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Impact of Non-Poissonian Activity Patterns on Spreading Processes

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Halting a computer or biological virus outbreak requires a detailed understanding of the timing of the interactions between susceptible and infected individuals. While current spreading models assume that users interact uniformly in time, following a Poisson process, a series of recent measurements indicates that the intercontact time distribution is heavy tailed, corresponding to a temporally inhomogeneous bursty contact process. Here we show that the non-Poisson nature of the contact dynamics results in prevalence decay times significantly larger than predicted by the standard Poisson process based models. Our predictions are in agreement with the detailed time resolved prevalence data of computer viruses, which, according to virus bulletins, show a decay time close to a year, in contrast with the 1 day decay predicted by the standard Poisson process based models.

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According to The WildList Organization International [1] there were 130 known computer viruses in 1993, a number that has exploded to 4767 in April 2006. With the proliferation of broadband “always on” connections, file downloads, instant messaging, Bluetooth-enabled mobile devices, and other communications technologies, the mechanisms used by worms and viruses to spread have evolved as well. Still, most viruses continue to spread through Email attachments. Indeed, according to the Virus Bulletin [2], the Email worms W32/Netsky.h and W32/Mytob with the ability to spread itself through Email, account for 70% of the virus prevalences in April 2006. When the worm infects a machine, it sends an infected Email to all addresses in the computer’s Email address book. This self-broadcast mechanism allows for the worm’s rapid reproduction and spread, explaining why Email worms continue to be the main security threat.

In order to eradicate viruses, as well as to control and limit the impact of an outbreak, we need to have a detailed and quantitative understanding of the spreading dynamics. This is currently provided by a wide range of epidemic models, each adopted to the particular realities of the computer based spreading process. A common feature of all current epidemic models [3–10] is the assumption that the contact process between individuals follows Poisson statistics, meaning that the probability that an agent interacts with another agent in a dt time interval is $dt/\langle\tau\rangle$, where $\langle\tau\rangle$ is the mean interevent time. Furthermore, the time τ between two consecutive contacts is predicted to follow an exponential distribution with mean $\langle\tau\rangle$. Therefore, reports of new infections should decay exponentially with a decay time of about a day, or at most a few days [3–7], given that most users check their Email on a daily basis, providing $\langle\tau\rangle$ of approximately a few days (see below). In contrast, prevalence records indicate that new infections are still reported years after the release of anti-

viruses ([2,3,11]), and their decay time is in the vicinity of years, 2–3 orders of magnitude larger than the Poisson process predicted decay times.

A possible resolution of this discrepancy may be rooted in the failure of the Poisson approximation for the interevent time distribution, currently used in all modeling frameworks. Indeed, recent studies of Email exchange records between individuals in a university environment have shown that the probability density function $P(\tau)$ of the time interval τ between two consecutive Emails sent by the same user is well approximated by a fat tailed distribution $P(\tau) \sim \tau^{-1}$ [12–17]. In the following we provide evidence that this deviation from the Poisson process has a strong impact on the Email worm’s spread, offering a coherent explanation of the anomalously long prevalence times observed for Email viruses.

Email activity patterns.—The contact dynamics responsible for the spread of Email worms is driven by the Email communication and usage patterns of individuals. To characterize these patterns we studied two Email data sets. The first data set contains Emails from a university environment, capturing the communication pattern between 3188 users, consisting of 129 135 Emails [12]. The second data set contains Emails from a commercial provider (www.free-mail.hu) spanning ten months, 1 729 165 users and 39 046 030 Emails. For the two Email data sets $P(\tau)$ is rather broad, following approximately a power law with exponent $\alpha \approx 1$ and a cutoff at large τ values (Fig. 1). Most important, the value of the cutoff depends on the time window T over which the data has been recorded [Figs. 1(a) and 1(b)]. By restricting the data to varying time windows we find that $P(\tau)$ goes to zero as $1 - \tau/T$ when τ approaches T . After correcting for the finiteness of the observation time window we obtain that the distributions for different T values collapse into a single curve [Figs. 1(c) and 1(d)], representing the true interevent time

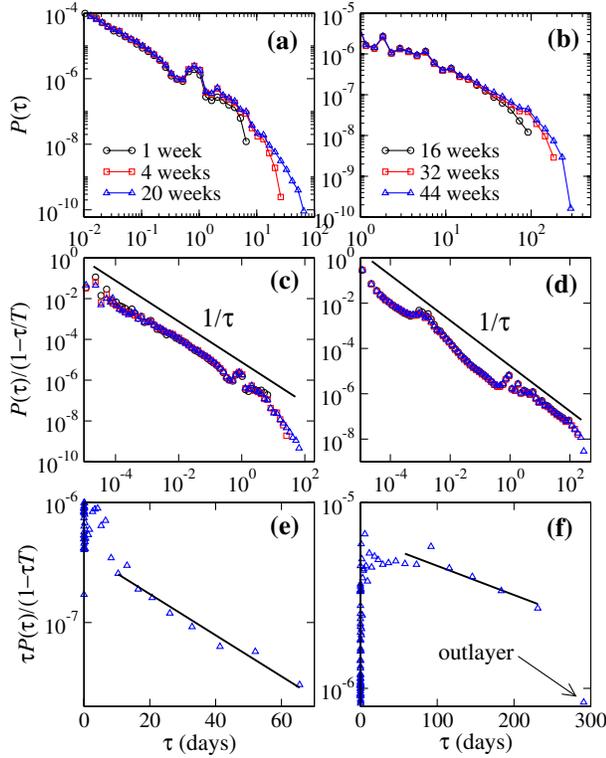


FIG. 1 (color online). Distribution $P(\tau)$ of the interevent time between two consecutive Emails sent by an Email user. The left-hand and right-hand panels represent the university and commercial data sets, respectively. For each data set we aggregate the interevent times of all users (the distribution for single users exhibits a similar behavior [15]) and apply a logarithmic binning to account for the fact that the number of observed events decreases with increasing τ . (a),(b) Log-log plot of $P(\tau)$ for $\tau > 10^{-2}$ days, emphasizing the large τ behavior for different time window sizes T . (c),(d) The same plots after removing the finite time window effects, the data collapsing into a single curve. The solid line represents the power law decay $P(\tau) \sim \tau^{-1}$. (e),(f) Semilog plot emphasizing the exponential decay at large τ , for the largest time window T . The solid lines are fit to an exponential decay $\tau P(\tau) \sim e^{-\tau/\tau_E}$ resulting in $\tau_E = 25 \pm 2$ days and 9 ± 1 months for the university (e) and commercial (f) data sets, respectively. The outlier in (f) was excluded when fitting to an exponential decay.

distribution. The obtained $P(\tau)$ is well approximated by a power law decay followed by an exponential cutoff [Figs. 1(c)–1(f)], i.e.,

$$P_E(\tau) = A\tau^{-\alpha} \exp\left(-\frac{\tau}{\tau_E}\right), \quad (1)$$

where A is a normalization factor. The power law decay at small and intermediate τ is clearly manifested on the log-log plot of $P(\tau)$ [Figs. 1(c) and 1(d)], consistent with $\alpha \approx 1$, spanning over four [Fig. 1(c)] to six [Fig. 1(d)] decades. The exponential cutoff is best seen in a semilog plot after removing the power law decay [Figs. 1(e) and 1(f)],

resulting in a decay time $\tau_E = 25 \pm 2$ days and $\tau_E = 9 \pm 1$ months (approximately 270 days) for the university and commercial data sets, respectively [see Figs. 1(c)–1(f)]. In contrast, the Poisson approximation predicts $P_P(\tau) = \exp(-t/\langle\tau\rangle)/\langle\tau\rangle$ [18], where $\langle\tau\rangle$ is the mean interevent time, taking the values 0.86 and 4.9 days for the university and commercial data, respectively.

Dynamics of worm spreading.—To investigate the impact of the observed non-Poisson activity patterns on spreading processes we study the spread of Email worms among Email users. For the moment we ignore the possibility that some users may delete the infected Email or may have installed the worm antivirus and therefore do not participate in the spreading process (we will return later to the possible impact of these events on our predictions). Therefore, the spreading process is well described by the susceptible-infected (SI) model on the Email network.

The spreading dynamics is jointly determined by the Email activity patterns and the topology of the corresponding Email communication network [12,19]. The Email activity patterns are reflected in the infection generation times, where the generation time is defined as the time interval between the infection of the primary case (the user sending the Email) and the infection of a secondary case (a different user opening the received infected Email). From the perspective of the secondary case, the time when a user receives the infected Email is random and the generation time is the time interval between arrival and the opening of the infected Email. In most cases received Emails are responded to in the next Email activity burst [12,15], and viruses are acting when Emails are read, approximately the same time when the next bunch of Emails are written. Therefore the generation time can be approximated by the time interval between the arrival of a virus infected Email and the next Email sent to any recipient by the secondary case. If we model the Email activity pattern as a renewal process [18] with interevent time distribution $P(\tau)$ then the generation time is the residual waiting time and is characterized by the probability density function [18]

$$g(\tau) = \frac{1}{\langle\tau\rangle} \int_{\tau}^{\infty} dx P(x). \quad (2)$$

Next we calculate the average number of new infections $n(t)$ at time t resulting from an outbreak starting from a single infected user at $t = 0$. Although the Email network contains cycles, it is very sparse; thus, we approximate it by a treelike structure. Previous analytical studies have shown that this approximation captures the main features of the spreading dynamics on real networks [3,20]. In this case $n(t)$ is given by [20]

$$n(t) = \sum_{d=1}^D z_d g^{*d}(t), \quad (3)$$

where z_d is the average number of users d Email contacts

away from the first infected user, D is the maximum of d , and $g^{*d}(t)$ is the d -order convolution of $g(\tau)$, $g^{*1}(t) = g(t)$, and $g^{*d}(t) = \int_0^t d\tau g(\tau)g^{*d-1}(t - \tau)$ for $d > 1$, representing the probability density function of the sum of d generation times. Substituting the Poisson approximation and Email data interevent time distributions into (2) and the result into (3), we obtain

$$n(t) = F(t) \exp\left(-\frac{t}{\tau_0}\right), \quad (4)$$

where $\tau_0 = \langle \tau \rangle$ for the Poisson approximation, $\tau_0 = \tau_E$ for the Email data, and

$$F(t) = \begin{cases} \frac{1}{\langle \tau \rangle} \sum_{d=1}^D \frac{z_d}{(d-1)!} \left(\frac{t}{\langle \tau \rangle}\right)^{d-1} & \text{Poisson approximation} \\ \sum_{d=1}^D z_d f^{*d}(t) & \text{Email data,} \end{cases} \quad (5)$$

where $f(t) = A\langle \tau \rangle^{-1} \int_{\tau}^{\infty} dx x^{-\alpha} e^{(\tau-x)/\tau_E}$. In the long time limit (4) is dominated by the exponential decay while $F(t)$ gives just a correction. The decay time is, however, significantly different for the Poisson approximation and the real interevent time distribution.

To test these predictions we perform numerical simulations using the detailed Email communication history. In this case a susceptible user receiving an infected Email at time t becomes infected and sends an infected Email to all its Email contacts at $t' > t$, where t' is the time he or she sends an Email for the first time after infection, as documented in the Email data. To reduce the computational cost we focus our analysis on the smaller university data set. The average number of new infected users resulting from the simulation exhibits daily (inset of Fig. 2) and weekly oscillations (main panel of Fig. 2), reflecting the daily and weekly periodicity of human activity patterns. More importantly, after ten days the oscillations are superimposed on an exponential decay, with a decay time about 21 days

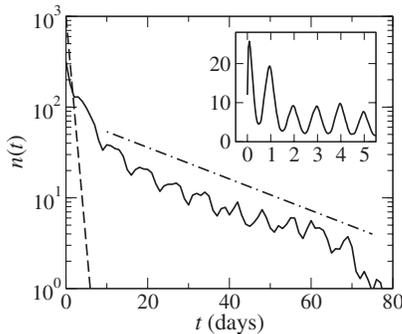


FIG. 2. Average number of new infections resulting from simulations using the Email history of the university data set (solid line), using a 1 day interval binning. The inset shows a zoom of the initial stages of the spreading process using a 1 hour interval binning. The lines correspond to the exponential decay predicted by the Poisson process approximation (dashed line) and the true interevent distribution (dot-dashed line).

[see Fig. 1(b)]. The Poisson process approximation would predict a decay time of 1 day, in evident disagreement with the simulations (Fig. 2). In contrast, using the correct interevent time distribution for the university data set we predict a decay time of 25 ± 2 days, in good agreement with the numerical simulations (Fig. 2).

The analysis of the university data set allows us to demonstrate the connection between the long τ behavior of the interevent time distribution $P(\tau)$ and the long time decay of the prevalence $n(t)$. Our main finding is that the prevalence decay time is given by the characteristic decay time of the interevent time distribution. More importantly, we show that the Poisson process approximation clearly underestimates the decay time. For Poisson processes the two time scales, the average interevent time and the characteristic time of the exponential decay coincide, being both of the order of 1 to at most a few days. Using measurements on the commercial data set, containing a larger number of individuals and covering a wider spectrum of Email users, we can extrapolate these conclusions to predict the behavior of real viruses. Given the value of τ_E for the commercial data set, we predict that the Email worm prevalence should decay exponentially with time, with a decay time about nine months. The prevalence tables reported by the Virus Bulletin web site [2] indicate that worm outbreaks persist for several months, following an exponential decay with a decay time around 12 months (Fig. 3). Our nine month prediction is thus much closer to the observed value than the $\langle \tau \rangle \approx 1-4$ day prediction based on the Poisson approximation. The fact that our prediction underestimates the actual decay time by about three months is probably rooted in the fact that the commercial data set, despite its coverage of an impressive

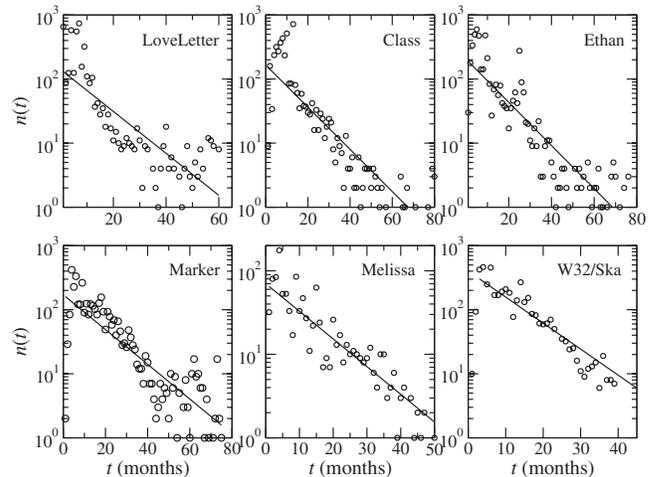


FIG. 3. Number of new infections reported for six worm outbreaks, according to the Virus Bulletin [2]. The lines are fit to an exponential decay resulting in the decay times (measured in months): LoveLetter (13 ± 2), Ethan (12 ± 1), Marker (14 ± 2), Class (12 ± 1), Melissa (13 ± 1), W32/Ska (11 ± 1).

1.7×10^6 users, still captures only a small segment (approximately 0.1%) of all Internet users.

As we discussed above, some other factors potentially affecting the spreading of Email worms were not considered in our analysis. First, some users may delete the infected Emails or may have installed the worm antivirus. Since these users do not participate in the spreading process they are eliminated from the average number of users z_d that are found d Email contacts away from the first infected user. While this would affect the initial spread characterized by $F(t)$ (5), the exponential decay in (4) and the decay time $\tau_0 = \tau_E$ will not be altered. Second, some Email viruses do not use the self-broadcasting mechanism of Email worms. For example, file viruses require the Email user to attach the infected file into a sent Email in order to be transmitted. In turn, only some Email contacts will receive the infected file. Once again, this affects z_d but not the Email activity patterns. Therefore, the prevalence of Email viruses in general should decay exponentially in time with a decay time τ_0 determined by the decay time of the interevent time distribution τ_E . Third, new virus strains regularly emerge following small modifications of earlier viruses. Within this work new virus strains are modeled as new outbreaks. An alternative approach is to analyze all strains together, modeling the emergence of new strains as a process of reinfection. In this second approach the dynamics is better described by the susceptible-infected-susceptible model [3]. Earlier work has shown that if reinfections are allowed in networks with a power law degree distribution, long prevalence decay times may emerge, which increase with increasing the network size [3]. The data shown in Fig. 3 represent, however, the spread of a single virus strain, which is better captured by the SI model. For the SI model, however, for a Poisson activity pattern we should get a rapid decay in prevalence, indicating that the empirically observed long decay times cannot be attributed to this reinfection-based mechanism.

A series of recent measurements indicates that power law interevent time distributions are not a unique feature of Email communications, but emerge in a wide range of human activity patterns, describing the timing of financial transactions [21,22], response time of internauts [23], on-line games [24], log-in times into Email servers [25], and printing processes [26]. Together they raise the possibility that non-Poisson contact timing is a common feature of human dynamics and thus could impact other spreading processes as well. Indeed, measurements indicate that the patterns of visitation of public places, like libraries [15], or the long range travel patterns of humans, involving car and air travel, is also driven by fat tailed interevent times [27]. Such travel patterns play a key role in the spread of biological viruses, such as influenza or SARS [28]. Taken together, these results indicate that the anomalous decay time predicted and observed for Email viruses may, in fact, apply more widely, potentially impacting the spread of biological viruses as well.

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