Justification For Altering the Model

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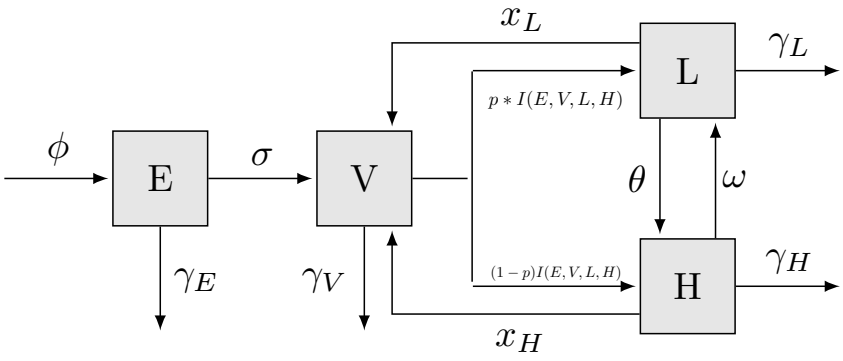
1 Current Model Formation

The current structure of our model to this point. The core assumptions that lead us to developing this model were that some PWID initiate more people into injection than others (ie L vs H) and that only individuals who are vulnerable to being initiated are at risk (ie E vs V). The current initiation function is a standard infectious disease function, measuring the probability of interacting with a PWID, assuming the odds of bumping into individuals is uniform. Here is that model :



2 Concerns With the Current Model

After beginning the literature review process, it feels that the model, as is, has some issues that are not reflective of the initiation process. Goldsamt (2010) found that almost all injectors were exposed to injectors before being initiated and that, even of those few who self-initiated, they learned from other injection drug users (as of writing this, Lit Review is ongoing). This gets at a core assumption that Dan has been insistent be in the model, that there are two necessary but not sufficient elements to being initiated: the first, which we have incorporated, is the idea of vulnerability due to factors unrelated to knowing

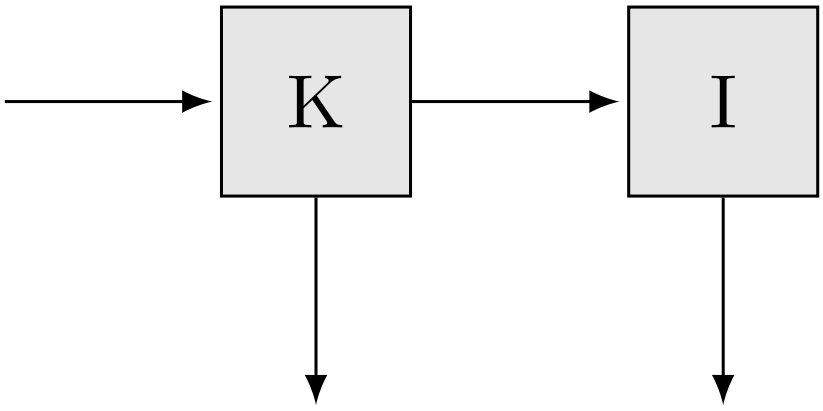
other injectors (ie housing status, health status); the second, which we do not have incorporated, is knowing and interacting with injection drug users.

Currently, our initiation function treats bumping into PWID as being a game of random chance, in which the probability of bumping into one is simply the probability of bumping into any specific person. It is a function of the entire population size. But it seems that initiation is just a function of the number of PWID and the set of people that they interact with... $|E|$ could be 10,000 or it could be 10,000,000, the number of people that know PWID is not dependent on the size of the entire population but only on the number of PWID (ie if the average PWID knows 10 people, then the number of people they know is just the number of PWID times 10, it doesn't matter the exact size of the overall population...). The literature suggests that initiation is a result of sharing space with PWID over a period of time, not random chance encounters.

So, the concern is that the assumption that knowing/interacting with PWID is a necessary component to being initiated is not currently incorporated appropriately into the model. We need a model that allows us to track both the population which knows PWID and the group within this that is vulnerable to being initiated.

3 Another (Simpler) Model Approach

So, the question, what does a model look like that assumes that initiation is a function of PWID and the people that they know. The initial set of compartments flows look like this, where I is the set of PWID (ie potential initiators) and K are the people that they know :



Regardless of whether or not an individual self-initiates or not, data indicates that these individuals are still exposed to PWID before their self-initiation. It may be worth a future conversation to ask if there should be an inflow into I which represents people who self-initiate without ever having met a PWID.

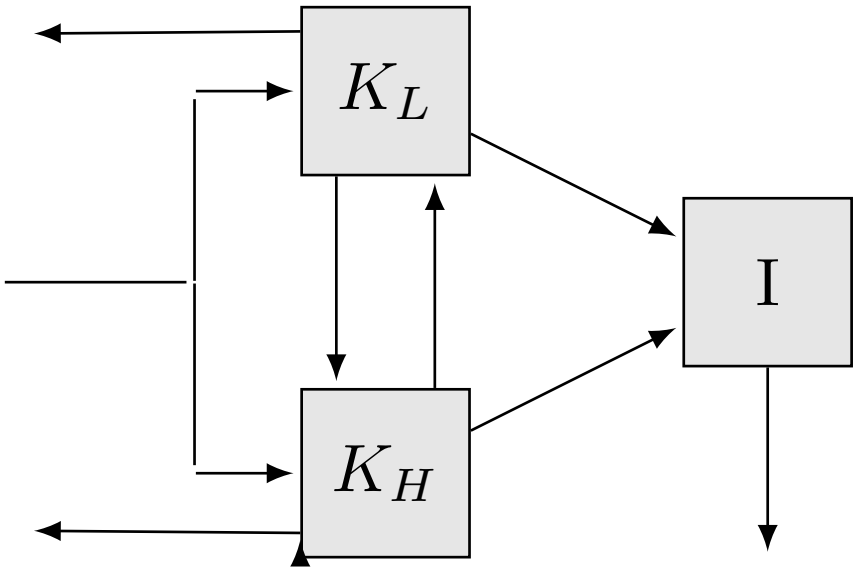
But, this only incorporates one of our two primary assumptions, and now we need to incorporate the idea of vulnerability into this model as well.

4 Breaking Down K

Now, we still want to incorporate our notion of "vulnerability", our assumption that initiation is a result of both contact and vulnerability. Thus, we need a way, either in our transition function or in a way we might subdivide K, to differentiate between varying levels of vulnerability amongst the K population. At its most simple level we want to differentiate between high and low vulnerability.

Note, that previously we were trying to do the opposite process, we were attempting to take the entire "vulnerable" population and determine which of them have contact with PWID. By first selecting for proximity to PWID, instead of vulnerability, we are immediately working with both a smaller and more relevant population – ie, those in proxmity to PWID.

So, for the first version of this model we can consider breaking K down into a low-vulnerability group K_L and a high-vulnerability group K_H. It seems reasonable to think that members of both compartments could be initiated and thus we would adjust our model accordingly :



5 Breaking Down I, also see 6.2

Now, we still also have to ask the question about differences in initiator behavior. A majority of PWID do not initiate anybody. A small group only initiate

some. A smaller group initiate multiple people over their injection careers. A core assumption was that if we divided PWID into "low-rate" and "high-rate" initiators that we would in essence be dividing PWID based on roles in the PWID community (ie that we would find "hit doctors" and other such individuals populating H). But, thus far, quantitative data does not suggest that people report getting initiated by "hit doctors" nor does it appear folx identify themselves as "hit doctors" (from the PRIMER data). This could be due to the fact that "hit doctor" sounds negative and people don't want to be labeled that way, but it also seems that there isn't hard enough data to suggest we can effectively hypothesize the difference between L and H. What we can tell is that initiator rates are power-law distributed amongst PWID, with the majority initiating zero people, the average initiating less than 1, and a small number initiating the majority of all new initiates. The question is, how do we incorporate this distribution of initiation into the model. Do we incorporate it into the flows or do we split I?

An Important Question (I don't know the answer, just musing) : Previously, we were modeling the probability of randomly "encountering" either a low- or high- rate initiator, but if K represents the population that already knows at least one PWID, isn't it fairly likely that their association to a "low-rate initiator" gives them access to high-rate initiators? In other words, previously, our model was giving high-rate initiators an agency by which they were more infectious than low-rate initiators, but is it possible that contact with PWID means contact with high-rate initiators is ensured? This speaks to the White (2015) paper, in which initiations can be credited to multiple people and that the relevant measure is not direct initiation, but presence at an initiation? The implication of this is that there isn't really a need to split the I population into multiple compartments, at least not by low- or high- rate.

On this note, the Goldsamt paper, and a few others note that the median number of aided injections an individual needs before they have the proficiency to self-inject is 3 to 4 injections. If we take inability to self-inject as a proxy for inability to directly initiate others, this may be a meaningful distinction by which to stratify I? (I'm not sold on this idea, just throwing it out there). But it at minimum suggests that there is an intermediate step between K and I where someone is now PWID but they are not yet able to initiate. But, this may be moot, as noted above, if someone knows a PWID who isn't able to initiate then that person still has access to the person(s) who initiated that other person. (Ie access to one PWID means access to initiators).

My impression from reading papers is that access to PWID networks is more important than access to specific PWID, and therefore stratifying I may be unnecessary.

6 System Dynamics

So, just some thoughts on how this system would be expected to behave. Clearly, the population size of I and K are directly tied together. If I grows than so does

K and vis versa. Therefore, this system is not going to have a stable population size. If I were to dip to 0, then by definition so must K. (Ie this is not a closed system (is that appropriate terminology?))

6.1 Defining System Flows

The flow into the K population is largely going to be based on the size of I. If I grows, then even though that growth is coming from K, then K will grow. Initially the size of K could be based on an average number of people an individual in I knows, and that when I changes, K changes by that rate.

The flow out of K_L and K_H can be a place where we define the impact of various interventions. If we decide to stratify I, then this function will be dynamic based on the proportion of I made up of these compartments, but at its most simple this flow will be a probability of initiation. Contact has already been established so we will not utilize an infectious disease function. Essentially, if before we had $c * \beta * \frac{(L+H)}{E+V+LH}$ as out initiation, now we are simply left with β. We will be able to incorporate the impact of various preventive efforts into this function.

6.2 Incorporating Environment

The data also suggests that environment is a significant factor in initiation. One of Dan's papers found that residence in Vancouver's Downtown Eastside was a significant factor in increased risk of being initiated. Therefore, we may want to consider incorporating an "environment function" either into the in-flow to K or the flow between K and I. The in-flow to K may be impacted by environment because if PWID are more visibile (ie public injecting), then they are likely to know more people. Environment could be incorporated in the flow from K to I as a modifier to the "infection" rate β (I am not convinced of this idea).

In addition (I like this idea quite a bit), this is where we may also think about splitting I up. PRIMER is amassing information on the factors that indicate what makes a "high-risk initiator", and if we opt to split I into L and H, *then environment could modify the rate at which PWID flow between L and H*. In other words, the environment of a given location could be a function which modifies the proportion of the I population which are in H (ie if public injecting is more prevalent we would expect H to represent a larger share of I).