Progmosis: Evaluating Risky Individual Behavior During Epidemics Using Mobile Network Data

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Abstract

The possiblity to analyze, quantify and forecast epidemic outbreaks is fundamental when devising effective disease containment strategies. Policy makers are faced with an intricate task of drafting realistically implementable policies that strike a balance between risk management and cost. Two major techniques policy makers have at their disposal are: epidemic modelling and contact tracing. Models are used to forecast the evolution of the epidemic both globally and regionally. While contact tracing is used to reconstruct the chain of the people that have been potentially infected, so that they can be tested, isolated and treated immediately. However, both techniques might provide limited information, especially during an already advanced crisis when the need for action is urgent.

In this paper we propose an alternative that goes beyond epidemic modelling and contact tracing, and leverages behavioral data generated by mobile carrier networks to evaluate contagion risk on a per-user basis. The individual risk represents the loss incurred by not isolating or treating a specific person, both in terms of how likely it is for this person to spread the disease as well as how many secondary infections it will cause. To this aim, we develop a model, named *Progmosis*, which quantifies this risk based on movement and regional aggregated statistics about infection rates. We develop and release an open-source tool that calculates this risk based on cellular network events. We simulate a realistic epidemic scenario, based on an Ebola virus outbreak; we find that gradually restricting the mobility of a subset of individuals reduces the number of infected people after 30 days by 24%.

1 Introduction

The world is now facing many severe healthcare challenges and, indeed, the recent Ebola outbreak seems one of the most worrisome and urgent. Mr David Nabarro, Special Envoy of the UN Secretary-General said at an informal UN meeting that he had never encountered a challenge like Ebola in 35 years of his professional life: "This outbreak has moved out of rural areas and it's com-

ing to towns and cities. It's no longer just affecting a very well-defined location, it's affecting a whole region and it's now impacting the whole world"[1].

Nowadays transportation systems make it possible for people to travel easily across a country and across the globe, but, unfortunately, they make that possible for diseases too. The spread of diseases is facilitated by today's rich transportation networks that enable human disease carriers to quickly move across distant regions Merler and Ajelli (2010). In this context, drastic measures like banning transportation to disease-affected areas are difficult to implement, have a high cost and are actually believed to worsen the outbreak Chamary (2014) Meloni et al. (2011). The need for smaller, targeted interventions matches the increasing availability of large-scale data, especially coming from mobile networks. The benefit of mobile-phone records to combat quickly-spreading diseases like Ebola is unquestionable Economist (2014).

When an outbreak becomes global, an infected person can be found anywhere, in cities as well as rural areas, and regardless of country boundaries; this might suggest that no place is really safe. However, we argue that some people and places are more exposed to risk than others.

We propose to use such heterogeneity to our advantage and to use mobile networks to unveil such heterogeneity. We envision a system that utilizes the data coming from mobile carriers and, where available, social networks and smartphones, to construct *individual-based risk models*. The system can assess the risk associated with a person, primarily based on that person's mobility patterns and, optionally, on other demographic or behavioral indicators that can be inferred from the data. We would like to characterising features of the proposed solution: first, it can use data that is readily available (such as cellphone carrier data), and second, it is be able to operate under uncertainty (it does not require the knowledge of the identity of the infected).

The risk model can be used in several real-world scenarios, especially when urgent response is required. Thus, the model can be used to answer the following questions. Who should be tested early for signs of the disease, and possibly put into quarantine if positive, given that vaccinations can be produced and performed with a certain rate? Who should get vaccinated first? Who should receive information about prevention, for example by means of text messages? All these scenarios describe individual-based interventions that are very hard to administer quickly over large populations. This model can prioritize the people to be targeted with the intervention sooner rather than later.

2 Motivation

People behaviour is highly heterogeneous. Existing epidemic models are based on analyses conducted at population level to assess how infectious a disease is, based on the basic reproductive ratio r_0, i.e., the average number of secondary cases generated by a single infected person. However, several studies have investigated that spreading processes are usually highly heterogeneous and some individualsm account for a large proportion of the spreading. The presence of these influential spreaders has been investigated for generic networks Kitsak et al. (2010), as well as in epidemics processes. Superspreading seems to be a common featufire of the spread of diseases and targeted individual-based control measures are much more effective than population-wide measures, as reported by Lloyd-Smith et al. Lloyd-Smith et al. (2005). For this reason, identifying superspreaders is extremely important in order to contain epidemics.

Existing techniques, such as contact tracing, are not sufficient. Moreover, efforts in fighting disease outbreaks mainly focus on contact tracing techniques, as it is happening for Ebola Murphy (2014). Contact tracing works by finding all the people who have been in contact with an infected person, and then interviewing, monitoring, isolating them when necessary. The process is repeated for everyone who is found to be infected. While contact tracing can be effective, it has some drawbacks. First of all, information provided by people might be subject to errors, due to fear, shame, faulty memory or other reasons. Secondly, contact tracing needs time: contact tracing only starts when a person is diagnosed with the disease already, or at least shows symptoms. Tracing the contacts also takes time: if the disease has an asymptomatic phase or highly infective, the contacts might be likely to have infected others before they are traced.

[1] `http://webtv.un.org/watch/david-nabarro-ebola-virus-outbreak-general-assembly-informal-meeting-69th-session-10-october-2014/3832613824001`

Localization techniques have already been used successfully during critical scenarios. Recently, Nigeria also resorted to GPS technology to improve, scale up and speed up contact tracing, repurposing GPS devices used for polio vaccinations Fasina et al. (2014); Gates (2014). The huge effort of the country resulted in eradication of Ebola and Nigeria was declared "Ebola-free" by the WHO (cite one of the many news that report this). While this success story demonstrates how location tracking can be very useful during similar scenarious, the very same strategy could have not been used if the epidemic was in a more advanced state, i.e., if many more people had already been infected. For this reason, we think it is very important to investigate the use of alternative systems that can provide coarser location tracking but for a very large number of individuals.

Medical treatment is scarce and costly. For example, in the case of Ebola, while it is seen as a serious challenge by the whole world, vaccinations have to face serious technical and financial issues[2]). When a commodity such as vaccinations is scarce, who should be given priority to receive vaccination?

3 Risk Model

In this section, we propose a method to quantify the risk associated with each person during an outbreak, depending on their mobility behavior, inferred from their phone-activity. Here we refer it as the risk model. Our goal is not the estimation of the individual cost (i.e., the chance of getting infected), but the cost that an entire community faces by not treating a specific person. Early testing, medical treatment, vaccination, quarantine of specific individuals might reduce cost sustained by the community at later time.

A general estimate of the total risk R associated to a set of events E is defined by:

$$R = \sum_{E} P_E \times L_E \tag{1}$$

where P_E and L_E are the probability and the expected loss for each event, respectively Vapnik (1998).

We bring this definition to the epidemiology domain by considering a scenario in which several geographic areas are associated different values of contagion risk that change in time. The risk measures how likely it is for an individual to get infected in a region. As in common models of infectious diseases, we assume it is directly proportional to the fraction of infected people in the region and we also assume homogeneous mixing within the region. Similarly, we assume that the risk to infect a healthy individual is directly proportional to the fraction of susceptible people in the region.

By staying in a geographic area with non-zero risk, a person will have some chances to get infected; the same person will also have a chance to infect someone else, increasing the risk of the geographic area. When moving between two or more areas, the person will affect the risk of these areas. We will not determine whether each person is in a susceptible, infective or recovered state. Instead, we will consider them in all the states and we will assess how risky their mobility behavior is.

In general, the way people transmit disease across geographic areas has been extensively studied in literature Bajardi et al. (2011); Balcan et al. (2009a); Merler and Ajelli (2010). Most of the studies dealing with the effects of mobility on epidemic spreading usually make the assumption that the mobility patterns of individuals in a subpopulation are homogeneous Colizza and Vespignani (2007), while they are indeed highly heterogeneous Dalziel, Pourbohloul, and Ellner (2013); Merler and Ajelli (2010). This is particularly true for developing countries, where highly irregular and temporally unstructured contact patterns have been observed Vazquez-Prokopec et al. (2013).

We consider a disease that has contagion rate per contact β (i.e., given a friendship between an infected and a susceptible person, a contagion will happen with rate β). Assuming the user u spends $T_{u,l}$ fraction of his time in each location $l \in \mathcal{L}_u$ (hence, $\sum_i T_{u,i} = 1$) we define a time-dependant contagion risk:

$$C_u(t) = \beta \sum_{l,m \in \mathcal{L}} T_{u,l} T_{u,m} [i_l(t) s_m(t) + i_m(t) s_l(t)]. \tag{2}$$

[2] http://news.sciencemag.org/health/2014/10/leaked-documents-reveal-behind-scenes-ebola-vaccine-issues

where $i_l(t)$ and $s_l(t)$ refer to the of fraction of infected and susceptible population in location l at time t, respectively. Note that now the probability of the event occurring, in this case, is the probability that a person becomes infected in a region, according to the time fraction spent there, while the expected loss is the number of people expected to be infected in another region, according to the time fraction spent there. As we do not know where the person might be infected, this formula accounts for all the combinations, which are assumed as equally likely. The maximum risk value, for a specific state of the network, is reached by an individual who equally spends his time in the region with the highest infected fraction of individuals and in the region with the highest susceptible fraction. We might calculate this normalized value but, for ranking purposes, it is not necessary, as it is a common factor; we can also ignore the rate β for the same reason.

Our proposed model could also be generalized by defining different risk classes depending on demographic indicators, which can be inferred from mobile data Zhong et al. (2013) or other behavioral indicators, such as those provided with the D4D-Dataset de Montjoye et al. (2014). It is important to emphasize that our model uses only information that is either already collected when outbreaks occur (e.g., estimated number of infected people in various geographic regions) or that can be obtained from telecommunication companies, provided that such use is compatible with existing legislation in the country.

4 Evaluation

Next, we evaluate the effectiveness of the risk identification and containment model proposed above. We set up a realistic epidemic scenario and perform stochastic simulations, following an approach similar to that implemented in GLEaM Balcan et al. (2009a), while keeping track of the movement of individuals following the real traces found in the dataset. We use the SEIR model, where each individual can be in one of the following discrete states at any given time instant: susceptible (S), exposed (E), infected (I), permanently recovered or deceased (R). This model has been used for the 2002 seasonal influenza outbreak Balcan et al. (2009a) and the 2014 Ebola outbreak Althaus (2014), among other outbreaks. It is described by the following set of equations:

$$\frac{dS}{dt} = -\beta S(t) I(t)/N \tag{3}$$

$$\frac{dE}{dt} = \beta S(t) I(t)/N - kE(t) \tag{4}$$

$$\frac{dI}{dt} = kE(t) - \gamma I(t) \tag{5}$$

$$\frac{dR}{dt} = \gamma I(t) \tag{6}$$

We inform a spreading model with the realistic parameters taken from estimates of the 2014 Ebola outbreak in Sierra Leone Althaus (2014), as reported in Tab. 4. Where σ^{-1} and γ^{-1} are the average durations of incubation and infectiousness, respectively. The transmission rate per day in absence of control interventions is β, and $r_0 = \beta/\gamma$ is the basic reproduction number.

σ^{-1}	5.3 [days]
γ^{-1}	5.61 [days]
r_0	2.53
β	0.45

Table 1: Parameters assumed for the simulation.

We simulate the epidemics in the following different contexts:

- in total absence of any treatment;
- when treatment is given with rate ξ per day and people given treatment are chosen randomly;
- when treatment is given with rate ξ per day to highest ranked people, according to the risk measure C_u.

For simplicity, in this paper we focus only on treatment that takes the form of travel restrictions, not allowing high-risk individuals to travel outside the metapopulation they are found when the treatment is applied. This is an extreme scenario, realistic only for diseases for which specific treatments or vaccinations are not available (e.g., Ebola virus). Without loss of generality, we can investigate the effects of vaccination and/or early treatment of people with higher-risk movement patterns. Since we use the same parameters for each metapopulation, and the treatment does not directly affect the epidemic process (i.e., it is not a vaccination or a cure) but only the movement of individuals, the local epidemic profiles will be similar and will be more or less shifted in time, depending on the travel fluxes. We will first show how much we can reduce synchronization by restricting the travel of high-risk individuals in a simple example.

As an illustrative case, we simulate a synthetic model. In Fig. 1 we show the total number of infections since the beginning for two metapopulations, in two specific contexts. Individuals are equally assigned to either metapopulation and they belong to two classes: a fraction of people $(1-f)$ who do not travel out of their metapopulation, and a fraction of people f who spend an equal amount of time, on average, in both. We use SEIR with the parameters mentioned before and we initialize the epidemics with a single infected case in one of the two metapopulations, chosen randomly. The top plot ($f = 0.1$) shows a high level of synchronization, while the bottom plot ($f = 0.01$) displays a clear delay in the growth of the epidemic size.

We then test our approach initializing simulations with real-data, so that a single randomly chosen region is the unique source of infection with 100 cases. We use the first six months, from January to June 2013, to learn the movement habits of individuals. Then we perform simulations under the three scenarios mentioned above: no countermeasures, people quarantined randomly and people quarantined according to their risk rank. We set an adaptive quarantine rate of $\xi = \beta i(t)$ to match the countermeasure efforts with the speed of growth of the outbreak. Fig. 2 shows results for the month of July 2013, in terms of how the global prevalence of the disease changes in time in the three cases. Despite the number of randomly quarantined people is pretty high at the end of the month (10% of the population), it does not delay the spreading. Targeted quarantine based on risk, instead, manages to delay the spreading; at the end of the month there are 24% fewer infected individuals than in the baseline cases.

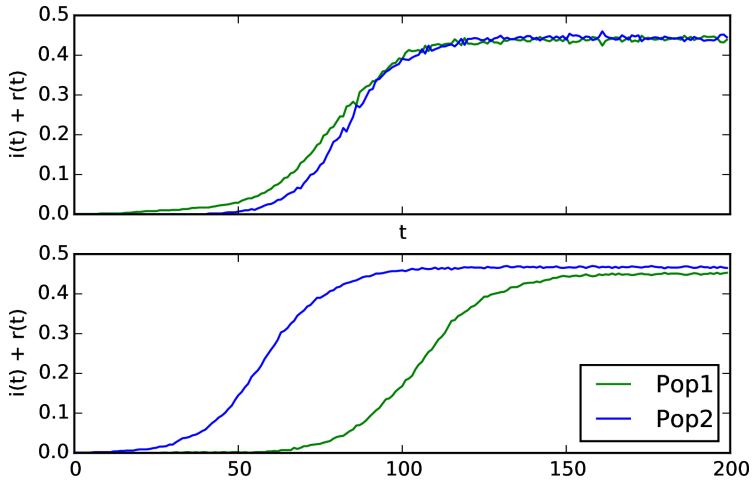

Figure 1: A simple example with two metapopulations composed by people who stay always in their own metapopulation and a fraction f of people who move between them randomly. In the top figure $f = 0.1$, while in the bottom figure $f = 0.01$. The outbreak dynamics in the second case are less synchronized.

This effect is obtained by restricting individuals who are in the areas with higher risk, specifically those who travel to low risk areas. This determines an increased number of infection cases in high-risk areas, as shown in Fig. 3 and a decreased number of infection cases in low risk areas, as shown in Fig. 4.

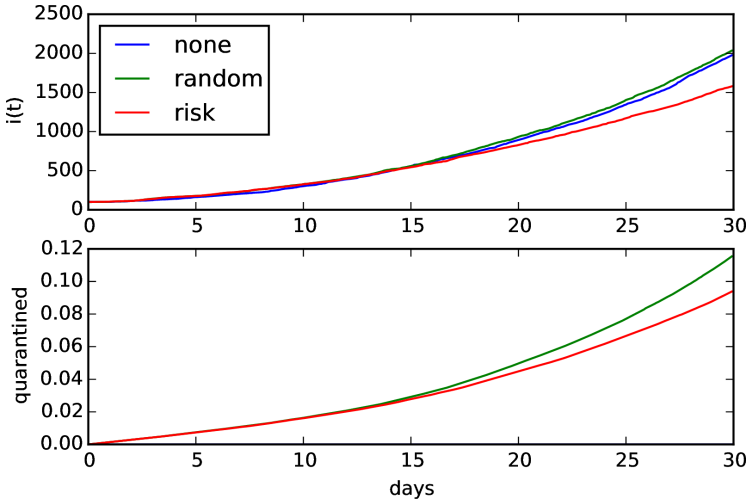

Figure 2: The top plot shows how the total number of infected people changes in time when no countermeasures are taken (none), when people are quarantined randomly (random) and according to the highest risk rank (risk). The bottom plot shows how the number of people who have been put into quarantine grows in time. The proposed identification method reduces the number of infected individuals with fewer people in quarantine, using only aggregated information of the number of infected and mobility patterns from mobile phone data providers.

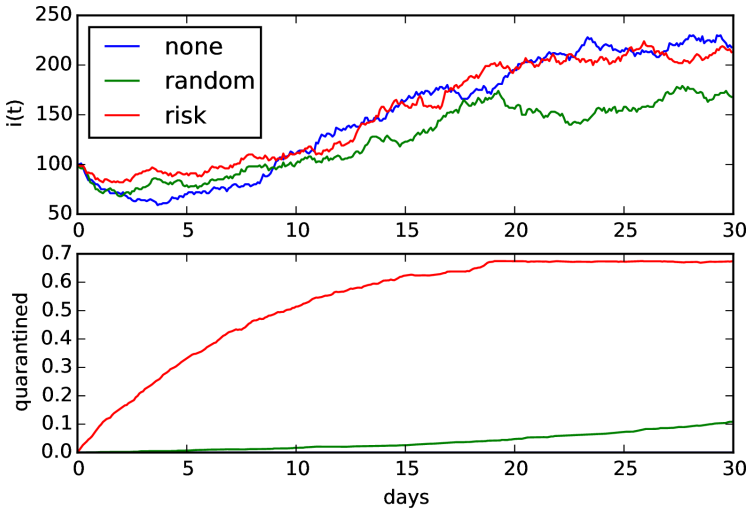

Figure 3: Number of infected (top plot) and quarantined (bottom plot) in the region where the first cases were initalized (hence, a region with higher risk than the others). Our proposed approach determines an increased number of infections in this region, while reducing the total aggregated number of infections.

5 Discussion and Limitations

This model assesses risk using data collected from mobile phones, hence it excludes people who do not use the mobile phones or share them with others. Since mobile penetration rates is already high and increasing in the vast majority of countries, including developing countries, we believe this problem will fade out as time goes. Another potential problem when dealing with network-data is their sparseness of the call activity, but recent studies try to overcome this limitation Leontiadis et al. (2014) by interpolating information in space and time. Furthermore, we would like to remark that the goal of this method is not to find *every* high-risk individual, but a *large proportion* of them,

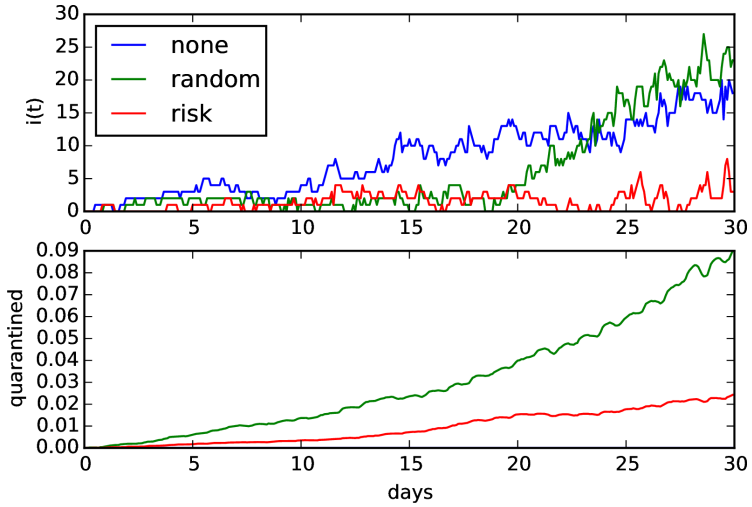

Figure 4: Number of infected (top plot) and quarantined (bottom plot) in a low-risk region. Our proposed approach determines a decreased number of infections in regions that have been less affected by the epidemic, such as the one shown here; this determines a delay in how the global number of infected grows in time.

given the data available. Moreover, it is worth noting that this method might be also be combined with other existing disease prevention and containment techniques already in use, such as contact tracing.

The model described in this paper requires access to sensitive data about individual call and mobility patterns. It is very important to take into account the important ethical and legislative issues arising from the use of these highly personal data. However, solutions based on the analysis of mobile data, such as that presented in this work, can play a critical role during emergencies. For this reason, we believe it is acceptable to use such system when the benefits exceed the risks. We envision the use of such a system only in well-defined circumstances, within specific time intervals and geographic boundaries, within the limits defined by the law and under user informed consent. The model could also be used to design a system that informs users only the users themselves about their own behavior, evaluating their the risk level and, potentially, suggesting them appropriate actions tailored to their risk profile (e.g. get tested, seek help, change lifestyle habits, etc.).

Finally, it is worth noting that we evaluate the model on traces that correspond to an epidemics-free case. People might change their mobility behaviour once they are aware of the epidemic Meloni et al. (2011). Future adaptations of the model might estimate this change by analysing mobility data in real-time.

6 Related Work

Human behaviour can have a significant impact on infective disease dynamics. In turn, a complex interplay of disease spread, awareness of the disease, and population beliefs affect human behaviour Funk, Salathé, and Jansen (2010). The mobility of a person, whether that person is infected or not, is a particularly important factor of disease spread Rizzo, Frasca, and Porfiri (2014). Awareness-induced changes in movement patterns, such as a decision to avoid unsafe infected areas, often have a detrimental effect and might lead to even higher disease spreading, since they result in bringing the infection into previously isolated communities Meloni et al. (2011); Wang et al. (2012). At the same time, international travel restrictions have been shown to have a limited impact on disease spreading, due to the high heterogeneity of human mobility patterns Bajardi et al. (2011). In fact, it is this heterogeneity, both in terms of population behaviour and a-priori infections, that drives disease development. In her discussion of HIV and other STDs transmission Aral argues that bridge groups, such as truckers, the police and the military personnel, transmit infections from highly infected groups, e.g., sex workers, to previously uninfected populations Aral (2000). Our work is

founded on the above observation, and we propose a model that explicitly takes the transmission of risk into account. While previous models consider artificial simulations Buscarino et al. (2014) and long-distance Merler and Ajelli (2010) or multiscale Balcan et al. (2009a) mobility networks in order to quantify possible outcomes of different metapopulations movement patterns on disease spread, we build our model upon individual mobility and interactions, as recorded by fine-grain cellular network traces.

Our work relies on mobile phone call records for estimating risk transfer in a population. The suitability of CDRs for tracking population movements and identification of spatial events in populations has been shown by Bengtsson et al. Bengtsson et al. (2011) and Candia et al. Candia et al. (2008). Furthermore, when it comes to infectivity modelling, in Eames, Read, and Edmunds (2009) Eames et al. show that simple interaction potential measures, such as the total number of a user's connections (total degree), perform almost as well as more complex measures of interaction, such as individually weighted links. In further work the total node degree might be used to approximate a user's potential for contact. Finally, in this work we do not modify the interaction network over time. Such modifications, orthogonal to our approach, are discussed in Kamp (2010), and can be accounted for by having a time-dependent contact network.

7 Conclusions

In this paper we have propose Progmosys, a model that goes beyond traditional epidemic modelling and contact tracing, and leverages behavioral data generated by mobile carrier networks to evaluate contagion risk on a per-user basis. The individual risk represents the loss incurred by not isolating or treating a specific person, both in terms of how likely it is for this person to spread the disease as well as how many secondary infections it will cause. We have developed and released an open-source tool that calculates this risk based on cellular network events. We have also simulated a realistic epidemic scenario, based on an Ebola virus outbreak. We have found that gradually restricting the mobility of a subset of individuals greatly reduces the number of infected people.

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