NEWS & VIEWS

COMPUTING

The wireless epidemic

Jon Kleinberg

As wireless communication technologies spread, so the potential for viruses to exploit them grows. Biological models of virus transmission will assume new relevance for assessing the emerging threat.

Ever since the first appearance of computer viruses on the digital landscape, our understanding of them has drawn on parallels with biology. Analogues of mutation, phylogenetic reconstruction and computational 'immune systems'[1] have all been investigated. But the central analogy has come from the use of epidemiological models to track how a computer virus spreads. A crucial ingredient in these models is a description of the contact network through which the epidemic propagates, where the links represent who has the potential to infect whom. Traditionally, computer viruses have propagated on networks that bear little resemblance to the networks of physical contact through which their biological counterparts spread. But a growing body of research[2–8] shows that the increasing use of short-range wireless communication networks might cause the two models to converge.

Accurately modelling the network through which a disease epidemic spreads is difficult in almost any setting. Diseases in plant populations, or animal diseases such as rabies, are heavily constrained by geographical proximity and the relatively fixed physical locations of the infected individuals[9]. Models of these diseases have been extended using detailed data on patterns of travel within cities[10,11] and by air worldwide[12] in attempts to analyse disease outbreaks in human populations.

Epidemics on the Internet are even more diverse. At the most general level, there is a distinction between computer viruses, which 'piggyback' on data exchanged between users, and computer worms, which more actively direct their own transmission through a network[13]. The networks on which these types of malicious code spread are based on patterns of file transfer or e-mail communication, or even on structures that evolve implicitly as a computer worm scans the Internet for targets.

Mathematical models of these different networks lie at various points on a broad conceptual spectrum. At one end are network models that reflect strong spatial effects, with nodes at fixed positions in two dimensions, each connected to a small number of other nodes a short distance away[9]. At the other end are 'scale-free' networks, which are essentially

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Human mobility and wireless networking could combine to abet the spread of computer viruses.

unconstrained by physical proximity, and in which the number of contacts per node are widely spread[14]. Models based on human travel data occupy an intermediate position in this spectrum of spatial constraints. The different network structures lead in turn to qualitative differences in the way epidemics spread: whereas epidemics can persist at arbitrarily low levels of virulence in scale-free networks[14,15], epidemics in simple two-dimensional models need a minimum level of virulence to prevent them from dying out quickly[9].

Very roughly, then, one could view models of biological epidemics as rooted in spatial networks, and expanding into less spatial realms to model the technologies that have accelerated human travel. Meanwhile, research on cyber-epidemics has occupied the non-spatial end of the spectrum, with its diverse and far-flung connections, when modelling global communication technologies such as the Internet.

But the spread of short-range wireless communication technologies such as Bluetooth, and the emergence of worms that exploit these systems[2–4], is disrupting this dichotomy by making possible computer-virus outbreaks whose progress closely tracks human mobility patterns. These types of wireless worm are designed to infect mobile devices such as cell phones, and then to continuously scan for other devices within a few tens of metres or less, looking for new targets. A computer virus thus becomes something you catch not necessarily from a compromised computer halfway around the world, but possibly from the person sitting next to you on a bus, or at a nearby table in a restaurant.

Wireless worms can also be used to attack 'mobile ad-hoc networks' (MANETs), which are designed to connect devices such as cheap, low-powered sensors using short-range wireless communication[5]. These networks have applications in environmental monitoring, disaster relief and military operations; when the nodes of such a system are placed in relatively fixed positions, there is a close analogy to some of the oldest and best-studied models of disease epidemics, based on short-range spatial contacts in two dimensions[9].

Although these types of worm have not yet achieved widespread penetration, prototypes have successfully exploited vulnerabilities in wireless protocols including Bluetooth, and it is to be expected that mobile devices will be increasingly targeted by malicious code. In assessing the risks of such attacks, and developing countermeasures against them, it is intriguing to contemplate how we might draw on expertise from the field of human epidemiology in understanding how contagion spreads.

These analogies will, of course, always be incomplete. In particular, the timescales over which highly successful mobile worms operate will probably be shorter than those of their biological counterparts. The initial outbreak, and the opportunities for recovery, will potentially progress much more quickly. Mobile worms are also restricted in a way that has no obvious biological analogy by the limited communication rates of the devices they infect. Particularly aggressive worms will also be confronted with the 'self-throttling' effects of many infected devices competing for limited wireless bandwidth[5,6].

Analogies to biological epidemics can also be exploited for beneficial purposes, in the design of computer-network protocols[16]. For mobile devices, epidemiology helps in dealing with the problem of intermittent connectivity: that the routing of traffic must conform to a dynamic and unpredictable network structure as the owners of mobile devices move around. The result is a growing interest in opportunistic routing, in which messages are passed between devices that come into physical proximity, with the goal of eventually reaching a specified recipient[7]. The development of such protocols has drawn on detailed data concerning human mobility and contact patterns[8].

These lines of work reinforce how the evolution of computer viruses and worms has always been closely linked with the legitimate concerns of computer networking. As that relationship extends into the domain of mobile devices, we are taking further steps towards a world where digital traffic flows not just over the wired backbone of the Internet, but also in small leaps through physical space as people pass one another on the street. ■

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AGEING

From stem to stern

Anne Brunet and Thomas A. Rando

Immortality is the stuff of myth and legend, but lifespan extension is the subject of serious scientific inquiry. Exploring the causes and effects of ageing in stem cells should aid this quest.

The explosion of research on stem cells has given the promise of treatments for degenerative diseases of ageing, enhancement of the repair of damaged tissues and possibly even slowing of decline-in-function that occurs with advancing age. But how stem cells are affected by the ageing process, and whether such changes are a cause or a consequence of organismal ageing, remain unclear[1]. Three research teams[2–4] have recently reported their findings on how age-related accumulation of DNA damage and changes in global patterns of gene expression might lead to the decline of stem-cell function.

In mammals, stem cells reside in many adult tissues and either continually produce new cells for tissues with a high turnover (blood, skin and gut) or serve as a reservoir for gradual cellular replacement or repair in the more stable tissues (liver, muscle and brain)[1]. Stem-cell function, just like that of other cells, declines with age. However, to what extent age-related changes in stem-cell function are due to intrinsic ageing of the cells or due to changes in the environment in which they reside[5] is still unclear.

Many theories have been put forth to explain the decline of cell and tissue function with age, but a main challenge for researchers who study ageing is to distinguish among potential causal influences, virtually all of which interact with one another and lead to organismal ageing (Fig. 1). The free-radical theory of ageing proposes that reactive oxygen species, which are by-products of normal metabolism, are responsible for damage to many cellular components, including DNA[6].

Several mechanisms of DNA repair that are essential for healthy tissues and long life[7] have evolved in cells of higher organisms. In humans or mice, mutations in genes encoding DNA-repair enzymes may lead to dramatic increases in the incidence of cancer and the shortening of lifespan. What has remained unclear is how susceptible adult stem cells are to the age-