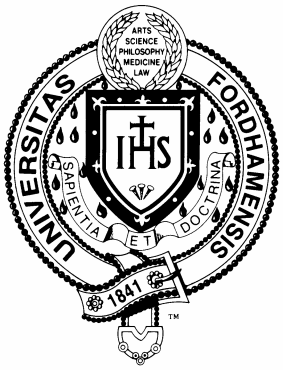

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Targeted Vaccine Subsidies for Healthcare Workers*

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Abstract

We study the public goods problem associated with vaccinations. The externality created by an infection is composed of two parts, the probability of infection and the marginal infections generated if infected. We argue that the key component in a successful vaccination strategy is the second of these items but that current public policy focuses on the first. We use a newly collected data set coupled with agent-based simulations to study the spread of influenza and other infectious diseases in hospitals. We estimate the marginal infections created by various worker groups in a hospital in order to prioritize vaccine allocations across different healthcare worker groups in times of vaccine shortages. One primary focus of this paper is identifying the individual hospital workers who are most important to vaccinate. Surprisingly, we find that many groups with patient care responsibilities, such as physicians, play a small role in spreading influenza while others, such as unit clerks, play a much larger role.

Keywords: Influenza Vaccine; Social Networks; Vaccine Subsidies

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1 Introduction

Vaccines are a primary way to stop or slow the spread of many infectious diseases, perhaps most notably, influenza. However, vaccines pose a form of the traditional externality problem, where each vaccination performed extends benefits to the individual vaccinated as well as individuals other than the recipient. A vaccine helps to protect individuals who come in contact with a vaccinated agent from being infected by the agent.

The lack of appropriate vaccination levels is a major health problem. For instance, influenza is a major cause of morbidity and mortality throughout the world despite the availability of a highly effective and inexpensive vaccine. In the U.S. alone, influenza causes an estimated 36,000 deaths and 120,000 hospitalizations annually yet only around 1/3 of healthcare workers are vaccinated each year (Thompson et al. 2003).

In this paper we study the use of vaccination subsidies as a means to increase vaccination levels. Subsidies for vaccinations contain a difficult problem in that the positive externality associated with a vaccination is the product of the probability of infection, the cost of the infection, and the marginal infections generated by an agent if infected. Yet, even with a subsidy the agent will not take account of the last of these items in her decision. Thus if the marginal infections generated by an infected agent vary across agents, it may be ineffective to set an across-the-board subsidy for all vaccinations performed. Since agents in a population have heterogeneous contacts with other agents (both in numbers and in specific contacts) public heath officials must understand the implications. This is the first paper to use specific micro-level contact data within a hospital that can be used to guide policy makers and public health officials in the problem of efficiently allocating vaccines.

Traditionally, epidemiology research has focused on well-mixed (randomly mixed) populations where agent contacts are homogeneous. Every agent in a population may "bump into" any other agent with equal probability, much as a gas molecule may bump into any other gas molecule with an equal probability over a fixed time period. Only recently have epidemiologists and other researchers begun to study the heterogeneous contact structures

between people over which infectious diseases spread (Comins et al. 1992, Grenfell and Harwood 1997, Wallinga et al. 1999).

We focus this study on healthcare workers and a particular infectious disease, influenza. Healthcare workers are at especially high risk of contracting influenza. One study of healthcare workers with a low rate of influenza vaccination demonstrated that 23% of healthcare workers had evidence of influenza infection during a single influenza season (Elder et al. 1996). Two features of influenza make its spread difficult to control in hospitals. First, people with influenza are infectious 1 to 4 days before the onset of symptoms. Thus, they can spread the virus when they are still feeling well and are unaware of their own infectious state. Second, only about 50% of infected persons develop classic influenza symptoms (CDC 2003, CDC 2002.) Consequently, restricting healthcare workers with influenza-like symptoms from the workplace will not completely prevent transmission of influenza because healthcare workers with atypical symptoms could continue working and spreading the virus. Furthermore, studies show that healthcare workers are more likely than other workers to return to work early or to keep working when they have influenza-related symptoms (Weingarten 1989).

Because of the ease with which influenza may be contracted and spread by healthcare workers, the Centers for Disease Control and Prevention (CDC) have, for the past two decades, recommended influenza vaccination for all healthcare workers. Yet, in the U.S., only 36% of workers who have direct patient contact are immunized against influenza annually (Smith and Bresee 2006).

Outside of concerns about traditional influenza, there are additional reasons to study the spread of infectious diseases in hospitals. First, healthcare-associated infections affect about 2 million patients in U.S. hospitals each year (Jarvis 1996). Second, there is a growing fear that hospitals could become a breeding ground for new strains of influenza such as the recent swine flu outbreak, the potential emergence of person-to-person transmission of avian flu, or other "new viruses." Much as SARS spread widely in hospitals to begin the SARS epidemic in Toronto (Chowell 2004), person-to-person transmission of avian flu may start in

hospitals as well, and, if a more lethal version of swine flu were to develop, hospitals again could be a breeding ground for new infections. This last point is of particular salience. With the recent swine flu outbreak and the subsequent work to develop a vaccine, questions are sure to arise in the coming months concerning which individuals to vaccinate first. It is likely that healthcare professionals will be high on the list. But, as we show below, not all healthcare workers are equal in terms of their importance in spreading infectious diseases. Thus, one primary focus of this paper is identifying the individual hospital workers who are most important to vaccinate.

There is a growing literature in economics on the vaccination choices of individuals and of the externalities associated with vaccinations. But scant attention is paid to heterogeneous contacts. For example, Francis (2004) solves for the optimal tax/ subsidy policy for influenza in an SIR model with a constant contact rate and random mixing among the population. Geoffard and Philipson (1997) examine how the individual incentives for vaccination decrease as disease incidence decreases and thereby argue that relying exclusively on private markets is unlikely to lead to disease eradication. Boulier et al. (2007), the most similar paper to ours, investigate the magnitude of the externality associated with a vaccination as a function of the number of vaccinations, the transmission rate of the disease, and the efficacy of the vaccination. They find non-montonic relationships between each of these items and the magnitude of the vaccine externality. However, like Francis, they do not consider the case of heterogenous contact number or heterogenous sorting among the population. Finally, much of the recent literature on the economics of infectious disease is summarized in Philipson (2000).

Even though there is great concern over the spread of infectious diseases in hospitals, there is no theoretical foundation that can be used to identify healthcare workers who are most likely to acquire and transmit infectious diseases in hospitals. If a vaccine is available for an infectious disease and the vaccine is in short supply or is expensive, it is imperative to know which individuals should have the highest priority in vaccine campaigns. In this study

we will use the foundations of social network theory to measure and understand the complex, interdependent relationships between persons such as healthcare workers and patients to help develop effective vaccination strategies.

To achieve this goal, we have collected person-to-person contact information on 140 individuals belonging to one of 15 types of healthcare workers at the University of Iowa Hospitals and Clinics (UIHC). The data contain information on the contacts between healthcare workers and patients and between healthcare workers and other healthcare workers at the hospital. With this information we develop a network model describing the spread of influenza in a hospital and estimate, through simulations, the effect of immunizing different hospital worker groups on the spread of infectious diseases in a hospital. Through these simulations we are able to identify the hospital worker groups that create the largest externality if vaccinated. We argue that methods such as those used in this paper can be used by hospitals, health care professionals, and epidemiologists to design efficient subsidy programs for healthcare worker vaccinations.

The data used in this paper is unique and detailed in comparison to other studies. We know of only one other paper that specifically uses micro-level data on hospital worker contacts within a hospital. Ueno and Masudo (2009) collect data on contacts between doctors, nurses, and patients. Their data is based on two calendar days from a small, 129 room, community hospital in Tokyo. They model contacts between nurses, physicians and patients on the basis of after-the-fact annotations in patient records (for doctor-patient and nurse-patient contacts) and uses "same unit" or "same ward" criteria to determine whether doctor to doctor, doctor to nurse, or nurse to nurse contacts occur. (In other words two healthcare workers are assumed to come in contact with each other if they work in the same hospital unit but actual contacts may or may not occur and are not directly observed.) In addition their paper does not consider contacts with and between other healthcare worker groups (other than nurses and doctors), patient families, and other visitors. Based on our data at the UIHC, these assumptions would ignore over 60% of hospital staff, including most

of the groups we identify as most crucial to the spread of an infection disease. In contrast, the data used in our paper consists of shadow data (where a research assistant follows a specific, randomly chosen hospital worker for an entire shift) for the 15 major groups of healthcare workers at the UIHC, a 700-bed major medical center. This results in over 600 hours of direct hospital worker observations and the notation of over 6,500 specific worker to worker or worker to patient contacts throughout the hospital. To the best of our knowledge, the data that we have collected comprises the most detailed micro-level healthcare worker contact data set in existence.

We begin by developing a simple model of infectious disease transmission. In the model, we initially assume homogeneous contacts as in traditional epidemiological models. We then discuss a similar model with heterogeneous contacts and explore the difficulties of using across-the-board subsidies to achieve efficient vaccinations. Following the theoretical discussion, we use our newly collected data on healthcare worker and patient contacts to simulate the spread of an infectious disease in a hospital setting. The simulations allow us to identify the healthcare worker groups that would be expected to play the largest role in the spread of infectious diseases in this hospital setting.

2 Background and a Simple Model

We begin by describing a simple model where agents in a population have contacts with each other with a uniform probability (this is the traditional random mixing model used by epidemiologists). The important results in this paper describe exceptions to this homogeneous contact assumption, but we use the simplified model to develop intuition before describing a richer model with heterogeneous contacts. In this simple model, we assume that all agents are homogeneous in that all agents have the same number of contacts with other agents and that these contacts are randomly drawn with uniform probability from the population at large.

Suppose that agents are assigned to one of three states: Susceptible (S), Infected (I), or

Recovered (R). A susceptible agent can transition to being infected with probability α if she is in contact with an infected agent. Once infected, an agent transitions to the recovered state according to a parameter κ. Once recovered, the agent is immune to the possibility of future infection. This is a classic SIR model for infectious diseases such as influenza. The description and parameters yield the following differential equations describing the flows of agents among the various states, assuming a constant population of size N and contact rate of γ. Each equation describes the rate of growth for one of the three populations in the Susceptible - Infected - Recovered (SIR) model.

$$\frac{dS_t}{dt} = -\alpha\gamma S_t \frac{I_t}{N} \tag{1}$$

$$\frac{dI_t}{dt} = \alpha\gamma \frac{S_t}{N} I_t - \kappa I_t \tag{2}$$

$$\frac{dR_t}{dt} = \kappa I_t \tag{3}$$

Equation 1 describes how susceptible agents contact γ other agents in the population, of which I_t/N are infected, and how, of these contacts with infected agents, a percentage α cause the susceptible agent to transition to being infected. Equation 2 describes the previously mentioned flows from susceptible into infected and that each infected agent moves to being recovered at rate κ. Finally, Equation 3 describes the flows from infected to recovered.

We can write these equations in terms of population shares by dividing each of the above equations by the population size N and using lower case letters to denote these population shares, $s_t = S_t/N$, $i_t = I_t/N$, and $r_t = R_t/N$, yielding the following population share equations:

$$\frac{ds_t}{dt} = -\alpha\gamma s_t i_t \tag{4}$$

$$\frac{di_t}{dt} = \alpha\gamma s_t i_t - \kappa i_t \tag{5}$$

$$\frac{dr_t}{dt} = \kappa i_t \tag{6}$$

Now note that an epidemic begins when an initial set of infected agents causes infections in the population to increase or when $\frac{di_t}{dt} > 0$. In other words the flows into the infected state from the susceptible state must exceed the flows out of the infected state into the recovered state. This condition is equivalent to $\alpha\gamma s_t > \kappa$ or $s_t > \frac{\kappa}{\alpha\gamma}$. If this inequality holds then we say that the population is above the *epidemic threshold*. Note that we cannot remain above the epidemic threshold forever without an introduction of new susceptible agents since $\frac{ds_t}{dt} < 0$: eventually the population will run out of susceptible agents to infect unless the susceptible population is replenished at a sufficient rate.

The goal of public policy is to attempt to place a population below the epidemic threshold so that the number of infectious agents in a population do not grow subject to some cost constraint. A population is most vulnerable to being above the epidemic threshold when the infectious disease first enters a population because $s_t \approx 1$. This implies that each infected agent infects approximately $\frac{\alpha\gamma}{\kappa}$ new agents in the population. This fraction is sometimes referred to as the initial *reproduction number* in the population and is commonly denoted as $R_0 \equiv \frac{\alpha\gamma}{\kappa}$. Without new individuals entering a population in the susceptible state this reproduction number can only decline as the infectious disease spreads.

2.1 Vaccinations

A successful vaccination moves an agent from state S to state R without incurring the costs of infection. If we reduce the initial population of susceptible individuals, s_0, by enough we can push the population below the epidemic threshold. In other words, if s_0 is such that $s_0 < \frac{\kappa}{\alpha\gamma}$ then the infectious disease dies out of its own accord without infecting additional

agents beyond those initially infected. Thus an epidemic is prevented whenever $s_0 < \frac{\kappa}{\alpha\gamma}$ which occurs when $(1 - \frac{\kappa}{\alpha\gamma})N$ agents are successfully vaccinated. Vaccinating enough agents to produce this effect is called *herd immunity*; once enough people are vaccinated, the entire population (herd) is effectively protected without everyone being vaccinated. This is an upper bound on the number of agents to vaccinate in a population. As long as the vaccine has some positive cost, $c(v) > 0$, we may not want to vaccinate more agents than is required to produce herd immunity.[1] The question then becomes, given a cost of vaccination, $c(v)$, what is the efficient level of vaccinations to provide in a population and how do we obtain this efficient level? We begin to approach this question by introducing standard value function notation.

In this initial model, once an agent enters state R she remains there forever. Thus the value of being in state R is simply the lifetime discounted utility received in state R. We also introduce the possibility of having heterogenous contacts at this stage by indexing agent j's contacts (γ_j) and other terms that we allow to vary across agents.

$$V_j(R) = \int_{t=0}^{\infty} \beta^t U_j(R) \tag{7}$$

Where $U_j()$=utility of agent j from the specified state and β is the discount rate.

If an agent is in state I, she will remain in state I for $1/\kappa$ periods until recovered and then enter state R.

$$V_j(I) = \int_{t=0}^{1/\kappa} \beta^t U_j(I) + \int_{t=1/\kappa}^{\infty} \beta^t U_j(R) \tag{8}$$

If an agent is in state S, she receives the same utility as she would if she was recovered, unless she becomes infected. The value to an agent of being in state S is the value of being in state R less the product of the probability that the agent becomes infected and the cost

[1] Of course there are some exceptions to this idea. As an example, suppose that a disease is automatically fatal to anyone infected and that there is some small probability of acquiring the disease from the natural world in addition to acquiring it from other agents. Then you may want to vaccinate an entire population to protect against non-social transmission even if herd immunity would be reached at lower vaccination levels.

of the infected period.

$$V_j(S) = V_j(R) - \pi(\gamma_j, \alpha, i)c_j \tag{9}$$

Where $\pi(\gamma_j, \alpha, i)$ is the probability of becoming infected over the course of the epidemic as a function of the contacts of the agent and the transmission rate of the infectious diseases, $c_j = [U_j(S) - U_j(I)](1/\kappa)$, and $U_j(I) < U_j(R)$.

With the value functions specified we can now specify the vaccination problem for the individual and the social planner.

The Individual Vaccination Problem

The individual will choose to be vaccinated if the value of being in the recovered state less the cost of the vaccination is greater than the value of being in the susceptible state. Thus the agent will choose to be vaccinated if

$$V_j(R) - c(v) > V_j(S) = V_j(R) - \pi(\gamma_j, \alpha, i)c_j \tag{10}$$

which implies the agent will choose to be vaccinated if $c(v) < \pi(\gamma_j, \alpha, i)c_j$.

The Social Planner's vaccination Problem

The social planner's vaccination problem is more difficult than the individual vaccination problem. Essentially, the social planner's problem is to vaccinate agent j if the cost of the vaccination is less than the expected dis-utility of the marginal increase in infections created by agent j if agent j is infected weighted by the probability that agent j is infected. This is a difficult problem for at least two reasons:

1) The marginal infections created by agent j are not simply the number of other agents that j infects. This is because some agents that j infects may get infected by agents other than j even if j does not infect them herself. Thus one needs to know information on the dynamics of the entire epidemic to measure the true marginal infections of a given agent.

2) The marginal value of vaccinating an agent eventually must be decreasing in the number of other vaccinations that are performed. In the extreme, if there are enough vaccinations in the population to produce herd immunity the marginal value of vaccinating an additional agent only involves the probability that the agent is infected from outside the agent population. In effect, the only value is preventing a single agent from infection because she cannot infect anyone else.

Define $m_j(\gamma_j, \alpha, \kappa, i, v)$ as the true marginal infections created by agent j if infected, where v is the number of agents vaccinated in the population. For the majority of the paper we will suppress the notation that does not differ across agents and simply refer to marginal infections as $m_j(\gamma_j)$ since the primary focus of the paper will be on the effect of heterogeneous contacts on the spread of infectious diseases. We assume that this term is increasing in γ and α, and decreasing in κ, v, and i.[2] As described above, as the number of agents vaccinated increases there are fewer agents remaining to infect. Similarly, as i increases, the susceptible population decreases and again, there are fewer agents to infect.

We can now state the social planner's vaccination problem.

Vaccinate agent j if:

$$c(v) < \pi(\gamma_j, \alpha, i)[1 + m_j(\gamma_j)]c_j(i) \tag{11}$$

Note that the individual and social planner's vaccination problems differ by the term $\pi(\gamma_j, \alpha, i)m_j(\gamma_j)c_j(i)$. This is the positive externality created by a vaccination when a vaccinated agent j protects other agents which he contacts from acquiring the disease from agent j.

The key terms to investigate in this externality are the probability agent j gets infected, and the marginal infections created by agent j if infected. Note that if these marginal infections, $m_j(\gamma_j)$, go to 0, the social planner's problem and the individual problem converge,

[2]As shown in Boulier et al. marginal infections may be increasing in v for sufficiently small v. But, marginal infections must eventually decrease in v; at the extreme, marginal infections are 0 for any level of v above the point at which herd immunity is reached.

and the externality is removed. Similarly the externality is removed if enough vaccinations are performed to reach herd immunity.

Proposition 1 *As v approaches $(1-\frac{\kappa}{\alpha\gamma})N$ the externality, $\pi(\gamma_j,\alpha,i)m_j(\gamma_j)c_j(i)$, goes to 0.*

Proof The proof is direct. As shown above, if $v = (1-\frac{\kappa}{\alpha\gamma})N$ then we are below the epidemic threshold and $i = 0$. Thus the probability of becoming infected goes to 0 as would the marginal infections of agent j if j is infected from outside the population of agents.□

Corollary 1 *For $\kappa > 0$, there exists a level of vaccinations $\bar{v} < 100\%$ at which additional vaccinations produce no benefit.*

Proof Follows directly from the previous proof. Once the probability of being infected reaches 0, there is no need to vaccinate additional agents. $\bar{v}$ is reached at a level strictly less than 100% of the population for any positive recovery rate κ.□

2.2 Heterogeneous Contacts

We assume throughout the paper that the cost of an infection is equal across all agents, $c_j(i) = c_k(i)$ for all j and k. Further, without loss of generality, we can assume that $c_j(i) = 1$. Thus the externality above is the product of the probability of infection and the marginal infections produced by the agent if infected. One question then emerges: how are $\pi(\gamma_j)$ and $m(\gamma_j)$ related? At a simple level, if contacts only vary in degree, that is if the only difference in contacts between two agents is the number of contacts and not other, qualitative, aspects of the contacts, then you would expect $\pi(\gamma_j)$ and $m(\gamma_j)$ to be highly correlated. If an agent has a high likelihood of being infected because he has many contacts then he also has many contacts to pass on the infection.

Example 1: Uniform Random Contacts with Low Connectivity

In this case, any agent who has a large number of contacts will also generate a large number of secondary infections since there is a lack of structure within the network population.

Thus any agent with a high probability of infection, $\pi(\gamma_j)$ will also be expected to generate a high level of marginal infections $m(\gamma_j)$.

Example one is fairly direct. However, various relationships are possible as we show below.

Example 2: Fully Connected Graphs

In this case each agent in a population is directly connected to every other agent in the population. If this is the case, then any agent that becomes infected is directly tied to all other agents and can infect anyone in the population. Thus once someone is infected each agent has a high probability of becoming infected (either from the original agent or from secondary infections). But, since the first agent contacts everyone in the population, and many agents will be infected from him, the other agents in the population may have a low $m(\gamma_j)$. Thus it is possible to have a high probability of infection $\pi(\gamma_j)$ and low marginal infections generated $m(\gamma_j)$ from the same agent.

Example 3: A Bridge between Two Separate Fully Connected Graphs

Imagine that there are three groups of agents in a population. Two of these groups, call them A and B, are separate fully connected graphs containing equal numbers of agents, who do not have any connections to the other group. In other words an agent $a \in A$ is connected to every agent $a' \in A$ but no agent $a \in A$ is connected to any agent $b \in B$. Suppose that group B is formed in a similar manner. The third group is composed of one agent, j. Agent j has only two contacts: one to agent $x \in A$ and one to agent $y \in B$. In this example it may be unlikely that agent j gets infected, especially if there is a low transmission rate, since he only has two contacts in the populations. But, if agent j is infected, he may be integral to spreading the disease to the second fully-connected group. Suppose an agent in A becomes infected and subsequently infects agent x (or any of the other agents in A) as well as several other agents in A. Agents in group B are safe from infection as long as agent j is not infected. But, if j becomes infected, then it is possible that a large fraction of agents in B may become infected as well. Thus agent j may have a low probability of being infected,

$\pi(\gamma_j)$, but create a large number of marginal infections, $m(\gamma_j)$, if he does become infected.

Note that each of these three examples offer different implications for public policy approaches to encouraging vaccinations. In the first example, each agent has a probability of being infected that is in line with the number of marginal infections generated. In the other two examples, the infection rate and the number of marginal infections generated may not have a simple relationship with each other. This is an important observation if one considers using subsidies or other means to encourage increased vaccination rates as we discuss below.

2.3 Vaccination Subsidies

The use of subsidies is one way to increase vaccination rates for healthcare workers who create large externalities in the spread of an infectious disease. Using the model outlined above, an agent will choose vaccination when a subsidy is offered if $c(v) < \pi(\gamma_j, \alpha, i)c_j(i)+T$, where $T > 0$ is the subsidy transferred to the agent if vaccinated. Rearranging terms yields the following incentive compatibility condition for agent vaccination:

$$\pi(\gamma_j) > \frac{c(v) - T}{c_j(i)}. \tag{12}$$

Notice that the decision to accept the subsidy is still only a decision made on the basis of the agent's probability of being infected and not of $m(\gamma_j)$. Agents with a low probability of being infected will not accept the subsidy but agents with high probability of infection will accept the subsidy irrespective of the the level of marginal infections generated.

Consider a uniform subsidy, T, offered to all agents in the population if they accept vaccination in the hope of controlling the externality, $\pi(\gamma_j)m(\gamma_j)c_j(i)$. As an additional condition for the subsidy, the positive externality associated with each vaccination should be larger than the subsidy, $\pi(\gamma_j)m(\gamma_j)c_j(i) > T$ or:

$$m(\gamma_j) \geq \frac{T}{\pi(\gamma_j)c_j(i)}. \tag{13}$$

Define a vaccine to be *cost effective* if Equation 13 holds. In other words, the marginal infections generated must be sufficiently large for the subsidy to be cost effective. Further it is possible that some agents would accept the subsidy but not satisfy the condition in Equation 13.

Non-Cost-Effective Vaccination Performed

If we rewrite this inequality, we can state the following relationship between marginal infections and the infection rate where an accepted subsidy would not be beneficial: if $\pi(\gamma_j) < \frac{T}{m(\gamma_j)c_j(i)}$ then the positive externality generated by the vaccination is less than the subsidy. We can now combine this with our incentive compatibility constraint and see that the following levels of probability of infection would imply that the agent would accept the subsidy and be vaccinated but not yield a cost effective vaccination:

$$\frac{T}{m(\gamma_j)c_j(i)} \geq \pi(\gamma_j) > \frac{c(v) - T}{c_j(i)} \tag{14}$$

Again, the marginal infections, $m(\gamma_j)$, must be sufficiently large for a vaccination subsidy to be cost effective: if the marginal infections are too small then some agents may accept the subsidy but not provide a sufficiently large positive externality for the subsidy payment to be cost effective.

Cost-Effective Vaccination Not Performed

The concern with a vaccination program subsidy offered to all members of a population is two-fold. Above, we discussed the possibility of non-cost-effective vaccinations if the probability of infection is large and the number of marginal infections generated are small. However, there is also a second problem: if an agent has a relatively low probability of infection but generates a large number of marginal infections this individual will choose not to be vaccinated even though vaccinating this particular agent is in the best interest of the population as a whole. In this case the vaccinations are cost effective, but not incentive compatible. Rearranging our inequality from above, this situation occurs for agents where:

$$\frac{c(v) - T}{c_j(i)} \geq \pi(\gamma_j) > \frac{T}{m(\gamma_j)c_j(i)} \tag{15}$$

In this situation the agent has sufficiently low probability of infection so that the left hand inequality holds but a sufficiently large marginal infection rate that the right hand inequality also holds.

Targeted Vaccination Subsidies

The problems discussed above with across the board subsidies can be overcome with targeted subsidies. Both of the problems occurred when there was a misalignment of the probability of infection and marginal infections generated. The issue is that we may want to give a larger subsidy to individuals that generate large marginal infections but have a small probability of infection and a smaller subsidy (or potentially tax) to individuals with a large probability of infection but who generate few marginal infections. Effectively, agencies may want to target their subsidies toward the marginal infections and less toward the probability of infection. The difficulty, of course, is in identifying the marginal infections created by a particular agent.

3 Data

In the remaining portion of the paper we examine a newly collected data set on contacts of and between healthcare workers and patients in a large hospital on the University of Iowa campus. We discuss the data and use simulations to identify the healthcare workers who have the potential to create large numbers of marginal infections in the hospital and to discuss the characteristics of these workers that can be used to guide efficient vaccination policies.

Observational data on contacts between healthcare workers and patients was collected during the winter and early spring of 2006-2007 (the 2006-2007 "flu season") at the University of Iowa Hospitals and Clinics (UIHC). The UIHC is a 700-bed comprehensive academic

medical center and regional referral center in Iowa City. Data were collected by randomly selecting UIHC employees from each of 15 job classifications (specified below) and then using graduate students to "shadow" the 140 selected employees and manually record their every human contact (within approximately three feet) over a total of 640 hours. A total of 6,654 contacts were recorded, with each contact indicating type of contact (patient or category of healthcare worker), location, length of contact time, whether physical contact was made, whether the contact took place in a patient room, and whether handwashing/sanitizing occurred prior to contact.[3]

The job categories and number of observed subjects in the data set are as follows: Floor Nurse (8), Food Service (11), Housekeeper (8), Intensive Care Nurse (8), Nurse Assistant (10), Pharmacist (8), Phlebotomist (10), Physical/Occupational Therapist (9), Resident/Fellow/ Medical Student (8), Respiratory Therapist (11), Social Worker (8), Staff Physician (11), Transporter (7), Unit Clerk (9), and X-Ray Technician (14). Each subject was shadowed by a research assistant (RA). The data for each group contain approximately 40 hours of shadowing. The data were summarized into tallies of contacts over 30-minute intervals and then aggregated into contacts per 8 hour shift by the authors. Table 1 lists the average number of non-repeated contacts[4] per eight hours that occur between the various worker (and patient) categories.

Note two things in the table: first, there is substantial variability in the number of contacts that occur across groups. Second, many, but not all, of the groups tend to have a large portion of their contacts with other members of their own group. We use this contact data to simulate the spread of an infectious disease across the UIHC hospital.

[3]The data collection was approved by the Institutional Review Board at the University of Iowa.

[4]A non-repeated contact is a contact between two workers or a worker and a patient that has not been observed before.

Table 1: Average Contacts between Worker Categories per Eight Hours

	F N	Food	HK	IC N	N Asst	Pat	Pharm	Phleb	P Ther	Res	R Ther	SW	Stf Phys	Trans	UC	X-Ray	Tot
F N	11	0	1	1	5	12	1	0	2	4	1	1	2	1	3	1	46
Food	5	8	1	3	3	22	1	1	0	1	0	1	1	0	6	0	53
HK	6	0	3	1	4	3	0	0	1	1	0	0	1	0	0	0	20
IC N	1	0	1	13	2	6	1	0	1	3	3	1	4	0	2	2	40
N Asst	8	1	1	4	3	16	0	0	2	1	1	1	0	1	3	0	42
Pat	12	22	3	6	16	0	4	38	16	10	10	8	16	23	5	17	206
Pharm	7	0	0	2	1	4	3	0	0	6	0	1	3	0	2	0	29
Phleb	5	0	0	0	1	38	1	4	0	1	1	0	0	0	2	0	53
P Ther	12	1	1	5	4	16	1	1	5	3	1	1	2	1	4	0	58
Res	5	1	0	6	1	10	1	0	0	9	2	1	3	0	2	1	42
R Ther	6	1	1	11	2	10	1	0	1	6	16	0	2	0	3	3	63
SW	29	0	1	3	1	8	1	0	3	11	1	4	5	0	6	0	73
Stf Phys	6	0	0	5	1	16	1	0	1	18	0	1	3	0	1	1	54
Trans	4	0	1	0	2	23	0	1	2	1	1	0	1	7	2	0	45
UC	52	1	1	13	15	5	1	0	5	17	1	4	5	2	4	1	127
X-Ray	3	0	1	4	2	17	0	0	0	1	1	0	1	1	1	15	47
Tot	172	35	16	77	63	206	17	45	39	93	39	24	49	36	46	41	998

Table 2: Employees at the UIHC.

Floor Nurse	804
Food Service	456
Housekeeper	356
IC Nurse	190
Nurse Asst	386
Patient	482
Pharmacist	276
Phlebotomist	74
Physical/Occupational Therapist	90
Residents/Fellows/ Med Students	666
Respiratory Therapist	108
Social Worker	114
Staff Physician	760
Transporter	108
Unit Clerk	126
X-Ray Technician	236
Total	5,232

4 Simulations

We simulate a model of the spread of an infectious disease across a hospital network. The network is constructed to match the distribution of worker groups at the University of Iowa Hospitals and Clinics. This totals 5,232 employees. The distribution of workers across the categories is given in Table 2. As one can see, there is a large amount of heterogeneity in the size of the worker groups in our sample.

We create a contact network among these agents. In the simulations, each worker in a given group connects to other workers according to the rates observed in our shadowed subjects given in Table 1. As an example, all floor nurses in the model create 11 contacts to other randomly selected floor nurses on average, 0 contacts to food service workers, etc... The contacts are symmetric, that is, a contact from a floor nurse to a housekeeper is also a contact from a housekeeper to a floor nurse. There are at least two reasons for this assumption. First, if our subject is in close enough proximity to pass on the influenza virus to a second agent, the second agent is also within close enough proximity to pass on the

influenza virus to our subject. Thus the ability to acquire or to pass on virus is a symmetric relationship. Second, the reader may note in the table that the matrix of observed contacts is not symmetric because of randomness in the observation of subjects. For instance one notices that the subject floor nurses were not observed to contact food service workers, but a small number of food service worker to floor nurse contacts were observed. Thus by assuming that all contacts that occur in the matrix are undirected, we create a symmetric contact matrix where the total number of contacts from a member of group x to group y (and from group y to group x) is one-half the sum of the observed average contacts from group x to group y and from group y to group x.

We create the contacts in a uniform random manner within groups. Let ρ_{ij} be the ratio of the average contacts between a member of group i and j (taken from Table 1) to the total number of group j employees (taken from Table 2.) We then take each pair of employees across each group and create a contact with probability ρ_{ij}. Specifically, let agent a be a member of group i and agent b be a member of group j. Then the probability that a and b are connected is $\rho_{ij} = C_{ij}/N_j$, where C_{ij} is the average number of contacts observed between members of groups i and j and N_j is the number of employees belonging to group j.

Once created, we use the contact network to simulate the spread of influenza in the hospital. Agents can be in one of three states, susceptible to acquiring influenza (S), infected with influenza (I), or recovered (and therefore immune) from influenza (R). Each infected agent recovers after 5 periods. Once recovered the agent enters state R and is therefore immune to further transitions to the infected state.

Initially all agents are in state S. Agents may be vaccinated against acquiring influenza. Vaccinations occur only in the initial period of the model. Once vaccinated, an agent moves immediately from state S to state R and is thus immune to influenza for the remainder of the simulation.

In the initial period of the simulation, each agent in state S (all agents that have not been vaccinated) is subject to infection with probability $\alpha_0 = 0.005$. These are the agents of

our model that seed the potential epidemic. Once these initial infections occur we assume that each contact in our network occurs once in each subsequent period of the model. If a contact occurs between a state I and a state S agent, the state S agent transitions to state I with probability α which varies across experiments. We continue the simulations until no agents remain in state I. In each period of the simulations we calculate the fraction of agents in each worker category in each state, (S, I, or R.)[5]

4.1 Experiments

The purpose of the simulations is to estimate $m(\gamma_j)$ and the externality generated for the network of contacts in the UIHC shadow data and, in turn, to identify the classes of workers most important to vaccinate. This is a two step process. First we perform a series of base-line simulations as described above with none of the healthcare workers and patients vaccinated. From these simulations, we observe the rate of infection for each class of agents in the hospital population (15 worker groups and patients for a total of 16 groups). We denote the infection rate of group k in the *base* simulations as a function of the transmission rate α as $\pi_b^k(\alpha)$ and the overall infection rate in the entire population as a function of α as $\pi_b^0(\alpha)$. Second, we want to calculate the marginal infections generated by each group. To do so, we change the vaccination rate for each group to 1.0, one group at a time, and re-perform the simulations. As an example, we run the simulations with all floor nurses vaccinated and no other vaccinations and observe $\pi_1^0(\alpha)$. Then we run the simulations with all housekeepers vaccinated and no one else and observe $\pi_2^0(\alpha)$ and so on for each group. We then compare the change in the average infection rates between the simulations, $\delta(b,k) = \pi_b^0(\alpha) - \pi_k^0(\alpha)$, which is the difference between the overall infection rate in the base simulation with no vaccinations and the overall infection rate in the simulation with all of group k vaccinated. Now, using the notation described above, the change in infections $\delta(b,k)$ is equal to the

[5]We have studied a wide range of parameters for our model and find the results reported below to be robust to changes in all of the parameters within reasonable bounds. Additional results and details are available from the corresponding author, as is the source code for the simulations.

change in number of people vaccinated, N^k, multiplied by the probability that each of these agents becomes infected if not vaccinated, multiplied by the number of additional infections each agent would generate:

$$\delta(b,k) = N^k(\pi_b^k(\alpha))(m^k) \tag{16}$$

One can then find an estimate of the marginal infections prevented per vaccination as:

$$\hat{m}^k = \frac{\delta(b,k)}{\pi_b^k(\alpha)N^k} \tag{17}$$

Effectively, this process removes each group, one at a time, from the hospital population. We then can observe the effect of each individual worker group on the simulated epidemic.

Base Experiments

We begin by varying the transmission rate, α, over the range [0.0004, 0.004] and observing the base infection rate $\pi_b^0(\alpha)$. The results are displayed in Figure 1. As one can see in the figure, a sufficiently large transmission rate is needed to generate an epidemic of reasonable size. Further, as expected, the number of infections generated monotonically increases as a function of the transmission rate, α. Our primary interest is in intermediate ranges of epidemic outbreaks. If the transmission rate is too high then almost everyone in a population needs to be vaccinated in order to reach herd immunity. And, if the transmission rate is too low, then there is not a large need to worry about vaccine priority. Thus, we concentrate on two intermediate levels of the transmission rate $\alpha = 0.0020$ and $\alpha = 0.0025$. With no vaccinations, these levels yield an epidemic where slightly more than one-half of the population is infected over the course of the epidemic.

Marginal Effect of Vaccinations

We now find the marginal effect of vaccinations across the hospital worker groups using the procedure described above for $\alpha = 0.0020$ and $\alpha = 0.0025$. We present results for the probability of infection, $\pi_b^k(\alpha)$, marginal infections generated per infection, $\hat{m}^k$, and the product of these two items which yields the decrease in infections per vaccination, $\frac{\delta(b,k)}{N^k}$ in

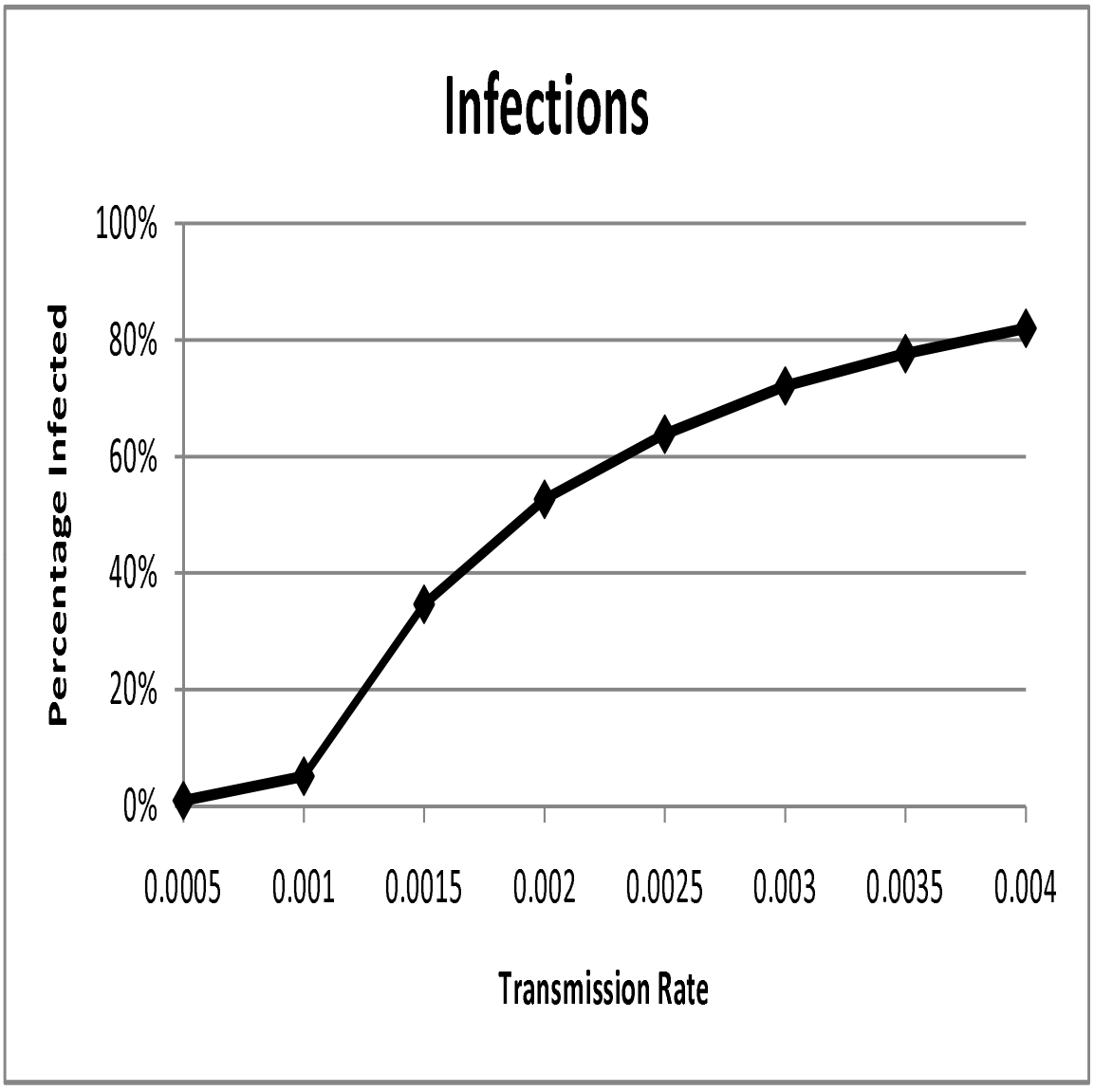

Figure 1: Infection rate as a function of transmission rate.

Table 3: Estimate of Marginal Infections Prevented per Vaccination, $\alpha = 0.0020$

Group	π_b^k	$\hat{m}^k$	$\frac{\delta(b,k)}{N^k}$
Floor Nurse	69.6%	2.13	1.48
Food Service	37.1%	1.36	0.50
Housekeeper	19.7%	1.30	0.26
IC Nurse	51.1%	1.77	0.90
Nurse Asst	47.0%	1.54	0.73
Patient	79.3%	3.26	2.59
Pharmacist	29.8%	1.44	0.43
Phlebotomist	89.4%	3.16	2.82
Physical/Occupational Therapist	83.1%	2.82	2.34
Residents/Fellows/ Med Students	47.3%	1.53	0.72
Respiratory Therapist	71.1%	2.31	1.64
Social Worker	86.1%	3.22	2.77
Staff Physician	36.6%	1.41	0.52
Transporter	69.9%	2.16	1.51
Unit Clerk	95.4%	5.14	4.91
X-Ray Technician	45.8%	1.56	0.71

Table 3 and 4.

From the decrease in infections per vaccination we have an indication of how much the vaccination of an individual group member is contributing to preventing the spread of an epidemic. The results of this experiment suggest where efforts should be directed in the event of an influenza vaccine shortage or in the event of the development of new disease for which a vaccine may be developed (e.g., avian flu, swine flu, etc.) but is initially in short supply until mass quanities may be made available. Note that some of the groups have vaccinations that prevent less than one infection per vaccination. This occurs because these groups have a low probability of infection and sufficiently low number of marginal infections that each member generates if infected. Groups with large decreases in infections per vaccination are the ones to prioritize in times of a vaccine shortage.

In these experiments we see five clear groups that stand above the others in terms of the effectiveness of vaccinations. For the parameters of the experiments, each vaccination of a unit clerk, social worker, physical and occupational therapist, phlebotomist, and hospital

Table 4: Estimate of Marginal Infections Prevented per Vaccination, $\alpha = 0.0025$

Group	π_b^k	$\hat{m}^k$	$\frac{\delta(b,k)}{N^k}$
Floor Nurse	80.7%	1.73	1.40
Food Service	48.5%	1.19	0.58
Housekeeper	27.5%	1.11	0.31
IC Nurse	65.3%	1.49	0.97
Nurse Asst	59.2%	1.28	0.76
Patient	90.0%	3.02	2.72
Pharmacist	41.0%	1.23	0.50
Phlebotomist	96.4%	2.16	2.08
Physical/Occupational Therapist	92.1%	2.11	1.95
Residents/Fellows/ Med Students	60.0%	1.39	0.83
Respiratory Therapist	83.9%	1.88	1.58
Social Worker	94.7%	2.57	2.44
Staff Physician	48.2%	1.30	0.62
Transporter	81.3%	1.48	1.21
Unit Clerk	98.8%	4.14	4.09
X-Ray Technician	58.5%	1.26	0.74

patient results in a decrease of 1.95 infections or more on average (we discuss characteristics of the network contacts of these groups below). In addition vaccinating unit clerks is extremely effective; each unit clerk vaccination results in a decrease of over four infections. Somewhat surprisingly, some of the groups that are seen as central to the functioning of a hospital play a very small or moderate role in spreading an infectious disease. Vaccinating staff physicians or nurse assistants results in two of the lowest decreases in infections at just over one-half and three-quarters of an infection decrease per vaccination; floor nurses also result in a surprisingly low decrease in infections per vaccination at just under one and one-half.

Also of note, as one would expect, as the transmission rate increases, the probability of infection increases. But, this has the effect of making individual vaccinations less beneficial at the margin. Note that $\hat{m}^k$ is smaller for each group for a higher transmission rate. This has the effect of lowering the variance of the decrease in infections per vaccination across groups. For the $\alpha = 0.0020$ case above the variance is 1.61, and for the $\alpha = 0.0025$ case the variance is 0.98. As the transmission rate increases, a larger fraction of individuals are

infected throughout the population. Thus there are more opportunities for each individual to be infected if she has not already been infected. Vaccinating a given person in the population will only prevent one of these multiple channels for infection. So, as the infection rate increases, the effectiveness of a vaccination becomes more uniform across the groups. This has direct policy applications. An infectious disease that is highly contagious could best be met with a uniform vaccination strategy since each individual in the population will create a similar level of marginal infections. But an infectious disease with a low level of contagiousness could most effectively be met with a targeted vaccination campaign (Bansal et al.).

Comparison of Marginal Infections and Probability of Infection

Recall that the externality generated from vaccination is a function of the product of the probability of infection for the agent and the marginal infections generated if infected, $\pi(\gamma_j)$ and $m(\gamma_j)$. In Table 3 and 4 we also present the value of $\hat{m}^k$. Also recall that it is possible that these two items may be positively or negatively correlated depending on the network structure as discussed above. As can be seen in the tables above there is a positive correlation between probability of infection and the number of marginal infections generated (specifically, the correlation is 0.88 for the $\alpha = 0.0020$ experiments and 0.77 for the $\alpha = 0.0025$ experiments). From a public policy perspective this is good news. Recall that the individual incentives to get vaccinated correspond to the probability of infection. Since this is positively correlated with the marginal infections that also drive the externality, the individual incentives help to alleviate the externality. In other words, it would be more difficult to control the externality if agents with the smallest individual incentive to get vaccinated were the ones most important to vaccinate from the perspective of controlling the externality.

4.2 Network Characteristics of Most Important Groups

We now move to discuss the important features of the contact network that create the externality. As we will see below, it is not just the number of contacts that an agent has but also which specific agents and groups the agent contacts, as well as who the agent's contacts connect to in turn.

In Table 5 we display some basic statistics of the contacts in our data. For each group, the table displays the total number of contacts, the percentage of total contacts that are with members of an agent's own group, the number of groups in which an agent has at least one contact, and the number of patient contacts. Total contacts and contacts with patients could be directly correlated with the likelihood of being infected and with passing on infections. The number of contacts outside of one's own group and the number of other groups contacted indicate how varied one's network is and how widespread one's connections are. For instance having contacts with all groups in the hospital probably puts one more at risk to be infected and also provides the possibility of introducing an infection to many groups within the hospital.

What is most interesting in Table 5 is the lack of a clear relationship between any of these variables and our previously-listed most important groups (highlighted in bold). Each of these four plausibly important characteristics fail to signify a statistically significant relationship with probability of infection or with the marginal infections generated. If we concentrate on the top four most important groups, some have relatively large numbers of contacts (unit clerks), although not the largest, while others have contacts significantly below the average (phlebotomists). Some have large numbers of patient contacts (phlebotomists) while others have some of the smallest number of patient contacts (unit clerks and social workers). What this implies is that there is not likely to be a simple relationship indicating which individuals are most important to vaccinate. The relationship will depend on the intricate and complex web of relationships that make up the entire contact network of the hospital.

Table 5: Contact Characteristics

Group	Total Contacts	% Contacts Outside Own Group	# Groups Contacted	Patient Contacts
Floor Nurse	196	174	15	11
Food Service	58	43	13	21
Housekeeper	29	20	8	2
IC Nurse	97	73	13	5
Nurse Asst	78	73	12	15
Pharmacist	34	29	10	3
Phlebotomist	**54**	**47**	**8**	**37**
Physical Ther	**67**	**59**	**16**	**16**
Residetn/Fellow	101	85	13	9
Respitory Ther	81	49	14	10
Soc Worker	**81**	**74**	**13**	**7**
Staff Phys	75	71	12	15
Transporter	48	36	11	22
Unit Clerk	**158**	**151**	**16**	**5**
X-Ray Tech	64	35	12	17
Average	81	68	12	13

Table 6: Estimate of Marginal Patient Infections Prevented per Vaccination, $\alpha = 0.0020$

Group	π_b^k	$\hat{m}^k(P)$	$\frac{\delta(b,k)}{N^k}(P)$
Floor Nurse	69.6%	0.12	0.08
Food Service	37.1%	0.11	0.04
Housekeeper	19.7%	0.03	0.01
IC Nurse	51.1%	0.07	0.04
Nurse Asst	47.0%	0.09	0.04
Pharmacist	29.8%	0.03	0.01
Phlebotomist	89.4%	0.65	0.58
Physical/Occupational Therapist	83.1%	0.28	0.23
Residents/Fellows/ Med Students	47.3%	0.06	0.03
Respiratory Therapist	71.1%	0.14	0.10
Social Worker	86.1%	0.16	0.14
Staff Physician	36.6%	0.06	0.02
Transporter	69.9%	0.30	0.21
Unit Clerk	95.4%	0.25	0.24
X-Ray Technician	45.8%	0.12	0.06

5 Protecting Patients

From one perspective a hospital's primary goal is to restore or improve patient health. Thus prioritizing healthcare worker vaccinations so as to best protect patients may be a legitimate goal. In other words hospital administrators may care about protecting patients from infection as much, or more, as they do about protecting the entire hospital population from infection. Of course these are clearly related. In Table 6 and 7 we display the same relationships as displayed above but this time only with regard to patient infections generated, not infections in the entire hospital ($\hat{m}^k(P)$ is the marginal patient infections prevented, and $\frac{\delta(b,k)}{N^k}(P)$ is the patient infections prevented per vaccination).

In this analysis we see very similar results to the overall population results. The four non-patient groups highlighted above (unit clerk, social worker, physical and occupational therapist, and phlebotomist) play an important role in transmitting to the hospital population as a whole as well as to patients specifically. However, we see some difference in the two groups of simulations. First, groups that have more direct patient contacts increase in

Table 7: Estimate of Marginal Patient Infections Prevented per Vaccination, $\alpha = 0.0025$

Group	π_b^k	$\hat{m}^k(P)$	$\frac{\delta(b,k)}{N^k}(P)$
Floor Nurse	80.7%	0.05	0.04
Food Service	48.5%	0.05	0.02
Housekeeper	27.5%	0.01	0.00
IC Nurse	65.3%	0.03	0.02
Nurse Asst	59.2%	0.05	0.03
Pharmacist	41.0%	0.01	0.00
Phlebotomist	96.4%	0.37	0.36
Physical/Occupational Therapist	92.1%	0.13	0.12
Residents/Fellows/ Med Students	60.0%	0.03	0.02
Respiratory Therapist	83.9%	0.07	0.06
Social Worker	94.7%	0.07	0.07
Staff Physician	48.2%	0.04	0.02
Transporter	81.3%	0.14	0.11
Unit Clerk	98.8%	0.10	0.10
X-Ray Technician	58.5%	0.05	0.03

importance. For instance, phlebotomists replace unit clerks as the most important group. Second new groups emerge as important for transmitting to patients. For instance, hospital transporters are among the top four groups in transmitting to patients but are significantly below the average in terms of transmissions to the general population. With this in mind it seems that giving vaccination priority to health care workers with direct patient contacts is more important for protecting patients than it is for protecting the general population. But still, some of the groups with the largest impact on infecting patients have few direct patient contacts (unit clerks and social workers, for example).

Finally, we briefly note the relationship between the network variables discussed above and the patients infected. Not surprisingly one can notice that groups with more patient contacts create more marginal patient infections relative to overall infections discussed earlier.

To summarize the results of this section, the same groups that create infections in the general population also create infections in the patient population. But, groups that have direct patient contacts have increased importance in transmitting to patients. Still, one

should not ignore other groups central to the network of the hospital that have only few direct patient contacts (e.g., social workers and unit clerks).

6 Conclusion

In this paper we analyzed the targeting of vaccine subsidies to specific groups of healthcare workers. The externality generated by a vaccination involves the product of both the probability an agent is infected and the number of additional infections generated by an agent if infected. Past public policy has focused more on the first item than the second. But in this paper we show that because the incentives for an agent to accept a vaccination is directly related to the agent's probability of infection, it is potentially more important to focus on the marginal infections generated when designing vaccination programs.

Further, we utilize a newly collected data set on contacts of health care workers at a large university hospital to estimate marginal infections for infectious disease transmission. Interestingly the most important groups to vaccinate tend to have heterogeneous contacts throughout the hospital. Groups such as social workers and unit clerks are very important to vaccinate even though they have been given low priority in past vaccine campaigns because of their relatively limited number of patient contacts. This mismatch of scientific results and past policy decisions suggests that future research in this area is warranted especially when one considers the public health dangers associated with the emergence of avian flu, a more lethal version of swine flu, or recent dangers such as SARS.

The results of this paper lead to important public policy considerations. Specifically, hospital workers with a low probability of infection may be likely to ignore recommendations for vaccination even if they are central to the spread of an infectious disease. One way to increase the overall vaccination level is with a subsidy program. But, as the results in this paper show, not all hospital workers are equal in terms of the positive externality generated by a vaccination. Because of the heterogeneous contacts throughout the hospital, some workers are more important to the spread of an infectious disease than others. Thus if hospitals

and other public health organizations want to efficiently distribute vaccines they need to target specific worker groups, perhaps by allocating subsidies, on the basis of discrepancies in probability of infection and marginal infections generated. This paper is the first to use specific micro-level contact data within a hospital to guide policy makers and public health officials in this endeavor.

To be clear, these results are not meant to be specifically calibrated to measure the exact effect of vaccinations in these groups. Instead our hope is that the orderings of the hospital worker groups (which are robust across the parameters that we have explored) indicate where public health officials can effectively intervene in order to prevent widespread epidemics within hospitals. And these experiments reveal interesting and surprising groupings. Prior to this study it had been argued that groups like unit clerks be excluded from influenza vaccine campaigns, in times of vaccine shortages, because of their minimal patient contacts. The results of this study suggest that would have been a very poor decision.

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