$ be the average number of infected nodes at time t, given that the packet has not been delivered; and $P(t)$ be the CDF of delivery delay. Before the timer expires, the packet propagates according to epidemic routing; while after the timer expires, the packet can be only forwarded to the destination. Therefore, $I(t)$, $P(t)$ satisfy the following ODEs:

$$I'(t) = \beta I(t)(N - I(t)), \quad t \leqslant T,$$
$$I'(t) = 0, \quad t > T,$$
$$P'(t) = \beta I(t)(1 - P(t)).$$

The initial conditions are $I(0) = 1$, $P(0) = 0$. When deriving these ODEs from the Markovian model, one has to take into account that the system has time-dependent transition rates (in particular they changes at time T). Nevertheless the same kind of convergence holds. It can be proven by applying Theorem 3.1 in [16] separately to the system trajectories before time T and after time T and then appropriately join them.

The ODEs can be explicitly solved, and allow us to derive the average delivery delay and the number

[4] Under the scheme they considered, when the packet timer expires, all copies and anti-packets of the packet are deleted from the network. We note that there is a non-zero probability that the packet is not delivered to the destination.

of copies made for a packet at delivery time (see Table 1 for the results). After time T, the packet can only be forwarded to the destination, hence the total number of copies made for a packet (exclude the copy to the destination) is given by $G_{gt} = I(T) + R(T) - I(0)$ under IMMUNE and IMMUNE_TX scheme, where $I(t)$ and $R(t)$ are solutions to Eqs. (A.1)–(A.4) respectively. Under VACCINE, the total number of copies made is given by $C(T)$, where $C(t)$ is solution to Eq. (A.8) in Appendix A. For the average buffer occupancy, $E[Q]$, the following equation (derived in Section 2.3) still applies: $E[Q] = \lambda \int_0^\infty I(t)\,dt$.

An alternative scheme is to delete all anti-packets and copies of the packet, except at the source node, when the timer expires. Compared to the previous scheme, this scheme saves buffer space but incurs larger delivery delay. Under this scheme, $P(t)$ satisfies the following ODE:

$$P'(t) = \beta I(t)(1 - P(t)), \quad t \leqslant T,$$
$$P'(t) = \beta(1 - P(t)), \quad t > T.$$

We derived close-form solution to the above ODEs, and obtained explicit formula for the average delivery delay (see the global timeout (2) in Table 1). The average number of copies made at delivery time, $E[C]$, and during a packet's lifetime, $E[G]$, are the same as under the original global timeout scheme.

3.3. Signaling overhead

We have so far studied the number of copies made for a packet and the average storage occupancy incurred by data packets, but ignored the signaling overhead. We now discuss the signaling overhead involved in epidemic style routing, using the global timeout scheme to delete anti-packets.

We assume that when two nodes move into the transmission range of each other, they perform the following steps:

1. exchange identification information, i.e., node ID,
2. exchange header information of data packets,
3. exchange anti-packets information,
4. actual packet exchanges.

The transmission cost of the step 4 has already been studied in Section 2.3. The amount of information exchanged in steps 2 and 3 are different for different forwarding and recovery schemes respectively. For example, under K-hop, the packet header for a packet with hop count $K - 1$ does not need to be sent to other relay nodes. Likewise, while IMMUNE_TX only propagate anti-packets to infected nodes, VACCINE propagate anti-packets to all nodes.

Extending ODE models to consider signaling overhead is straightforward as we illustrate using the basic epidemic routing with IMMUNE recovery and global timer of duration T as an example. Let $I(t)$ and $R(t)$ be the average number of infected and recovered nodes respectively at time t, taking into account the recovery process. We have:

$$I'(t) = \beta I(N - I - R) - \beta I, \quad t \leqslant T,$$
$$I'(t) = -\beta I, \quad t > T,$$
$$R'(t) = \beta I, \quad t \leqslant T.$$

Since all anti-packets are deleted after the timer expires, we have $R(t) = 0$ for $t > T$. Under a packet arrival rate of λ, Q_{anti}, the average per-node buffer occupancy of anti-packets, is given by $Q_{\text{anti}} = \lambda \int_0^T R(t)\,dt$, following an argument similar to that in Section 2.3.

Now let us consider the overhead of exchanging packet headers and anti-packets. Let $H(t)$ and $A(t)$ respectively denote the average number of packet headers and anti-packets that are exchanged among all the nodes up to time t, we have:

$$H'(t) = \beta I(N + 1), \quad t \leqslant T,$$
$$H'(t) = \beta I, \quad t > T,$$
$$A'(t) = \beta I.$$

Intuitively, before time T, the packet header is sent by infected nodes to every node they meet.[5] After the timer expires, the packet header is sent only to the destination node. Anti-packets are transmitted by the destination node to infected nodes when they meet under IMMUNE, before or after T. For any packet, the average total number of times the packet header and anti-packet is exchanged is given by $Q_h = H(\infty)$ and $Q_a = A(\infty)$, respectively. Numerical techniques can be used to evaluate these metrics.

[5] For the purpose of clarity, we ignore some optimizations that can be used to save overhead. For example, when two infected nodes for packet i meet, after one node sends its packet headers, the other node, knowing the previous node has packet i, need not send packet i header to B.

4. Model validation

We have developed a simulator that simulates various routing schemes and recovery schemes under random waypoint and random direction models. The results we present here are for a specific setting considered in [6]: $N+1$ nodes move within a 20×20 terrain according to random direction model [2,7]. Each node chooses an initial direction, speed and travel time, and then travels in that direction with given speed for the chosen travel time. When the travel time expires, the node chooses a new direction, speed and travel time at random, independently of all previous directions, speeds and travel times. If a node hits the boundary of the terrain, it wraps around at the other side of the terrain. The node speed is chosen uniformly in the range 4–10, and the mean travel duration is 1/4. The transmission range of the nodes is chosen to be 0.1. The pair-wise meeting rate for this setting is found to be $\beta = 0.00435$ using the formula in [6].

We simulate $N+1$ unicast flows, with each node being the source of one flow, and the destination of another flow. Each flow generates packets at a Poisson rate of $\lambda = 0.01$. The simulation is run long enough such that at least 500 packets are generated and delivered. We then use the 500 observations to calculate the mean and 95% confidence interval for packet delivery delay and the total number of copies made for a packet. Average buffer occupancy is calculated after removing the initial transient period from the trace. These simulation results are then compared with the ODE model predictions. We report *relative modeling error*, defined as $(V_s - V_m)/V_m$, where V_m is the model predicted value and V_s is the simulation result. We calculate the 95% confidence interval for the relative modeling errors using the 95% confidence interval for V_s. We do not consider signaling overhead as we expect to observe similar prediction performance for these metrics.

We first consider basic epidemic routing. We vary N between 5 and 160, and plot the mean and 95% confidence interval of packet delivery delay obtained from simulation, and the model prediction in Fig. 1a. We find that the model is able to accurately predict the delivery delay, capturing the performance trend as N increases. Fig. 1b compares, for $N = 160$, the CDF of packet delivery delay obtained from simulation with the one predicted by Eq. (4). It shows that ODE model under predicts the packet delivery delay. To investigate modeling errors, we ran another simulation with nodes meeting according to a Poisson process with rate $\beta = 0.00435$ (i.e., we set the meeting rate in the simulation to exactly match the model's meeting rate) and the results of the two simulations are very close (see the curve labeled as "Poisson Simulation" in Fig. 1b). This suggests that the error introduced by the Poisson meeting process approximation is negligible. We conjecture that the prediction errors are mainly due to the small number of initially infected nodes and/or total nodes number. This is confirmed by simulations where we vary the number of initially infected nodes, and found the modeling error becomes smaller when the number of initially infected nodes is large. We also used a moment-closure technique to derive an ODE system involving second moments using the MVN method (details are given in Appendix C). The modified ODE provided a better prediction for average delivery delay and the CDF of delivery delay (Fig. 1).

For epidemic routing with different recovery schemes, Fig. 2 plots $E[G_{ep}(N)]/N$, and the average buffer occupancy $E[Q]$ as predicted by the model and obtained from simulation. We find that the

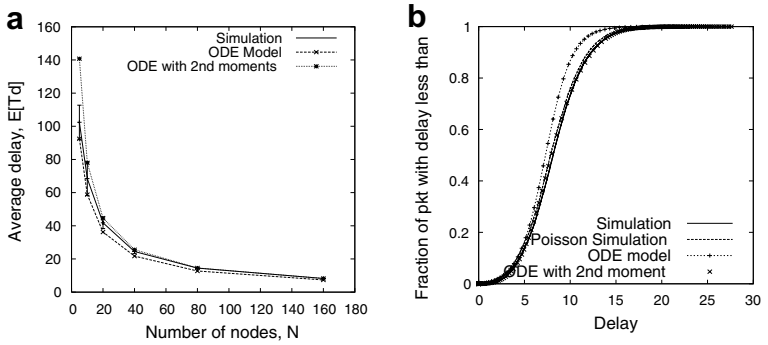

Fig. 1. Delay under epidemic routing. (a) Average delay for different N. (b) CDF of delay with $N = 160$.

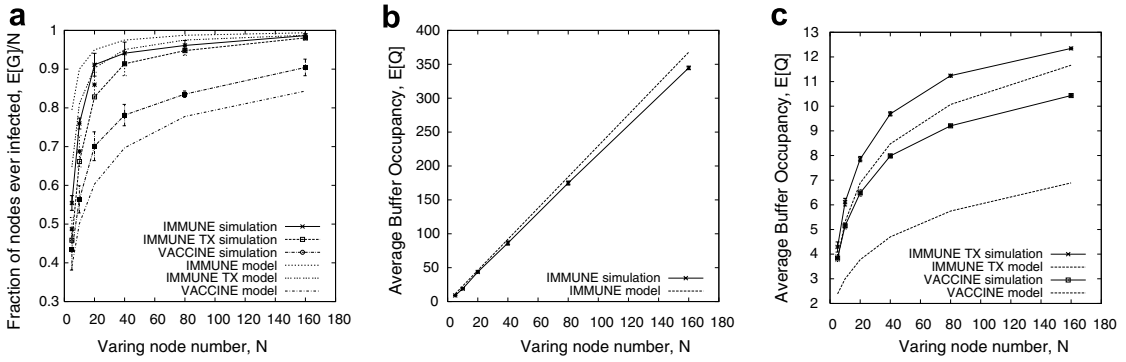

Fig. 2. Copies sent and buffer occupancy under epidemic routing. (a) $E[G_{ep}]/N$. (b) Buffer occupancy under IMMUNE. (c) Buffer occupancy under IMMUNX_TX and VACCINE.

ODE models are more accurate for IMMUNE than for VACCINE. In some sense, any error in the infection process modeling is amplified by the exponentially fast recovery of VACCINE. We observe that IMMUNE_TX only slightly reduces the number of copies sent for each packet, while VACCINE further reduces the number of copies sent. The reduction in buffer requirements is similar for IMMUNE_TX and VACCINE.

Fig. 3 plots the relative modeling error for these three recovery schemes. We observe that as N increases, the error decreases. While ODE models over predict the copies sent and average buffer occupancy for IMMUNE recovery, they under predict buffer occupancy for IMMUNE_TX recovery, and under predict both metrics for VACCINE recovery.

Next, we present validation results for the forwarding schemes introduced in Section 3, focusing on the following three metrics, average delay, $E[T_d]$, average buffer occupancy, $E[Q]$, and average total number of copies transmitted, $E[G]$ under IMMUNE recovery. We expect the prediction errors to be slightly larger for IMMUNE_TX and VACCINE recovery as observed for epidemic routing.

For the 2-hop forwarding, Fig. 4 compares the three metrics under varying number of nodes, N, showing a good match between the modeling results and simulation results. Fig. 6a plots the relative prediction error.

For probabilistic forwarding scheme, Fig. 5 plots the three metrics, comparing the model prediction with the simulation result, Fig. 6b plots the relative prediction error for probabilistic forwarding. We observe a larger prediction error for $p \in [0.01, 0.1]$, and error decreases as p increases and approaches to 1. We conjecture the large prediction error in $p \in [0.01, 0.1]$ is due to the larger variance when p takes a value in this range (see Appendix C). Like for epidemic routing, the ODE models under predicts the delay, whereas over predicts the number of copies sent and the average buffer occupancy.

For limited time forwarding (with no reinfection after timeout) under varying average timeout value, $1/\mu$, Fig. 7 plots the three metrics as predicted by the model and as obtained through simulation, and

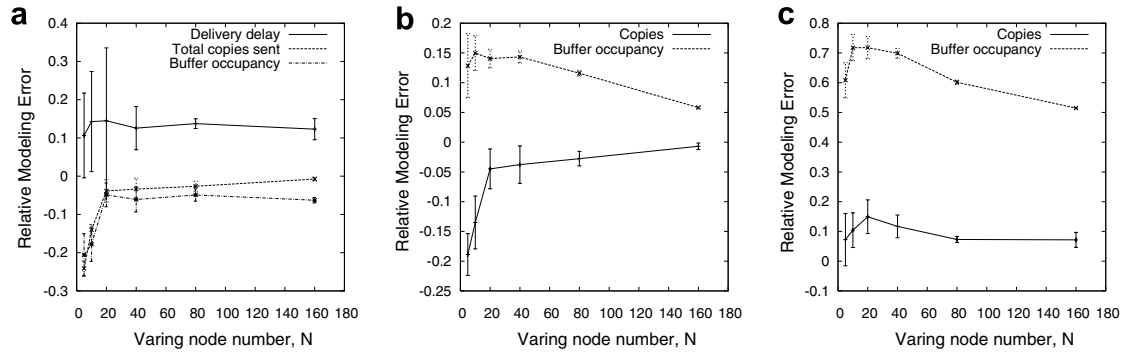

Fig. 3. Relative modeling error for epidemic routing. (a) IMMUNE recovery; (b) IMMUNE_TX recovery and (c) VACCINE recovery.

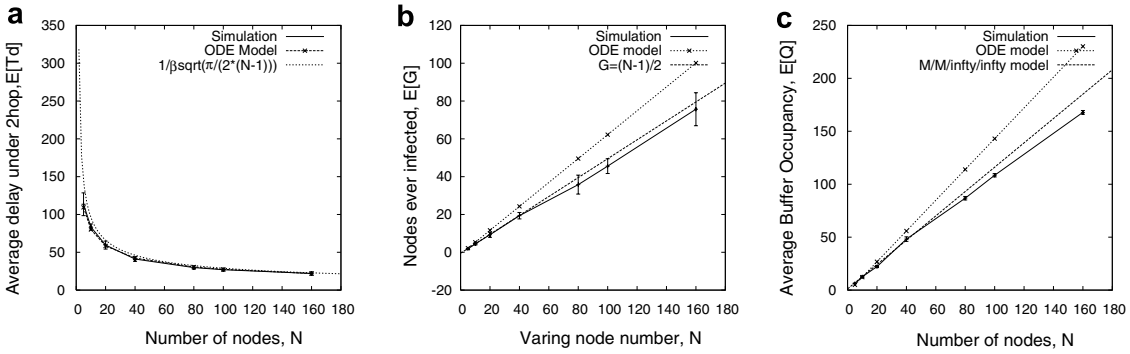

Fig. 4. 2-hop forwarding. (a) Delivery average, (b) copies transmitted and (c) buffer occupancy.

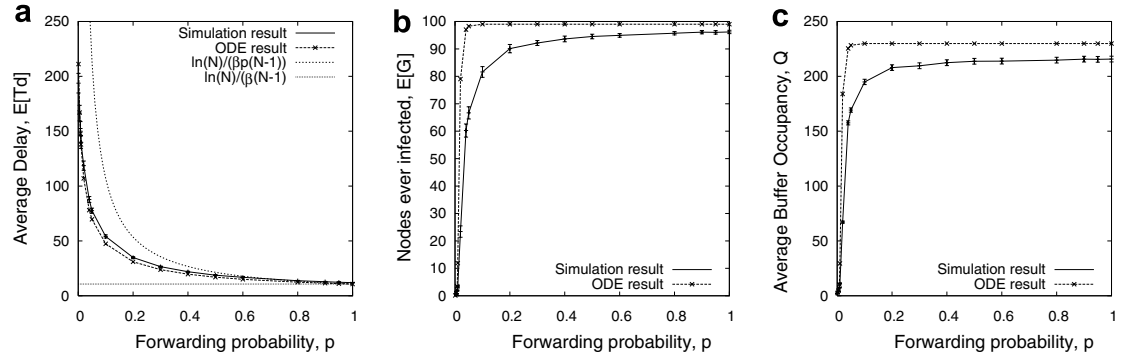

Fig. 5. Probabilistic forwarding. (a) Delivery delay, (b) copies transmitted and (c) buffer occupancy.

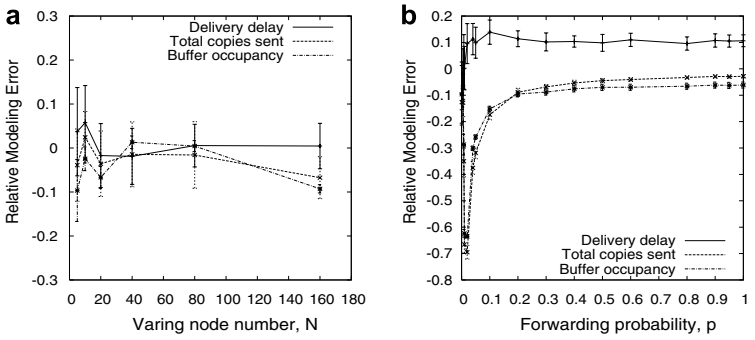

Fig. 6. Relative modeling error for *2*-hop and probabilistic schemes. (a) 2-hop forwarding and (b) probabilistic forwarding.

Fig. 9a plots the relative modeling error. We observe that the relative modeling error decreases as the average timeout value increases. This was expected because the higher the number of infected nodes, the better is the fluid approximation. As in the case of epidemic routing, the model under predicts the delay, and over predict the number of copies sent and the average buffer occupancy.

Finally, for epidemic routing with IMMUNE recovery and global timeout mechanism, Fig. 8 plots the three metrics under different global timeout value, T, Fig. 9b plots the corresponding relative prediction errors. We observe that the ODE models under predict the delay, and over predict the number of copies sent and the average buffer occupancy, as the case for epidemic routing. The

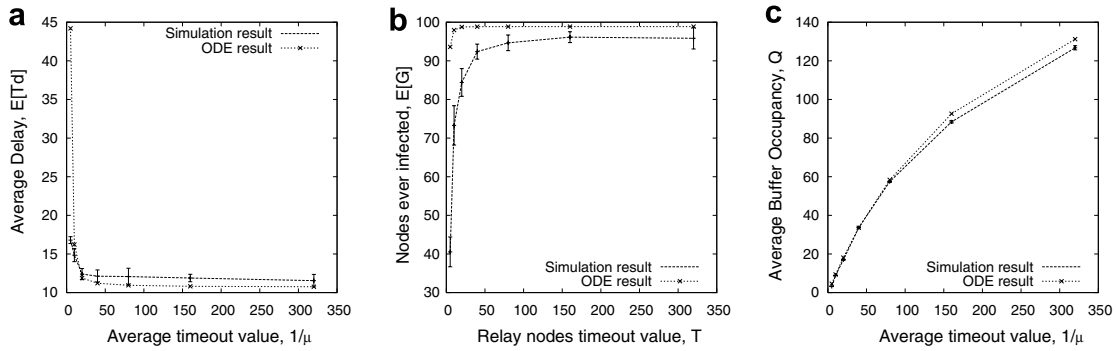

Fig. 7. Limited-time forwarding. (a) Delivery delay, (b) copies transmitted and (c) buffer occupancy.

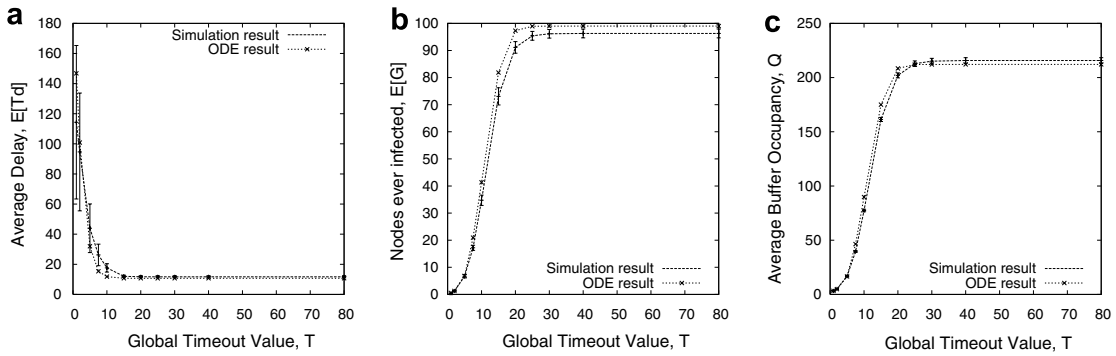

Fig. 8. Global timeout scheme. (a) Delivery delay, (b) copies transmitted and (c) buffer occupancy.

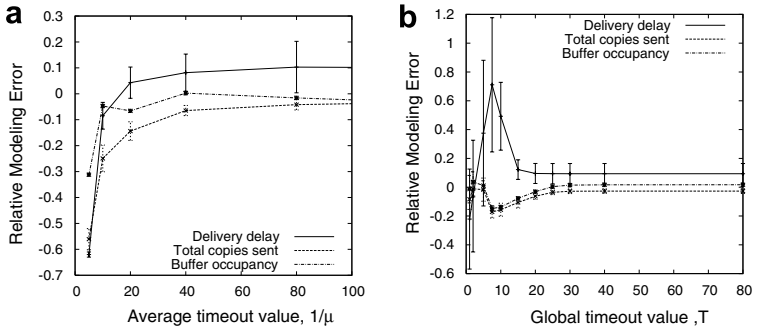

Fig. 9. Relative modeling error for limited-time and global timeout scheme. (a) Limited-time forwarding and (b) global timeout scheme.

relative modeling error decreases as the timeout value T increases, as for limited time forwarding.

5. Performance trade-offs

In this section, we show how the ODE models we derived can be employed to quantitatively explore the tradeoff between delivery delay and resource consumption under different forwarding and recovery schemes, and to determine configuration criteria. It is not our intent to exhaustively explore all the possible dimensions of epidemic routing (forwarding schemes, recovery schemes, methods to manage anti-packets) in order to determine the best candidate to be used in a specific scenario to optimize a specific performance metric.

Previous work [8,25] investigated the buffer-delay tradeoff by varying the number of nodes. However, we believe that the number of nodes is often given, and it is consequently more important to evaluate the

performance tradeoffs achieved by different schemes and/or understand how performance changes as configurable parameter values change. In terms of the tradeoff between delay and the number of copies transmitted, previous work [25,26] only considered the tradeoff achieved by a special scheme that enforces a fixed number of copies.

We discuss the delay versus number of copies transmitted for a packet and delay versus buffer occupancy tradeoff achieved by different forwarding schemes under IMMUNE (Section 5.1) and VACCINE (Section 5.2). We ignore signaling overhead in this discussion, because to consider anti-packets overhead, we need to incorporate ways to delete anti-packets, for example, by introducing global timeout scheme. For each scheme and the particular parameter setting, choosing a different global timer T results in a different tradeoff between delivery delay and resource consumptions; there is not an optimal choice of T unless a optimization goal is given, for example by assigning weights to the different metrics (i.e., delivery delay, copies made for packet and anti-packet, and total storage occupancy). The latter optimization consideration is beyond the scope of this paper. The reader interested into this topics can refer to our work [20].

The results are mainly based on numerical solution of the previous ODEs (for $N = 100$, $\beta = 0.00435$, $\lambda = 0.01$), but we also employ asymptotic results for qualitative considerations.

5.1. Performance trade-off under IMMUNE

Fig. 10a and b respectively plot the delay-versus-number-of-copies-sent and the delay-versus-buffer-occupancy trade-offs achieved by different forwarding schemes when IMMUNE recovery is employed. In the figure, there are four singleton points corresponding to direct source–destination transmission, 2-hop, 3-hop forwarding and epidemic routing. Three curves have been obtained for probabilistic forwarding, limited-time forwarding (without reinfection) and global timeout respectively; for these curves, each point corresponds to a different value of the forward probability p, the mean timeout interval $1/\mu$ or the global timeout T, respectively. All these parameter values are shown in Table 2.

Let us first consider the delay-versus-number-of-copies-sent trade-off. One can reduce the number of copies sent by decreasing p, $1/\mu$ or T, but at the same time the delay will increase. Intuitively, these schemes behave as the original epidemic routing as $p \to 1$, $1/\mu \to \infty$ and $T \to \infty$, whereas $p \to 0$, $1/\mu \to 0$ or $T \to 0$ correspond to a no-relaying scenario in which the packet is only delivered directly from the source to the destination. The only difference is for $1/\mu \to 0$, the number of copies converges to $N/2$ (which is the average number of nodes the source uselessly infects before meeting the destination). Global timeout scheme appears to be the best choice when limiting the number of copies

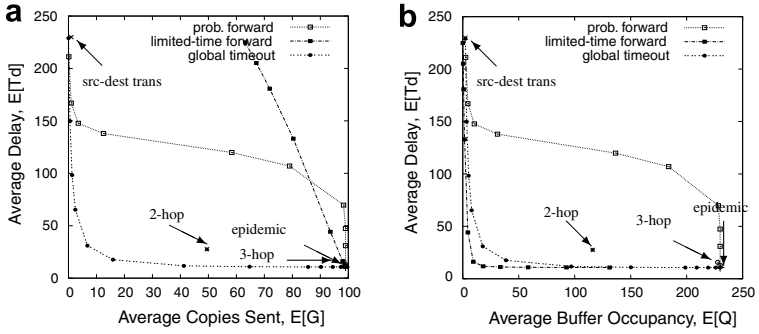

Fig. 10. Comparison with IMMUNE recovery. (a) Delay vs number of copies sent tradeoff and (b) delay vs buffer occupancy tradeoff.

Table 2
Settings considered for probabilistic, limited-time forwarding, and global timeout scheme

Probability (p, %)	0.1	0.5	0.8	1	1.5	2	5	10	20	80			
Timeout ($1/\mu$)	0.1	0.5	1	2	5	10	20	40	80	160	320		
Global timer (T)	0.01	1	2	3	5	7	10	12	15	16	18	20	80

transmitted is the main concern. As a rule of thumb, one can choose $T \approx E[T_d]/2$ (=5 in this specific setting), where $E[T_d]$ is the average delay under epidemic routing. This choice significantly reduces the number of copies sent from nearly 100 to 6.8, with delivery delay increased from around 10 to around 30.

Fig. 10b shows that for probabilistic and K-hop forwarding, the delay-versus-buffer-occupancy tradeoffs are similar to the delay-versus-copies tradeoffs. This is due to the proportionality between the number of copies sent and the buffer occupancy that we have shown in Section 2.3 for epidemic routing under IMMUNE. This relation holds for all schemes where copies are deleted only after the meeting with the destination, hence also for probabilistic and K-hop forwarding under IMMUNE, but not for limited-time or global timeout forwarding. We observe that the limited time forwarding is the best choice when limiting buffer occupancy is of primary concern. With a value of $1/\mu \approx 2E[T_d]$ (=20 in this specific setting), the average buffer occupancy is decreased to about one tenth of that of epidemic routing, with a small increase in the delivery time. The delay-versus-buffer tradeoff achieved by global timeout scheme is very close to that of limited time forwarding.

5.2. Performance improvement by VACCINE

Fig. 11 shows the tradeoff under various forwarding schemes when VACCINE recovery is employed. For the delay-versus-copies tradeoff (Fig. 11a), compared to IMMUNE recovery, VACCINE recovery decreases the average number of copies sent for a packet and the average buffer occupancy for each forwarding scheme. However, for different schemes, different amount of improvements are achieved by VACCINE recovery: in particular, the largest improvement is achieved for probabilistic forwarding, followed by K-hop forwarding, and then limited-time forwarding and the global timeout scheme. The relatively small improvements for limited-time forwarding and the global timeout scheme are due to their intrinsic recovery features: nodes delete packet copies when the timer expires and they cannot be reinfected. The explanation is more complex for the probabilistic and K-hop forwarding schemes. Because of the two counteracting processes – the counter-infection recovery process due to anti-packets spreading and the continuing ongoing packet infection – the total recovery speed depends not only on the recovery scheme but also on the specific infection process. Given the same average delivery delay, when the recovery process starts, the average number of nodes infected and the current infection rate are higher under probabilistic forwarding (its infection rate is exponential, hence in the long term it is faster than K-hop). For this reason, we expect the IMMUNE recovery process to be significantly "longer" for probabilistic forwarding than for K-hop forwarding, leading to larger buffer occupancy and more copies transmitted for a packet (as shown in Fig. 10). Conversely under VACCINE, the recovery process is much shorter; the buffer occupancy is mainly determined by the initial infection process (before the delivery), and the difference in the copies transmitted and the buffer occupancy under probabilistic forwarding and K-hop scheme becomes much smaller, as shown in Fig. 11.

Fig. 11b and c plot the delay-versus-buffer-occupancy tradeoff for various forwarding scheme under VACCINE recovery, where (c) zooms into the small

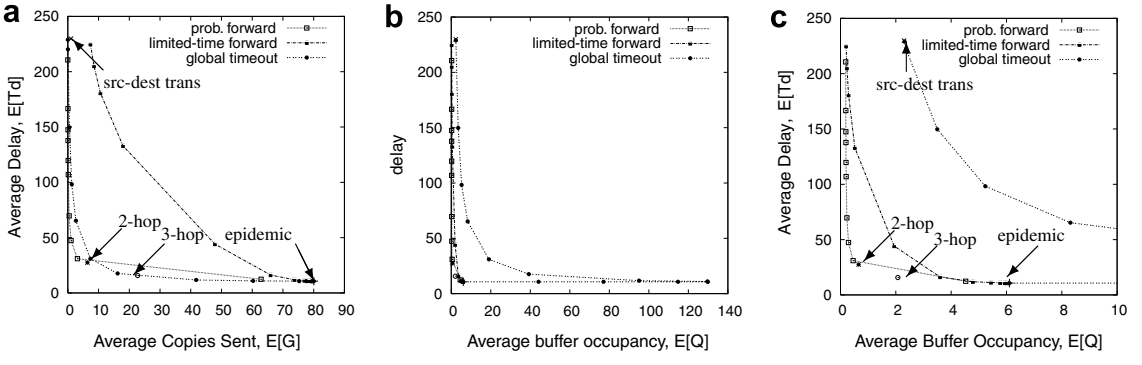

Fig. 11. Comparison with VACCINE recovery. (a) Delay vs number of copies sent tradeoff, (b) delay vs buffer occupancy tradeoff and (c) delay vs buffer occupancy tradeoff.

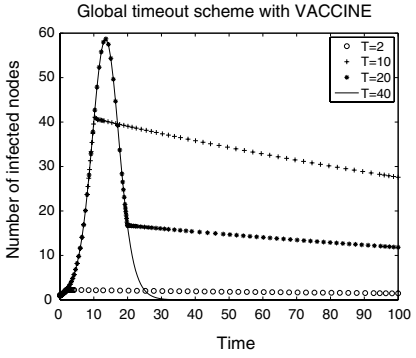

Fig. 12. $I(t)$ for different T under global timeout scheme.

buffer occupancy range. Comparing Fig. 11c with a, we find that the delay-versus-buffer tradeoff is similar to that of delay-versus-copies tradeoff except for global timeout scheme. For global timeout scheme, as Fig. 11b shows, as T increases, the delay decreases monotonically; whereas, the buffer occupancy increases first and then decreases. To see why this is the case, Fig. 12 plots the numerical solutions for $I(t)$, the number of infected nodes at time t, under different settings of T. Basically, under global timeout scheme, the recovery process after time T is IMMUNE recovery, which is much slower than VACCINE recovery, therefore, increasing the timeout value T not only leads to longer epidemic spreading phase, but also results in faster overall recovery process. When T is smaller than a certain threshold (which is around 15 for the specific setting considered here), the first effect outweighs the second one, leading to larger buffer occupancy (as illustrated by $T = 2$ and $T = 10$ curves); when T increases further, the second effect becomes dominant factor, leading to small buffer occupancy under larger T (as illustrated by $T = 20$, and $T = 40$ curves).[6]

6. Epidemic routing under constrained buffer

Thus far, we have assumed that each node has sufficient space to store all packets. In reality, however, mobile nodes often have limited storage due to cost and form factor. Sizing the buffer to limit end-to-end packet losses due to buffer overflow in store-carry-forward networks is hard. For example, [8] studied buffer occupancy variability for the purpose of buffer sizing, but their model requires an empirical distribution obtained from simulation. In this section, we examine the performance of epidemic routing under the constraint that each node can store at most B packets. We consider three buffer management strategies: (i) *droptail* where newly arriving packets are dropped if the buffer is full (previously studied in [28] through simulation), (ii) *drophead* where the oldest packet in the buffer is dropped to accept newly arriving packets, and (iii) *drophead_sp*, source-prioritized drophead, which gives priority to packets arriving directly from the node itself. We describe the model for drophead_sp here; a full analysis can be found in [29].

Under *drophead_sp*, when a packet arrives to a full buffer, the node discards the oldest relay packet (i.e., a packet it has received from other node) to make space for the new packet. If all buffered packets are source packets, and the arriving packet is a source packet, the oldest source packet is deleted. Relay packets arriving to a buffer filled with source packets are not accepted. Therefore, given P_f, the probability that a node's buffer is filled with source packets, the effective infection rate is then $\beta(1 - P_f)$. P_f can be derived by modeling the number of node-buffered source packets as a Markov chain (details can be found in [29]).

As before, we focus on the spreading of a single packet. Let $\overline{G}_{dhs}$ be the average number of copies made for each packet under the *drophead_sp* policy. Let $I_j^s(t)$, for $j = 1, 2, \ldots, B$, be the probability that the packet is the jth newest source packet in the source node's buffer, $I_j(t)$, for $j = 1, 2, \ldots, B$, be the average number of infected *relay* nodes where the copy is the jth newest packet in the buffer, $S(t)$ be the average number of susceptible nodes, and $D(t)$ be the average number of nodes that have dropped the packet. Using arguments similar to those in Section 2.1, we can then use the following ODEs to model packet spreading in the case of buffer limits. At infected relay nodes (Eqs. (10) and (11)), the packet becomes older whenever another packet arrives, with rate $(\overline{G}_{dhs} + 1)\lambda$ (this is the total packet arrival rate to a node by an argument similar to that in Section 2.3). At the source node (Eqs. (11) and (12)), the copy of this packet becomes older at rate λ, the rate at which new source packets arrive.

$$S'(t) = -\beta(1 - P_f)S\sum_{i=1}^{B}(I_i^s + I_i), \tag{8}$$

$$I_1'(t) = \beta(1 - P_f)S\sum_{i=1}^{B}(I_i^s + I_i) - (\overline{G}_{dhs} + 1)\lambda I_1, \tag{9}$$

$$I_j'(t) = (\overline{G}_{dhs} + 1)\lambda(I_{j-1} - I_j), \quad 2 \leqslant j \leqslant B, \tag{10}$$

[6] Recall that $Q = \lambda\int_0^\infty I(t)\,dt$, as derived in Section 2.3.

$$I_1^{s\prime}(t) = -\lambda I_1^s, \tag{11}$$

$$I_j^{s\prime}(t) = \lambda(I_{j-1}^s - I_j^s), \quad 2 \leqslant j \leqslant B, \tag{12}$$

$$D'(t) = (\overline{G}_{dhs} + 1)\lambda I_B + \lambda I_B^s, , \tag{13}$$

$$P'(t) = \beta \sum_{i=1}^{B} (I_i^s + I_i)(1 - P). \tag{14}$$

The initial conditions are given by: $S(0) = N - 1$, $I_1^s(0) = 1$, $I_j^s(0) = 0$, for $j = 2, \ldots, B$, $I_k(0) = 0$, for $k = 1, \ldots, B$, $D(0) = 0$, $P(0) = 0$. We find $\overline{G}_{dhs}$ by solving the following fixed-point problem using a binary search algorithm: given $\overline{G}_{dhs}$, we numerically solve the corresponding extended ODE model (including the recovery process) and calculate the accumulated amount of flow from state S to I_1, i.e., $\overline{G}_{dhs}$. Given the new value of $\overline{G}_{dhs}$, we then again solve the ODEs.

We have simulated these schemes, using the same setting as before ($N = 100$, $\lambda = 0.01$, $\beta = 0.00435$), with different buffer sizes $B = 5$, 10, 20, and compared our ODE results with simulation. Table 3 tabulates the packet loss probability, i.e., the probability that all the copies of a packet are dropped before the destination receives one. Fig. 13 plot the delay distributions predicted for $B = 5$, 10, in the range $[0, 200]$ and $[0, 50]$ respectively so that the difference between schemes can be seen.

Table 3
Loss probability under constrained buffer

Buffer size	Simulation/ model	Droptail	Drophead	Drophead_sp
5	Simulation	0.9696	0.2234	0.0536
	Model	0.8544	0.0928	0.0079
10	Simulation	0.9471	0.0315	0.0
	Model	0.7891	0.0088	0.0
20	Simulation	0.899	0.0016	0.0
	Model	0.7011	0.0	0.0

We observe that the models provide reasonable accurate loss probability predictions, and reflect the relative performance of the three dropping schemes. The shape of the distribution probability function for delivery delay is also well-captured by the model [29]. We observe that naive *droptail* performs poorly. *Drophead* provides fast infection, as relay packets are always accepted; however, significant packet losses are incurred for $B \leqslant 10$. With *drophead_sp*, although the infection spreads slower than under *drophead*, more packets are delivered. If the packet rate is so high that the buffer can only hold its own source packets, *drophead_sp* degenerates to direct source–destination transmission. Note that with infinite buffers, the average buffer occupancy for this setting is over 200 (Fig. 2b). Our results here suggest that similar performance can be achieved by *drophead* and *drophead_sp* with a much smaller buffer size, equal to only 20 packets.

7. Related work

In the mathematical epidemiology field, there exists a vast volume of literature about mathematical models on the spreading of infectious diseases, including both stochastic and deterministic models [1,3]. These mathematical techniques have been applied to various computer networking problems that exhibit a strong analogy to epidemic spreading of disease. For example, [14,27,19,31] modeled the spread of computer virus and worms in computer networks by adapting epidemiological models. Also, a number of network applications (protocols) have adopted epidemic-style spreading communication for data dissemination and resource discovery, and therefore epidemic models are a natural way to

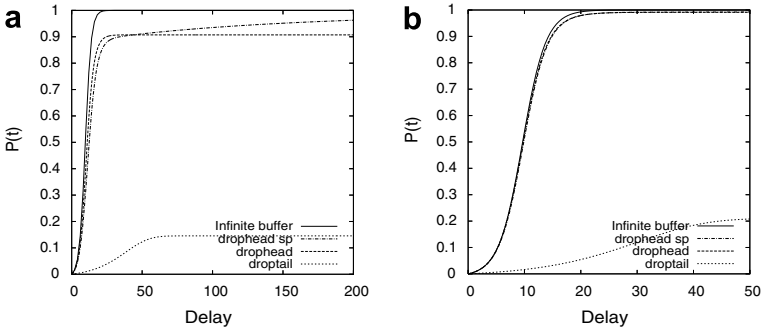

Fig. 13. $P(t)$ under $B = 5$, 10. (a) $B = 5$ and (b) $B = 10$.

study their performance. These include epidemic algorithms [4] for maintaining consistency of replicated database, gossip (rumor-based) protocol [11], broadcast communication [15] and peer-to-peer data sharing [21] in mobile ad hoc network, and more recently, epidemic routing [28,8,30] in Delay Tolerant Networks. Epidemic routing differs from the other above mentioned broadcast based protocols in that it supports *unicast application*, using epidemic style flooding to decrease the delivery delay.

Based on earlier results in [5], we have used a homogeneous mixing model employing a single parameter (derived from mobility parameters) to capture the contact rate between mobile nodes. Refs. [15,19] considered similar network settings as our work, i.e., mobile ad hoc network. Ref. [15] made a similar homogeneous mixing assumption, and obtained the contact rate through finding best-fitting formula from results of many simulation runs. Ref. [19] considered a network with higher node density and slower nodal mobility than our paper, and extended Kephart–Whilte model [13] to model the virus spreading, characterizing the fraction of nodes with varying connectivities under given mobility models.

Another important difference between our work and the above mentioned work lies in the fact that we are interested in performance metrics that are unique to the unicast application in DTN. We have seen that there exists a tradeoffs between the delivery delay and resource consumptions in terms of the number of transmissions made for a packet and buffer occupancy. Using ODE models, we have studied the performance of various epidemic style routing, and explored the tradeoffs they achieve.

The work most closely related to ours is [8], where an ODE model is applied to study delay under epidemic routing, and Markov chain model is used to study the storage requirement under different recovery schemes. While both our work and [8] study the delay, storage requirement, and transmission numbers of epidemic routing, our work goes beyond this single scheme to study schemes such as 2-hop forwarding, probabilistic forwarding, limited-time forwarding, global timeout scheme, and epidemic routing in the buffer-constrained scenario. In addition, our analysis leads to new closed-form expressions and asymptotic results, when the number of nodes increases, for a number of schemes. Furthermore, we study epidemic routing under buffer-constrained scenario using ODE models coupled with Markov models to compare different buffer management strategies. We also note that the approach in [8] is a hybrid approach and requires obtaining some model parameters, such as the number of nodes infected at the time of delivery, from simulations. We derive all metrics as part of the model itself. Last, because our focus is on the use of ODE models, we provide insight into when they do or do not work and why, and show how moment closure techniques could be employed to improve the model predictions.

Another closely related work is [5]. Based on the result of Poisson meeting process, the authors modeled 2-hop forwarding and epidemic routing using Markov chain models, and derived the average delay and the number of copies generated at the time of delivery for these two schemes. Using ODE models, we have more easily derived similar results. Ref. [9] later extended this work to consider a variant of 2-hop scheme with exponential timers at each node with and a limit on the maximum number of copies. Through Markovian analysis, the author derived close-form formulas and numerical solutions for delivery delay, number of copies transmitted for these two schemes respectively. Given the difficulty in deriving asymptotic formulas from Markovian analysis, ODE models were employed to derive asymptotic formulas for moments of delivery delay, and copies made at delivery time for these two schemes.

8. Summary

In this paper, we proposed and investigated a unified framework based on ODEs to study the performance of various forwarding and recovery schemes. We derived ODE models as limiting processes of Markovian models under a natural scaling as the number of nodes increases, and employed the ODE models to obtain a rich set of close-form formulas regarding the packet-delivery delay, number of copies sent, and buffer occupancy under various schemes. We validated the models through simulations, and observed a good match between the model prediction and simulation results. We used the ODE models to explore performance tradeoffs achieved by various schemes, and obtain insights into the different schemes. We further considered the buffer-constrained case, and showed that with appropriate buffer management schemes, a much smaller buffer can be used with negligible effect on delivery performance.

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Appendix A. Derivation of ODEs that consider recovery processes

In this section, we derive ODE models to include the recovery process from Markov Chain models. In order to derive the limiting equation the number of destinations, n_D, need to scale with the number of nodes N. We first consider IMMUNE scheme. Let $n_R(t)$ denote the number of recovered nodes at time t, then the state can be denoted as $(n_I(t), n_R(t))$. We have the following transition rates:

$$\begin{aligned} &r_N((n_I(t), n_R(t)), (n_I(t)+1, n_R(t))) \\ &\quad = \beta n_I(t)(N - n_I(t) - n_R(t)), \\ &r_N((n_I(t), n_R(t)), (n_I(t)-1, n_R(t)+1)) \\ &\quad = \beta n_I(t) n_D. \end{aligned}$$

The transition rates can be written in a "density dependent" form, given that the number of destinations n_D scales in a manner similar to the scaling of the number of initially infected nodes, i.e., $\lim_{N\to\infty} n_D/N = d$. Then by Theorem 3.1 in [16], we get that, as N increases, the fraction of infected nodes (n_I/N) and recovered nodes (n_R/N) converge asymptotically to the solution of the following equations:

$$\begin{aligned} &i'(t) = \lambda i(t)(1 - i(t) - r(t)) - \lambda i(t) d \quad \text{for } t \geqslant 0, \\ &r'(t) = \lambda i(t) d \quad \text{for } t \geqslant 0, \end{aligned}$$

where $d = n_D/N$, and the initial conditions are $i(0) = \lim_{N\to\infty} n_I(0)/N$, $r(0) = 0$.

The number of infected and recovered nodes then converges to $I(t) = Ni(t)$, $R(t) = Nr(t)$ in the sense of Footnote 1. The following equation can be derived for $I(t)$, $R(t)$ from the previous ODEs:

$$I'(t) = \beta I(N - I - R) - \beta I n_D, \tag{A.1}$$

$$R'(t) = \beta I n_D \tag{A.2}$$

with initial condition $I(0) = Ni(0)$, $R(0) = 0$. We consider $I(0) = 1$, $R(0) = 0$, $n_D = 1$.

ODE models for IMMUNE_TX and VACCINE scheme can be similarly derived. For IMMUNE_TX the transition rates are (omitting the dependence from time, t):

$$\begin{aligned} &r_N((n_I, n_R), (n_I+1, n_R)) = \beta n_I(N - n_I - n_R), \\ &r_N((n_I, n_R), (n_I-1, n_R+1)) = \beta n_I(n_R + n_D). \end{aligned}$$

The limiting equations are

$$\begin{aligned} &i'(t) = \lambda i(t)(1 - i(t) - r(t)) - \lambda i(t)(r(t) + d) \\ &\quad \text{for } t \geqslant 0, \\ &r'(t) = \lambda i(t)(r(t) + d) \quad \text{for } t \geqslant 0. \end{aligned}$$

The following equations can be immediately derived:

$$I'(t) = \beta I(N - I - R) - \beta I(1 + R), \tag{A.3}$$

$$R'(t) = \beta I(1 + R). \tag{A.4}$$

For VACCINE we need to specify how many destination nodes have received the packet, let n_{DR} denote this number.[7] We assume that all the destinations have to receive the packets from an infected node.[8] The transition rates are: $r_N((n_I, n_R, n_{DR}), (n_I+1, n_R, n_{DR})) = \beta n_I(N - n_I - n_R)$, $r_N((n_I, n_R, n_{DR}), \quad (n_I-1, n_R+1, n_{DR})) = \beta n_I(n_R + n_{DR})$ and $r_N((n_I, n_R, n_{DR}), (n_I-1, n_R+1, n_{DR}+1)) = \beta n_I(n_D - n_{DR})$ and $r_N((n_I, n_R, n_{DR}), (n_I, n_R+1, n_{DR})) = \beta(N - n_I - n_R)(n_R + n_{DR})$. The limiting equations are as follows, where $d_r(t) = \lim_{N\to\infty}(n_{DR}/N)$:

$$\begin{aligned} &i'(t) = \lambda i(t)(1 - i(t) - r(t)) - \lambda i(t)(r(t) + d) \\ &\qquad \text{for } t \geqslant 0, \\ &r'(t) = \lambda i(t)(r(t) + d) + \lambda(1 - i(t) - r(t))(r(t) + d_r(t)) \\ &\qquad \text{for } t \geqslant 0, \\ &d_r'(t) = \lambda i(t)(d - d_r(t)) \quad \text{for } t \geqslant 0. \end{aligned}$$

[7] There is no such a need for the previous schemes because only a destination can recover an infected node. Hence even if the destination has not received the packet, the destination receives it when it meets the infected node.

[8] Different assumptions can be made, for example a destination could receive the packet from another destination, or a destination could receive the antipacket from a recovered node and propagate it without having received the packet. The latter case is meaningful when we deal with an anycast communication (the packet has to reach at least one of the destinations) or if we can rely on the fact all the destinations will receive a copy of the packet from the destination that started the recovery process. These different assumptions lead to minor differences in the final equations.

If we consider the average populations ($Ni(t), Nr(t)$ and $Nd_r(t)$), and assume $n_D = 1$, we observe that $Nd_r(t)$ satisfies the same ODE as $P(t)$, and derive the following equations:

$$I'(t) = \beta I(t)(N - I(t) - R(t)) - \beta I(t)(R(t) + 1), \tag{A.5}$$

$$R'(t) = \beta I(t)(1 + R(t)) + \beta(N - I(t) - R(t))(R(t) + P(t)), \tag{A.6}$$

$$P'(t) = \beta I(t)(1 - P(t)). \tag{A.7}$$

Let $C(t)$ be the number of nodes that are ever infected by the packet, then we have

$$C'(t) = \beta I(t)(N - I(t) - R(t)). \tag{A.8}$$

These ODE models allow us to evaluate the number of times a packet is copied during its lifetime (excluding the copy to the destination), $G = C(\infty)$, and the average buffer occupancy, $Q = \lambda \int_0^\infty I(t)\,\mathrm{d}t$.

Appendix B. Derivation of the total number of copies

For IMMUNE scheme, Eqs. (A.1) and (A.2) model the infection and recovery process. Note that as $R(t)$ is a strictly increasing function of t, $I(R)$ is well defined. Dividing Eq. (A.1) over (A.2) yields (we assume $n_D = 1$):

$$\frac{\mathrm{d}I}{\mathrm{d}R} = N - I - R - 1.$$

The solution to this ODE with initial condition $I(0) = 1$ is

$$I(R) = (-N + 1)\mathrm{e}^{-R} - R + N.$$

As $\lim_{t\to\infty} I(t) = 0$, we can solve $I(R) = 0$ for R to find $\lim_{t\to\infty} R(t)$. For N large enough ($N > 10$), the solution gives $\lim_{t\to\infty} R(t) \approx N$. Since $I(t) + R(t) - (I(0) + R(0)) = I(t) + R(t) - 1$ is the number of times a packet is copied in the system by time t, we have $E[G_{\mathrm{ep}}(N)] = \lim_{t\to\infty} I(t) + R(t) - 1 \approx N - 1$.

Similarly, for IMMUNE_TX scheme, from Eqs. (A.3) and (A.4), we can solve $I(R)$ and get:

$$I(R) = \frac{-R^2 + (N-1)R + 1}{R + 1}.$$

As $\lim_{t\to\infty} I(t) = 0$, we find $\lim_{t\to\infty} R(t)$ by solving $I(R) = 0$ for R. $I(R) = 0$ has two roots $(N - 1 \pm \sqrt{N^2 - 2N + 5})/2$. Discarding the negative root, we have $\lim_{t\to\infty} R(t) = (N - 1 + \sqrt{N^2 - 2N + 5})/2$. Therefore, for IMMUNE_TX scheme, we found

$$E[G_{\mathrm{ep}}(N)] = \lim_{t\to\infty}(I(t) + R(t) - 1) = \frac{N - 3 + \sqrt{N^2 - 2N + 5}}{2}.$$

Appendix C. Derivation of ODEs from Markov chain through moment closure techniques

In this section, we show how the ODE model can be derived from Markov Chain model by ignoring variability and how variability can be taken into account using differential equations involving higher moments.

We consider the generic epidemic routing under IMMUNE recovery with a pair-wise infection rate of γ, and per-node recovery rate of β. Under the basic epidemic routing, we have $\gamma = \beta$; for probabilistic forwarding, we have $\gamma = p\beta$. A bivariate Markov chain as illustrated in Fig. C.1 can be used to model the infection and IMMUNE recovery process, with state $(S(t), I(t))$ denotes a state where there are $S(t)$ susceptible nodes, and $I(t)$ infected nodes at time t, given that $S(0) = N - 1$, $I(0) = 1$.

Define the state probabilities: $P_{s,i}(t) = \Pr\{S(t) = s, I(t) = i | S(0) = N - 1, I(0) = 1\}$. The Kolmogorov forward equation for the process is

$$\frac{\mathrm{d}P_{s,i}(t)}{\mathrm{d}t} = -P_{s,i}(t)(\beta i + \gamma s i) + P_{s,i+1}(t)\beta(i+1) \times P_{s+1,i-1}(t)\gamma(s+1)(i-1).$$

Let $M(\theta_1, \theta_2, t) := E[\mathrm{e}^{\theta_1 s + \theta_2 i}]$ be the *moment generating function*. Multiplying the above equation with $\mathrm{e}^{\theta_1 s + \theta_2 i}$, and summing over all possible s, i, we get:

$$\frac{\partial M}{\partial t} = \beta(\mathrm{e}^{-\theta_2} - 1)\frac{\partial M}{\partial \theta_2} + \gamma(\mathrm{e}^{\theta_2 - \theta_1} - 1)\frac{\partial^2 M}{\partial \theta_1 \partial \theta_2}. \tag{C.1}$$

We define the *cumulant generating function*, $K(\theta_1, \theta_2, t) := \log M(\theta_1, \theta_2, t)$, and observe that the following equations hold:

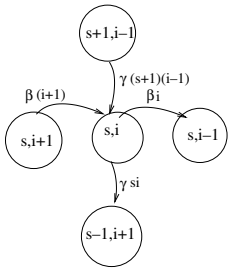

Fig. C.1. Markov chain for epidemic routing.

$$\frac{\partial K}{\partial t} = \frac{1}{M}\frac{\partial M}{\partial t},$$
$$\frac{\partial K}{\partial \theta_1} = \frac{1}{M}\frac{\partial M}{\partial \theta_1},$$
$$\frac{\partial^2 K}{\partial \theta_1 \partial \theta_2} = -\frac{\partial K}{\partial \theta_1}\frac{\partial K}{\partial \theta_2} + \frac{1}{M}\frac{\partial^2 M}{\partial \theta_1 \partial \theta_2}.$$

Substitute these equations into Eq. (C.1), we get:

$$\frac{\partial K}{\partial t} = \beta(e_2^{\theta} - 1)\frac{\partial K}{\partial \theta_2} + \gamma(e^{\theta_2-\theta_1} - 1) \times \left(\frac{\partial^2 K}{\partial \theta_1 \partial \theta_2} + \frac{\partial K}{\partial \theta_1}\frac{\partial K}{\partial \theta_2}\right). \quad (C.2)$$

By taking partial derivatives of θ_1 and θ_2 respectively on Eq. (C.2) and setting $\theta_1 = \theta_2 = 0$, we can get the following ODE system:

$$\frac{d\overline{S}}{dt} = -\gamma(\overline{IS} + C_{IS}),$$
$$\frac{d\overline{I}}{dt} = -\beta\overline{I} + \gamma(\overline{IS} + C_{IS}),$$

where $\overline{S}(t) = E[S(t)], \overline{I}(t) = E[I(t)], C_{IS}(t) = \mathrm{Cov}(S(t), I(t))$.

If we ignore covariance of $I(t)$ and $S(t)$, and set $C_{IS} = 0$, we get:

$$\frac{d\overline{S}}{dt} = -\gamma\overline{IS},$$
$$\frac{d\overline{I}}{dt} = -\beta\overline{I} + \gamma\overline{IS}.$$

This is exactly the ODEs we have derived as limiting process of Markov Chain model.

If we continue this process, we could derive ODEs for second-order moments by taking second order partial derivatives of θ_1 and θ_2 respectively on Eq. (C.2) and setting $\theta_1 = \theta_2 = 0$:

$$\frac{dV_S}{dt} = \gamma(\overline{IS} + C_{IS}) - 2\gamma(T_{SSI} + V_S\overline{I} + \overline{S}C_{IS}),$$
$$\frac{dV_I}{dt} = \beta\overline{I} - 2\beta V_I + \gamma(C_{IS} + \overline{IS}) + 2\gamma(T_{SII} + C_{IS}\overline{I} + \overline{S}V_I),$$
$$\frac{dC_{IS}}{dt} = -\beta C_{IS} - \gamma(C_{IS} + \overline{IS}) - \gamma T_{SII} - \gamma C_{IS}\overline{I} - \gamma\overline{S}V_I + \gamma T_{SSI} + \gamma V_S\overline{I} + \gamma\overline{S}C_{IS},$$

where $V_s(t) = \mathrm{Var}(S(t))$, $V_I(t) = \mathrm{Var}(I(t))$, and T_{SII}, T_{SSI} are the third central moments: $T_{SII} = E[(S - ES)(I - EI)^2]$, $T_{SSI} = E[(S - ES)^2(I - EI)]$.

One could keep on this procedure to derive ODEs for the third and higher moments, but eventually a moment closure technique is needed to truncate the equations at certain order. We experiment with three different methods [12,22,18].

- MVN (multi-variate normal) method: setting third central moments to zero. This is equivalent to assuming a multi-variate normal distribution of the state variables $(S(t), I(t))$.
- Lognormal method: if we assume a lognormal distribution for the state variables, then the third moments can be expressed in terms of the lower moments.
- Third-order moment: truncate the equations by setting fourth-order moments to zero.

In order to compare the performance of these different methods, we simulate probabilistic forwarding, varying p in the range between 0.001 and 1.0, with $N = 100$, and compare the model predictions with the simulation results.

For the basic epidemic routing, i.e., $p = 1.0$, $\gamma = \beta$ case, Fig. C.2 plots the average infected node number, the covariance of infected node number of susceptible node, and the CDF of delay, comparing

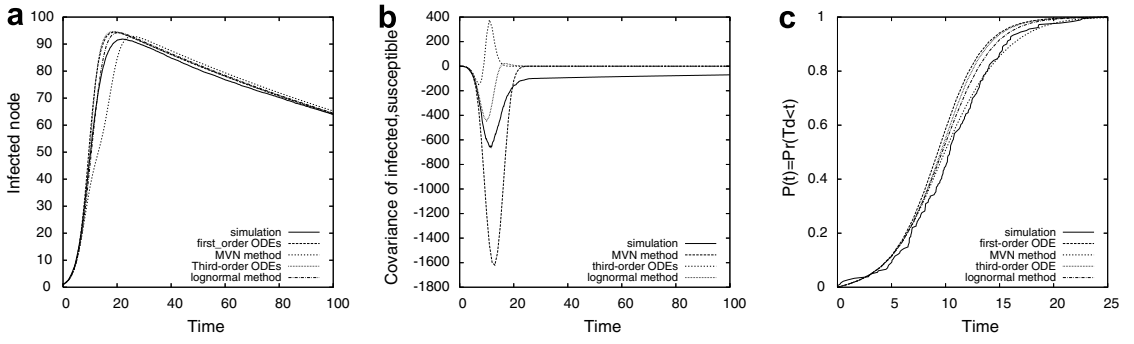

Fig. C.2. Comparison of different moment equations for the case $p = 1.0$. (a) $I(t)$, (b) $\mathrm{COV}(I(t), S(t))$ and (c) $P(t)$.

simulation results with the prediction of different moment equations. We observe that third-order ODEs gives similar result as first-order ODEs, with slight improved match with simulation results. Like first and third order ODEs, lognormal equations under estimates the covariance, and therefore over predicts the infection spreading process, and under predicts the delivery delay. On the other hand, MVN method over estimates the covariance, and under estimates the spread of the infection (as Fig. C.2a shows). For this case, MVN method performs best in prediction of delivery delay as shown also in the Fig. 1 in Section 2.

However, MVN method has a drawback. For P in the range $[0.01, 0.3]$, the MVN ODEs have no stable equilibrium, i.e., the solution diverges. Ref. [18] observed this drawback of MVN method (under a different model), and attributed it to the large variability under the scenario considered.

Appendix D. Delay asymptotic results

Here we are going to derive the different bounds and asymptotic values we presented in the paper. For each of the following forwarding schemes, closed-form expressions can be derived for the number of infected nodes $I(t)$ and the cumulative distribution of delay $P(t) = \Pr(T_d < t) = 1 - Q(t)$. The expected delay can be evaluated as $E[T_d] = \int_0^\infty Q(t)\,\mathrm{d}t$, so we are going to show how this integral can be approximated for the different schemes.

- 2-hop forwarding (Section 3.1.1)The expected delay is equal to

$$E[T_d] = \frac{1}{\beta}\int_0^\infty \mathrm{e}^{-t}\mathrm{e}^{(N-1)(1-t-\mathrm{e}^{-t})}\,\mathrm{d}t,$$

$\mathrm{e}^{(N-1)(1-t-\mathrm{e}^{-t})}$ has a single maximum for $t = 0$, hence according to the saddle point approximation when $N \to \infty$ we can consider:

$$\mathrm{e}^{-t}\mathrm{e}^{(N-1)(1-t-\mathrm{e}^{-t})} \approx \mathrm{e}^{-0}\mathrm{e}^{-(N-1)t^2/2},$$

hence

$$E[T_d] \approx \frac{1}{\beta}\int_0^\infty \mathrm{e}^{-(N-1)t^2/2}\,\mathrm{d}t = \frac{1}{\beta}\sqrt{\frac{\pi}{2}}\frac{1}{\sqrt{N-1}}.$$

- Probabilistic routing (Section 3.1.3). In this case

$$Q(t) = \left(\frac{N}{\mathrm{e}^{N\beta p t} + N - 1}\right)^{\frac{1}{p}}.$$

This expression can be easily bounded:

$$\frac{N}{\mathrm{e}^{N\beta t} + N - 1} \leqslant Q(t) \leqslant \frac{N}{\mathrm{e}^{N\beta p t} + N - 1}.$$

Note that these bounds correspond to the comparison of the probabilistic forwarding with epidemic routing with inter-meeting rates of β and βp respectively: probabilistic forwarding is slower than the first one, but faster than the second one.If we integrate the previous inequality, we get:

$$\frac{\ln(N)}{\beta(N-1)} \leqslant E[T_d] \leqslant \frac{\ln(N)}{\beta p(N-1)}.$$

- Limited-time scheme with reinfection (Section 3.1.3). In this case:

$$Q(t) = \frac{(a_2 - a_1)\mathrm{e}^{-a_1\beta t}}{(a_2 - 1) + (1 - a1)\mathrm{e}^{(a2-a1)\beta t}},$$

where a_2 and a_1 are respectively the positive and the negative solution of the equation $\beta I(N - I) - \mu(I - 1) = 0$ (to be solved for I), obtained by imposing $\frac{\mathrm{d}I}{\mathrm{d}t} = 0$.

We consider three different asymptotic values: for $N \to \infty$, for $\mu \to \infty$ and for $N = \frac{\mu}{\beta} \to \infty$.

As regards the first bound, we proceeded in the following way: we considered a function $Q_{a,N}(t) > 0$ which approximates $Q_N(t)$ (we have stressed the dependence from N), and for which we can closely evaluate $\int_0^\infty Q_{a,N}(t)\,\mathrm{d}t$. This is an asymptotic value for the expected delay if:

$$\lim_{N\to\infty} \frac{\int_0^\infty Q_N(t)\,\mathrm{d}t - \int_0^\infty Q_{a,N}(t)\,\mathrm{d}t}{\int_0^\infty Q_{a,N}(t)\,\mathrm{d}t} \to 0.$$

In order to prove it, we proved that $\frac{Q_N(t) - Q_{a,N}(t)}{Q_{a,N}(t)}$ converges uniformly to zero in $\mathbf{R}^+$ as N diverges:

$$\frac{Q_N(t) - Q_{a,N}(t)}{Q_{a,N}(t)} \underset{N,\infty}{\overset{u}{\to}} 0.$$

In fact in this case $\forall \epsilon > 0$, $\exists n_\epsilon \in \mathbf{N}$ such that $\forall t \in \mathbf{R}^+$ and $\forall N > n_\epsilon$

$$\frac{|Q_N(t) - Q_{a,N}(t)|}{|Q_{a,N}(t)|} < \epsilon,$$

hence

$$\frac{\left|\int_0^\infty Q_N(t) - Q_{a,N}(t)\,\mathrm{d}t\right|}{\left|\int_0^\infty Q_{a,N}(t)\right|} \leqslant \epsilon.$$

The asymptotical behavior of a_2 and a_1 as $N \to \infty$ ($\lim_{N\to\infty} a_2 = +\infty$, $\lim_{N\to\infty} a_1 = 0$) suggests to consider:

$$Q_{a,N}(t) = \frac{a_2 - a_1}{(a_2 - 1) + (1 - a_1)e^{a_2\beta t}},$$

which can be easily integrated.

$$\left|\frac{Q_N(t) - Q_{a,N}(t)}{Q_{a,N}(t)}\right| = \frac{(1 - e^{a_1\beta t})}{e^{a_1\beta t} + \frac{(1-a_1)}{a_2-1}e^{a_2\beta t}} \leqslant \frac{(1 - e^{a_1\beta t})}{\frac{(1-a_1)}{a_2-1}e^{a_2\beta t}}.$$

We can easily evaluate the maximum of the right expression, and we get:

$$\left|\frac{Q(t)_N - Q_{a,N}(t)}{Q_{a,N}(t)}\right| \leqslant \frac{-a_1(a_2 - 1)}{(1 - a_1)(a_2 - a_1)}\left(\frac{a_2}{a_2 - a_1}\right)^{-\frac{a_2}{a_1}}.$$

The maximum converges to 0 when N diverges, hence the convergence is uniform.

The asymptotic value is

$$\int_0^\infty Q_{a,N}(t)\,dt = 1/\beta\frac{a_2 - a_1}{(a_2 - 1)a_2}\ln\left(\frac{a_2 - a_1}{1 - a_1}\right),$$

which behaves asymptotically as

$$\frac{1}{\beta}\frac{\ln(N - \frac{\mu}{\beta})}{N - \frac{\mu}{\beta}}.$$

In the same way we have found the second bound as $\mu \to \infty$. In this case $\lim_{\mu\to\infty} a_2 = 1$, $\lim_{\mu\to\infty} a_1 = -\infty$, and we consider

$$Q_{a,\mu}(t) = e^{-a_2\beta t}.$$

$$\left|\frac{Q_N(t) - Q_{a,N}(t)}{Q_{a,N}(t)}\right| = \frac{1}{\frac{a_2-a_1}{(a_2-1)(1-e^{-(a2-a1)\beta t})} - 1}.$$

The supremum is achieved for $t \to \infty$ and is equal to

$$\frac{a_2 - 1}{1 - a_1},$$

which converges to 0 as μ diverges.

The asymptotic value is

$$\int_0^\infty Q_{a,\mu}(t)\,dt = \frac{1}{\beta a_2} \underset{\mu\to\infty}{\sim} \frac{\mu - N\beta}{\beta\mu}.$$

Finally, as regards the third bound, a closed form can be found for $E[T_d]$, considering $N = \mu/\beta$:

$$E[T_d] = \frac{2\,\mathrm{arccot}\sqrt{\frac{\sqrt{N-1}+1}{\sqrt{N-1}-1}}}{\beta\sqrt{N-2}}$$

and

$$E[T_d] \underset{N\to\infty}{\sim} \frac{\pi}{2\beta\sqrt{N-2}}.$$

Appendix E. Number of copies

In this section we show how the results about the average number of copies occurred until the delivery (C_d) can be derived.

First note that for all the considered schemes, except *those based on timers*, the number of copies (excluding the copy to the destination) coincides with the average number of infected node in the system when the packet is delivered minus one. Hence

$$C_d = \int_0^\infty I(t)P'(t)\,dt - 1,$$

where $I(t)$ is the number of infected nodes at time t, given that the packet has not been delivered at time t.

By replacing $P'(t)$ and integrating by parts, we have:

$$\begin{aligned}\int_0^\infty I(t)P'(t)\,dt &= \beta\int_0^\infty I^2(t)Q(t)\,dt \\ &= \beta\int_0^\infty \frac{I^2(t)Q(t)}{-\beta I(t)Q(t)}\,d(Q(t)) \\ &= \int_0^\infty I'(t)Q(t)\,dt + 1.\end{aligned}$$

By replacing $I'(t)$ according to the equation of the specific schemes and considering that $\int_0^\infty \beta I(t)Q(t)\,dt = P(\infty) - P(0) = 1$, we can get the following results, respectively for epidemic routing, 2-hop and probabilistic routing.

$$C_{\mathrm{ep}} = \frac{N - 1}{2},$$

$$C_{\mathrm{2hop}} = \beta NE[T_d] - 1 \underset{N\to\infty}{\sim} \sqrt{\frac{\pi}{2}}\sqrt{N},$$

$$C_{\mathrm{prob}} = \frac{p(N - 1)}{1 + p}.$$

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